CLÉMENT / DYSRHYTHMIA DURING CALORIC NYSTAGMUS

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An electronystagmographical study of the quantitative and qualitative aspects of caloric nystagmus and how these are influenced by arousal in the normal individual

ACADEMISCH PROEFSCHRIFT

TER VERKRIJGING VAN DE GRAAD VAN DOCTOR IN DE GENEESKUNDE AAN DE UNIVERSITEIT VAN AMSTERDAM, OP GEZAG VAN DE RECTOR MAGNIFICUS MR. A. D. BELINFANTE, HOOGLERAAR IN DE FACULTEIT DER RECHTSGELEERDHEID, IN HET OPENBAAR TE VERDEDIGEN IN DE AULA VAN DE UNIVERSITEIT (TIJDELIJK IN DE LUTHERSE KERK, INGANG SINGEL 411, HOEK SPUI) OP DONDERDAG 23 APRIL 1970 DES NAMIDDAGS TE 5 UUR PRECIES

door

PETER ADELIN RICHARD HENDRIK ELODIE MAXIMILIAAN CLÉMENT
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PROF. DR. L. B. W. JONGKEES

Dit proefschrift werd bewerkt in de vestibulaire afdeling van de Keel-, Neus- en Oorheelkundige kliniek, Wilhelmina Gasthuis, Amsterdam.

Aan Vera

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CHAPTER I

Introduction

Nystagmus is a phenomenon which has been studied for many years. Especially the quantitative parameters received a great deal of attention. In recent years, however, a growing interest has developed in the varying forms and periodic changes which a nystagmus (nystagmus pattern) can show. Some authors have attached an important diagnostic value to these patterns. But before we can say anything of value about the pathology of these patterns, a study of their behaviour in normal subjects is urgently necessary. As far as we know, up to now no extensive study has been published about this subject. The difficulty is, however, that under physiological circumstances a nystagmus does not exist. We shall have to use a provoked nystagmus e.g. a caloric stimulation.

The caloric vestibular test is the only method which gives us an impression of the irritability of a single vestibular organ. The mechanism of this method of investigation was described and discussed in detail by Jongkees (1948). The electronystagmographic (E.N.G.) method of investigation by which eyemovements are recorded makes an accurate quantitative analysis of the caloric test possible, the main advantage being that a permanent record is obtained. In the routine caloric test recorded by means of E.N.G. one generally calculates the frequency and duration of the nystagmus as well as the maximum velocity of the slow phase. Beside these parameters of the caloric test we have amongst others: total amplitude, "Wirkungszahl" (Mittermayer and Christian 1954), mean amplitude, mean velocity of the slow phase, maximum intensity (Stahle 1956).

Beside the above-mentioned parameters which indicate quantitative differences, there also are qualitative differences (nystagmus pattern) observed in the readings obtained by the use of E.N.G. which we are able to study accurately. The nystagmus pattern is characterized by irregularities which concern the form of separate nystagmus beats as well as periodical changes during nystagmus. The aim of our investigation is to study the caloric-nystagmus pattern in normal subjects, and how this is influenced by various states of mental activity.

Review of the literature

Much progress has been made in the techniques of recording eyemovements. The first attempts to do this were described by Berlin (1891), Högyes (1899), Buys (1909), Ohm (1914, 1916), Struycken (1918), Dohlman (1925) and Kuilman (1934).

The techniques of recording described over the years can be classified as follows (Gabersek, Aboulker, Pialoux and Laurent 1963):

1. mechanical recording techniques:

   a. the semi-mechanical method of Ahrens (1891) and Orschansky (1899) whereby the reflected light of a beam is observed with a mirror placed on the cornea, and the method of Lamare (1892) who used a technique making eyemovements audible.
   b. direct transmission by means of a system of levers, used by Berlin (1891) and Ahrens (1891).
   c. pneumatic transmission by which one records eyemovements using pressure-differences in a cylinder placed on the surface of the eye (Topolanski 1898).
2. strobography and cinematography: one differentiates between the indirect method of the reflected beam which is recorded on a moving photographic plate (Dodge and Cline 1901) and the direct photo- or cinematography of a white spot placed on the iris (Judd, Mac Allister and Steele 1905), or of a mirror placed on the sclera (Adler and Fliegelman 1934) or of the pupil (Kunz and Ohm 1915, 1917).
3. photo-electronystagmography (Peng): reflected infra-red rays are recorded by photo-electric cells (Dohlman 1936, Torok, Guillemin and Barnothy 1951).
4. recording of biological potential differences:
   a. electromyography (Björk 1952) for which method electrodes are placed in the extrinsic eyemuscles).
   b. electronystagmography (Schot 1922, Meyers 1929, Mowrer, Ruch and Miller 1935): the method most widely used whereby one measures the variations in the electric field surrounding the eye, induced by the corneo-retinal potential difference.

In the beginning phases of E.N.G., investigators occupied themselves primarely with the study of the quantitative parameters and in particular with the determination of normal values of these parameters in normal subjects (Aschan 1956, Hamersma 1957, Maas 1960 and Jongkees and Philipszoon 1964). A basic standard technique for the caloric test had already been established (Thornval 1917, Fitzgerald and Hallpike 1942, Hamersma 1957). We made use of the above mentioned technique in the following way:

> We alternatively syringed both ears for 30 seconds at a time with 250 cc of water at 7 centigrades above and 7 centigrades below body temperature on a subject, lying in the supine position, the head flexed forward over an angle of 30°. Between each irrigation and the next one there must be a 20 minute interval in order to avoid interference with a previous reaction (Jongkees 1949, Aschan 1956, Hamersma 1957, Oosterveld 1968).

There is still a controversy as to whether one should perform the test in complete darkness with open eyes (Henriksson 1969) or with closed eyes (Aschan e.a. 1956, Anderson e.a. 1958, Jongkees 1960). The majority of investigators give preference to the last method. Although much attention has been paid to standardization of the quantitative parameters, it is remarkable how little attention has been paid to the qualitative differences. Even before the application of E.N.G. it was noticed that a caloric nys-

authors	example	denomination	signification
Aschan, Bergstedt, Stahle 1956		dysrhythmia	cerebral lesion
McLay, Madigan, Ormerod 1957		intermittent suppression	
Riesco-MacClure, Stroud 1960		dysrhythmia	disturbance of the cerebello-vestibular interrelation
Bertrand, Montreuil 1966		dysrhythmia and inhibition	cranial trauma
Aboulker, Laurent 1967		pauses or inhibitions	cranial trauma and arteriosclerosis

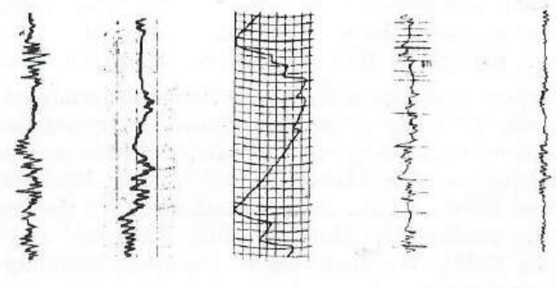


Fig. 1.

tagmus did not only show variable quantitative results but also qualitative differences were observed (Leidler 1939). Also remarkable is the different nomenclature of certain qualitative variations mentioned in literature, as well as their clinical importance (fig. 1: see page 12).

Before we had modern E.N.G. apparatus Spiegel and Aronson (1933) described a phenomenon they called pauses. By means of a kymograph they recorded the eyemovements of a cat kept under superficial ether anaesthesia and saw that the nystagmus was interrupted by periods of ocular rest. These pauses only appeared during continuous stimulation of the labyrinth.
Leidler (1939) evaluated the caloric nystagmus with the help of a pair of dark glasses. In patients with brain lesions, he found atypical forms of vestibular nystagmus due to a disturbance of the rhythm, such as:

1. predomination of the slow phase:
   a. the nystagmus occurs with the eyes in the midline axis, but the fast phase is slowed down and persists for several seconds.
   b. after a latent period a conjugated deviation of the eyes appears in the direction of the slow phase. During the reaction the eyes remain in this position and exhibit a nystagmus of small amplitude.
   c. at first there is a typical nystagmus followed by a deviation of the eyes in the direction of the slow phase.

2. rhythm disturbances:
   a. the nystagmus is irregular and may appear in periods interrupted by periods of rest.
   b. mostly there is a nystagmus of four to five beats (sometimes arhythmical) of small amplitude and at a frequency so fast as to make differentiation between fast and slow phase extremely difficult.

Kristensen and Zilstorff-Pedersen (1953) recorded — by means of optic nystagmography — broad deflections in an otherwise normal caloric nystagmus.
Christian (1954) established that every nystagmographic recording is characteristic for a certain individual. As points of identification he described the tremor of the eyes, "Augenunruhe" or eye unrest, as well as the degree of regularity, the periodic rhythm of the nystagmus, and the appearance of pauses.
In patients with headinjury Montandon (1956) described irregularities of the nystagmus during the liminal rotation-chair test. The irregularities are characterized by a dysharmony of amplitude and frequency. The most typical example of this dysharmony is the absence of a decrease in size of these parameters during increasing supra-liminal values.
Rossberg (1956) described a caloric nystagmus which was interrupted by pauses of approximately the same duration.
The term dysrhythmia was used by Aschan, Bergstedt, and Stahle (1956). They described a caloric nystagmus interrupted by periods of complete rest or pauses, and considered this dysrhythmia to be extremely

rare in normals and in patients with peripheral nerve lesions. They described this phenomenon especially in cases of post-concussional syndrome, brain lesions, and in patients with an abnormal electroencephalogram.
Stahle (1956, 1958) defined dysrhythmia as alternating periods of high and low intensity during which nystagmus is occasionally interrupted for several seconds. He observed it too in normal individuals, but only as a result of the first syringing. Striking dysrhythmia was seen in patients with a central lesion.
An intermittent nystagmus was described by Mahoney, Harlan, and Bickford (1957) who found in ten of their eighteen normal subjects submitted to the caloric test a nystagmus occurring in salvos and interrupted by periods of inhibition.
In his thesis Hamersma (1957) described dysrhythmia during the caloric nystagmus as alternating periods of high and low intensity, the nystagmus being sometimes completely interrupted.
McLay, Madigan, and Ormerod (1957) pointed out that the occurrence of an intermittent rhythm can be mistaken for an early cessation of the caloric nystagmus.
Riesco-MacClure and Stroud (1960) and Riesco-MacClure (1964) differentiated between inhibition and dysrhythmia. Inhibition is a suppression of nystagmus that can be overcome by distracting the patients attention. Dysrhythmia is an irregular nystagmus, in which large oscillations alternate with small ones, fast frequency with slow frequency and in which periods of nystagmus-jerks sometimes alternate with periods of rest. Persistent dysrhythmia in a patient with the eyes open is due to a disturbance of the cerebello-vestibular interrelationship.
Suzuki and Totsuka (1960) studied post-rotatory nystagmus and recorded an irregular pattern both by using weak stimulations and at the end of strong stimulations.
Neveling and Poeck (1960) described intermittent fast phases in the Wallenberg syndrome. In these cases the fast phase was interrupted by periods of ocular rest.
Hennebert (1960) observed nystagmus beats separated by periods of ocular rest; sometimes the nystagmus beats appeared in salvos with very high frequency.
Dysrhythmia was also defined by Lidvall (1961) as periods of distinct nystagmus beats alternating with periods of less distinct or indiscernable beats.
Megighian and Waldecker (1961) found nystagmus pauses in normal individuals.
In a publication on per- and post-rotatory nystagmus in patients with head-lesions. Montandon and Dittrich (1962) distinguished various changes in the nystagmus which they considered to be of purely central origin:

1. dysrhythmia or a nystagmus of small amplitude.
2. periodic dysrhythmia or a temporary increasing of the frequency (5 to 7 beats per sec.) with minimal amplitude.

3. nystagmus arhythmia: atypical aperiodical oculo-vestibular reactions on which a pure vestibular biphasic nystagmus is superimposed.
4. blockade: nystagmus of a very small amplitude (smaller than $1°$/sec.) which sometimes even disappears. This form is induced by an inhibition of the central vestibular pathways.
5. hypermetria is a nystagmus of large or very large amplitude.

Fernandez and Fredrickson (1963) found a prolongated caloric nystagmus after removing the nodulus cerebelli in cats. Electrical stimulation of these centres produced an inhibitory effect on the nystagmus. In a second experiment (1964) these authors destroyed the midline structures of the brainstem by radiation using radio-active palladium needles. They found a dysrhythmia during the caloric reaction as described by Riesco-MacClure.
With the aid of photo-electronystagmography Gulick and Pfaltz (1964) demonstrated that 30 % of the cortical and brainstem lesions showed abnormal recordings, characterized by multiple culminations, and in extreme cases by bursts of nystagmus.
Mehra (1964) described dysrhythmia as a periodic cessation of nystagmus beats from 5 to 10 seconds.
Bodo (1965) found irregularities in amplitude and frequency during the caloric reaction in cases of central vestibular disease.
In a publication from 1965 Pfaltz stated that he was not able to confirm the findings of Riesco-MacClure on dysrhythmia in cases of cerebellar lesions.
In 1963 Montandon described the qualitative aspect of the nystagmogram. Some pathological conditions create irregularities of the nystagmus in the form of inhibitions, tachynystagmic salvos or desorganisation of the pattern. He attached great diagnostic value to these irregularities and found them in central syndromes, especially in lesions of the central vestibular pathways (mostly not associated with hearing lesions) such as:

1. subtentorial lesions e.g. syringobulbia, Wallenberg syndrome, syndrome of the vertebral artery, tumours, cerebellar syndrome.
2. supratentorial lesions e.g. Parkinsonism, lesions of the basal nuclei.

Gabersek and Jobert (1965) described positional nystagmus to the right in a patient, showing salvos interrupted by many pauses, which were even more striking during a caloric stimulation inducing a nystagmus to the right.
Aubry, Pialoux and Chouard (1965) described various qualitative phenomena, such as:

a. disturbance of the rhythm: either a regular or irregular nystagmus interrupted by pauses.
b. abnormal configurations: "petite écriture" (i.e. a nystagmus of small amplitude) and "crochetage" (i.e. a nystagmus with a dentation of the slow phase).

They found that dysrhythmia accompanied by salvos was not characteristic of peripheral lesions. They considered paradoxal inversion at the end of the caloric reaction to be typical for tumours of the fourth ventricle and "crochetage" to point to cerebellar lesions (cerebellar dysrhythmia).

Dysrhythmia was described by Sokolovski (1966) as an irregular nystagmus made up of short intervals of regular nystagmus which are replaced by longer periods of either nystagmus-free intervals or distorted nystagmus.

Greiner, Conraux, Collard, Picart, and Riberolles (1966) described various nystagmus patterns induced by torsion swing stimulation in patients with cranial trauma. They found:

a. irregular aspect:
   1. salvos or bursts of fast and small nystagmus beats.
   2. inhibitions or intervals lasting a few seconds during which nystagmus stops.
   3. facilitation or reappearance of the nystagmus at a larger amplitude.
b. increased frequency: "petite écriture" i.e. frequent nystagmus beats of small amplitude (3 to 5 beats/sec.).

Two forms of dysrhythmia were described by Kornhuber (1966). Fatigue dysrhythmia, interrupted by arousal techniques, and a dysrhythmia induced by a disturbance of the fast phase due to central lesions. The latter occurs in spite of arousal.

Bertrand and Montreuil (1966) described dysrhythmia and inhibitions in 60 % of the cases of patients after cranial trauma. Both disturbances occurred in 47 % of normal subjects and in cases of peripheral lesions.

Dufour, Fellitti, Lazzaroni, and Zibordi (1966) described qualitative variations of the caloric nystagmus in cases of central lesions at the bulbo-pontine level. They found rounding of the apex of the nystagmus beat, hook forming (dentation) in slow and fast phase and a predomination of the slow phase.

Maran (1966) using a minimal stimulation test found during calorisation a dysrhythmia consisting of large oscillations alternating with small oscillations, and a high frequency alternating with a low frequency.

Mounier-Kuhn, Morgon, and Achache (1966) described qualitative changes, especially during the torsion-swing test and the liminal rotation-chair test. Their classification is as follows:

1. comparison of frequency and amplitude
   a. small amplitude, high frequency: this is the "petite écriture" of Montandon.
   b. small amplitude, low frequency; hyporeflexia.
   c. large amplitude, high frequency.
   d. large amplitude, low frequency.
2. regularity of the nystagmus pattern with regard to:
   a. rhythm: dysrhythmia, arythmia.

   b. variations in amplitude.
   c. salvos or "bouffées tachynystagmiques"; periods of inhibitions or pauses.

Coats (1966) observed subjects who suppressed the caloric nystagmus either continuously or periodically causing nystagmus bursts to alternate with silent periods.

Bertrand and Arbour (1966) defined dysrhythmia as variations of both the frequency and amplitude, interrupted by periods of total inhibition. Such a dysrhythmical response accompanied by intermittent blinking of the eyelids is pathognomic for the post-concussional syndrome.

In patients with central lesions Cavaller and Capella (1966) described pauses during caloric nystagmus.

In 1966 Karbowski compared the regularity of the tracings between electroencephalography and electronystagmography. He established that in cases of an irregular E.E.G., the E.N.G. is also irregular.

Montandon (1967) points to the fact that in head injury the occurrence of an irregular rhythm, tachynystagmic salvos, pauses, and desorganization of the pattern are typical qualitative findings.

Dysrhythmia was described by Pialoux, Chouard, and Fontelle (1967) as a sign of cerebellar compression caused by a neurinoma. According to them the differential diagnosis between 8th-nerve neuritis and advanced Ménière's disease is:

In neurinitis after the irrigation of the healthy ear pauses occur more often while "crochetage" is rarely seen. In advanced Ménière's disease they found a decreased reaction with sometimes "petite écriture" and in most cases "crochetage", especially in the first half of the reaction after irrigation of the healthy ear.

Aschan (1967) drew attention to the importance of the arousal technique in dysrhythmia. He often observed dysrhythmia in patients with a disturbed state of wakefullness due to brain tumours or drugs.

In 1967 Barber and Wright described various nystagmus patterns:

1. habituation: response decline due to repeated stimulation.
2. dysrhythmia: appearance of nystagmus-beats of varying amplitude and frequency.
3. suppression: unexpected absence of nystagmus reappearing if arousal techniques are used.

It is interesting to notice that the authors mentioned the occurrence of suppression in cases of vertigo, but the pathological value of this symptom could not be estimated as they did not use appropriate arousal techniques.

Two types of dysrhythmia were described by Tokunaga (1967). Firstly dysrhythmia, characterized by nystagmus of irregular amplitude and frequency, typical for supra-tentorial lesions; and a second type, characterized by a regular nystagmus, interrupted by pauses, pointing to an infratentorial lesion. Cerebellar lesions showed both types.

An extensive classification of nystagmus patterns in patients with cranial lesions was given by Aboulker and Laurent (1967). They described pauses or inhibitions i.e. suppression of every nystagmus beat during a short or longer period, and saw nystagmus in salvos when longer pauses were present. If one wishes to consider them real pauses, one should be sure that they are not induced, e.g. by vertical eyemovements or surrounding noises.

A vertical nystagmus with the same rhythm as the horizontal component preceded by this for a fraction of a second can be found. Sometimes the vertical component persists longer than the horizontal one.

"Petite écriture" is a nystagmus with frequent beats of small amplitude. During real salvos, due to the fast rhythm, the pen does not return to the baseline. In this instance a short timeconstant of the apparatus prevents the recording of the true position of the eyes and makes a differentiation between these two phenomena impossible.

The authors describe "crochetage" as a bifide nystagmus beat, in which the second apex is of smaller amplitude than the first.

According to the authors a nystagmus basculans or Kipp-nystagmus is a nystagmus with beats alternating in opposite directions.

Sometimes the authors observed an inversion of the nystagmus during one or more calorizations, though there was no trace of spontaneous nystagmus.

In these cases of cranial lesions blinking frequently occurred.

In 1967 Gabersek and Scherrer described the appearance of pauses and irregular nystagmus patterns as typical symptoms of a central lesion.

Oosterveld and van Vliet (1968) described aberrant forms of the caloric nystagmus in patients with cranial lesions e.g. dysrhythmia, i.e. nystagmus interrupted by periods of rest; dysmetria, i.e. a nystagmus with a large amplitude; "petite écriture", i.e. a nystagmus with very small amplitude; "crochetage", i.e. a nystagmus beat in which both forms of the amplitude (the small and the large) occur.

In a neuro-otological study on brain injured ex-servicemen Kirjavainen (1968) found in 43 % of the cases an irregular nystagmus and sometimes a dysrhythmia or nystagmus with pauses during caloric nystagmus.

Pulec (1968) defined dysrhythmia as nystagmus with an irregular rhythm and periods of no eyemovement, often seen in patients with a history suggestive of brainstem ischemia. According to this author apparent dysrhythmia can occur if the patient is not alert.

Henriksson, Janeke, and Claussen (1969) described irregularities during the caloric test in patients with central disturbances. They noticed dysrhythmic patterns or an irregular nystagmus due to disturbance of the mechanism that triggers off the fast phase, and a deviation in the direction of the slow phase, due to incomplete abolishment of the fast phase.

McNally (1969) observed dysrhythmia and points out that possibly dysrhythmia can be induced by "a faulty technique in carrying out the test".

"Petit écriture", dysrhythmia, "crochetage" and groupformation in caloric nystagmus are mentioned by Jongkees (1969).

A detailed description of nystagmus forms during the torsion swing test is given by van de Calseyde, Ampe, and Depondt (1969).

According to these authors the variations are characterized by a disturbance of the frequency-amplitude ratio.

They recorded two types of tracings. The first a regular nystagmus of low frequency and large amplitude. This type occurs particularly in normal individuals, who for 80—90 % were between ten and fifty years old. Secondly, a nystagmus demonstrating successive variations in frequency and still greater successive variations in amplitude. This type occurs particularly in children under ten and adults over fifty. In the latter group they found an added central component such as disturbance of the regularity by temporary inhibitions, salvos, and blinking.

In patients with central lesions they found various types of nystagmus patterns, such as: "petite écriture"; inhibition or nystagmus suppression lasting for a few seconds, due to a blockade of the fast phase. This blockade is caused by an inhibition of the vestibular function or for lack of alertness. Dysrhythmia is bursts of nystagmus of varying frequency. This is found especially in cerebellar tumours with compression of the fourth ventricle or in children under ten. The authors observed salvos i.e. bursts of high frequency which can be made up of a succession of slow and fast phases, or a slow phase of large amplitude returning stepwise. Kipp-nystagmus consists of two opposite fast phases separated by a short period of ocular rest and occurs especially in the vicinity of the threshold.

Different nystagmus patterns

We have found ourselves obliged to define each nystagmus pattern and give it a suitable name. Since each author uses his own nomenclature, as can be seen in our literature review, there is a great deal of confusion in the naming of the different nystagmus patterns:

1. during caloric nystagmus the appearance of periods without nystagmus is a phenomenon of frequent occurrence, already described by Leidler (1939). For this reason it has been given the most divergent names e.g.

   pauses: Spiegel e.a.; Christian; Rossberg; Aschan e.a.; Riesco-MacClure e.a.; Hennebert; Megighian e.a.; Gabersek e.a.; Aubry e.a.; Cavella e.a.; Montandon; Mounier-Kuhn e.a.; Aboulker e.a., and Pialoux e.a.

   dysrhythmia: Aschan e.a.; Stahle; Lidvall; Kirjavainen; Pulec, and Jongkees.

   intermittent nystagmus: Mahoney e.a.; Riesco-MacClure; Fernandez; Montandon; Greiner e.a.; Mounier-Kuhn e.a.; Aboulker e.a., and van de Calseyde e.a.

   suppression: Coats; Barber e.a.; van de Calseyde e.a.

   blockade: Montandon e.a.

   The best way to indicate a phenomenon is to choose a name which describes the phenomenon as accurately as possible. Since it concerns periods without nystagmus we prefered the name *pause*. Pause indicates the absence of nystagmus. It is usually combined with ocular rest. During this pause there is no fast phase. The fast phase determines the rhythm, in other words the terms dysrhythmia and intermittent rhythm do not describe the pause itself but place it inside the entire caloric reaction as an alternating occurrence of active periods consisting of nystagmus and periods without nystagmus. The words inhibition, blockade, and suppression already try to explain the phenomenon or presume a central influence. We consider these names unsuitable because the phenomenon still lacks a definite explanation. Intermittent nystagmus also involves the entire caloric reaction.

2. Leidler (1939) had already described nystagmus that is not always regular. The term dysrhythmia is mostly used to describe this irregular pattern.
This is a term derived from E.E.G. In E.N.G. it was described by Riesco-MacClure e.a.; Fredrickson e.a.; Aubry e.a.; Pflatz; Sokolovski; Bertrand e.a.; Maran; Mounier-Kuhn; Barber e.a.; Tokunaga; Pialoux e.a.; Pulec; van de Calseyde e.a.; Henriksson e.a.
We are not content with the term, because it is used in order to cover every irregularity of caloric nystagmus and as such forms a collective term that makes a detailed study difficult. For this reason we see in literature so many different syndromes attached to this name. We will therefore use *dysrhythmia* as a collective term to include all irregularities of rhythm and amplitude.

3. Montandon named an atypical aperiodic oculo-vestibular reaction on which a nystagmus is superimposed, *arhythmia*. We will use the term arhythmia to describe an eyemovement on which nystagmus-beats are superimposed, but with no recognizable rhythm. It was also described by Mounier-Kuhn e.a.

4. *Intermittent fast phase* we call a nystagmus with a fast phase interrupted by short pauses (Neveling e.a.; Dufour e.a.).

5. Montandon also described a nystagmus of very large amplitude and named it hypermetria. Oosterveld e.a. and Jongkees discussed this phenomenon as *dysmetria*. This is a neurological term meaning a badly adjusted movement (dysmetria) usually excessive (hypermetria) (Biemond 1961). In E.N.G. we shall use the term to describe a nystagmus-beat with large amplitude. The phenomenon was already noticed by Christensen and Zilstorff-Pedersen in 1953.

6. A nystagmus of small amplitude and high frequency was described by Montandon as dysrhythmia, later as periodic dysrhythmia and finally as "*petite écriture*". This name is also used by Aubry e.a.; Greiner e.a.; Pialoux e.a.; Oosterveld e.a., and van de Calseyde e.a. and ourselves.

7. Leidler already called the attention to a nystagmus occurring in groups, of small amplitude and high frequency. Here also different names were given:
tachynystagmic salvos: Montandon; Greiner e.a.; van de Calseyde e.a.
salvos: Mounier-Kuhn e.a.; Gabersek; Aubry e.a.; van de Calseyde e.a.
groupformation: Jongkees.

Aboulker e.a. also used the name salvos, but observed that the pen of the nystagmograph does not return to the base-line as long as the salvo lasts. We will use *salvo* as defined by the last author.

8. A nystagmus beat with multifid top is described by Dufour e.a. as dentated nystagmus and by Aubry e.a.; Oosterveld e.a., and Jongkees as "*crochetage*". We shall use this term to describe a bifid nystagmus-beat.

9. Inversion (Aubry e.a.; Aboulker e.a.) is the changing of direction of the nystagmus. If this inversion occurs at the end of the reaction we shall call it *secondary-phase nystagmus*.

CHAPTER II

INVESTIGATION

A. The caloric test without arousal

1. *Method of investigation*

A nystagmus can be provoked in different ways. We prefer the caloric method, because it gives a prolonged nystagmus mainly in one plane and in one direction, of vestibular origin, and easy to repeat.
The irrigation is not too unpleasant. The technique of irrigation we used has already been described (see page no. 11). The choice of the normal subjects used in our investigation had to conform with the following criteria:

a. in good physical health i.e. a case history free of pathology, especially concerning their ears. They may not have had vertigo-complaints in their history.
b. between 15 and 30 years old, since deviations appear in younger and older people (van de Calseyde e.a. 1969).
c. both sexes had to be investigated.
d. no pathological spontaneous or positional nystagmus.
e. no pathological labyrinth predominance or directional preponderance.
f. during the test the subject must be awake but resting (alpha-rhythm must be present in the E.E.G.).
g. no use of drugs or alcohol for at least 24 hrs. preceding the tests.
h. normal audiological findings.

We used 17 subjects who complied with the above mentioned criteria. By irrigating each subject four times, we obtained 68 recordings.
Beside the horizontal lead, the "derived nystagmogram" (described by Henriksson; see page 45) of the horizontal eyemovements, a monocular vertical lead of the right eye, and a simple E.E.G. lead were recorded. Since our purpose was not to perform a detailed E.E.G. investigation and since we were primarily interested in the alpha-rhythm, we used an antero-posterior bipolar lead between p4 and f4 (Jasper), i.e. on the side of the non dominant hemisphere (Hootsmans 1961), as all our subjects were right-handed.
For the E.E.G. we used the same electrodes as for the E.N.G., i.e. hollow metal plates with a diameter of half a centimeter. After cleaning the skin we attached the electrodes with collodion, this in order to prevent shifting during the test. After putting the electrodes in the right place, contact substance is injected into the electrode through a hole.
Between two irrigations we waited at least twenty minutes. in order to exclude influences of the preceding calorization. During this period the patients were given a break and allowed to leave the dark room in order to avoid drowsiness. To correct possible variations in the corneo-retinal potential difference (Hart 1969), the eyemovements were calibrated before each irrigation. During calibration of the horizontal eyemovements

one must see to it that practically no vertical component appears and vise versa, as this would indicate that the electrodes are wrongly attached. In order to maintain an unchanged state of alertness during the caloric reaction all noise is avoided as far as possible.
Although the subject is lying in another room, we cannot completely avoid that he hears noises coming from the waterpumps and the electronystagmograph.
We found this noise to consist of the following octave frequencies: at 63Hz 54dB; 125Hz 49 dB; at 500Hz 41 dB; at 1000Hz 35 dB; 2000Hz 28 dB; at 4000Hz 25 dB and at 8000Hz 25 dB.
The whole experiment was performed in an anechoic bunker in order to eliminate noise from the outside.
If something went wrong with an irrigation it was repeated twenty minutes after the fourth.

2. *Working out of the recordings*

Each nystagmogram was first worked out quantitatively. We determined the maximum speed of the slow phase and the moment it occurs in the reaction. Also the total duration of every reaction was measured, i.e. the time between the first and the last nystagmus-beat.
Square waves, secondary-phase nystagmus, and counter-jerks were not calculated in the duration of the reaction (Hamersma 1957). The sizes of both labyrinth predominances and directional preponderances were calculated by Jongkees's formula (Jongkees and Philipszoon 1964) (table I). The number of nystagmus-jerks or the frequency and the total

Table I.

Jongkees's formula

A left cold irrigation — (1) — elicits a nystagmus to the right.
A right cold irrigation — (2) — elicits a nystagmus to the left.
A left hot irrigation — (3) — elicits a nystagmus to the left.
A right hot irrigation — (4) — elicits a nystagmus to the right.

Labyrinth predominance in per cent is

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

Directional preponderance in per cent is

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

amplitude were only measured during the culmination period (Torok 1957, 1961, Pfaltz and Gulick 1962). According to Torok, this period lies between the 25th and 35th second, according to Pfaltz, between the 60th and 100th. This difference is due to a difference in stimulation techniques. In a period of 5 seconds Torok injected 10 cc of water into the external auditory canal; Pfaltz injected 250 cc in 30 seconds.
We used the latter technique. A culmination point being between the 60th and 120th second tallies with the findings of Cawthorne and Cobb

(1954) who, before they destroyed the diseased labyrinth and after calorization, recorded the variations in temperature with a probe in the interior of the lateral semi-circular canal in patients suffering from Ménière's disease.
They found that — by syringing the external auditory canal during 40 seconds — the maximum difference in temperature occurred after approximately 100 seconds.
Afterwards we studied the qualitative properties of each recording and searched for possible relations with the quantitative findings.

3. *Results*

I. QUANTITATIVE FINDINGS

According to Maas (1960) discussing the maximum speed of the slow phase, a variation in the labyrinthine irritability greater than 15 % is pathological as well as a variation in the directional preponderance greater than 18 %, in the Amsterdam population examined under our conditions.
When the right labyrinth is less excitable than the left one, the formula proposed by Jongkees (table I) gives a positive value, in the reverse case a negative value is the result.
In case of a directional preponderance to the right we find a positive value and a negative value in case of directional preponderance to the left. According to these criteria (see table II, page 25) subject no. 2 would show a directional preponderance to the left (—30 %), subject no. 8 a directional preponderance to the left (—35 %), and a less irritable right labyrinth, or a labyrinthine predominance to the left (+ 41 %) subject no. 9 a pathologically decreased irritability of the left labyrinth (—25 %); subject no. 12 a directional preponderance to the left (—25 %); and subject no. 17 a decreased irritability of the left labyrinth (—21 %) (see table II). Later it will appear that this apparent pathology was due to lack of arousal.
We did not consider the labyrinthine predominance and nystagmus preponderance by means of the total duration, the frequency during the culmination period, and the total amplitude during the same period.
In the thesis of Hamersma it appears that the duration is not the best parameter for the evaluation of the caloric reaction.
Van Egmond and Tolk (1954), Aschan e.a. (1955), Henriksson (1955) and Jongkees e.a. (1964) determined that the speed of the slow phase is the best parameter to measure the cupula activity.
Figure 2 (see page 26) shows the beginning and the end of the nystagmus during each calorization. It follows from figure 3 (see page 26), that, without arousal, a nystagmus can always be demonstrated in every reaction between 60 and 120 seconds after the beginning of the irrigation. There is one irrigation which does not show a reaction in the period between 30 and 60 seconds and two irrigations which do not show a reaction in the period between 120 and 150 seconds.

Table II.

	without arousal					with arousal					presence of a non pathological spontaneous nystagmus		presence of spontaneous		
	la pr		di pr		SPN	la pr		di pr		SPN					
No S	ss	d	ss	d		ss	d	ss	d		ho ny	ve ny	pe mo	je	P.O.S.
1	— 5%	+ 5%	— 5%	—12%	—	+ 8%	+ 4%	—15%	+10%	—	—	—	—	—	+
2	+12%	+ 3%	—30%	—22%	—	+ 9%	—12%	— 9%	—28%	+	+	+	—	—	—
3	+ 9%	+ 6%	— 9%	+ 1%	—	+ 5%	+17%	— 4%	+22%	+	+	—	—	+	—
4	— 5%	+ 6%	+ 5%	+ 4%	+	—12%	+25%	— 7%	+16%	+	—	—	—	+	—
5	+12%	0%	— 3%	+26%	—	+ 6%	+ 3%	+ 8%	+ 2%	—	—	—	+	—	—
6	+ 8%	+ 7%	+ 2%	— 3%	—	— 2%	+ 7%	— 2%	— 1%	+	—	+	—	—	—
7	+10%	— 4%	— 3%	0%	+	— 7%	— 7%	— 7%	— 8%	+	—	+	—	—	—
8	+41%	— 3%	—35%	—31%	+	+10%	+ 2%	—15%	— 4%	+	—	—	+	—	—
9	—25%	—14%	—13%	+26%	—	— 1%	—14%	+10%	— 1%	+	—	—	—	—	—
10	+10%	+19%	—10%	+14%	—	— 2%	+ 7%	— 8%	+ 2%	—	—	—	—	—	—
11	0%	+ 9%	0%	—38%	—	+ 3%	— 1%	—13%	—21%	+	—	—	—	—	—
12	+12%	+ 3%	—25%	+29%	—	0%	+ 4%	—11%	+43%	—	—	+	+	—	—
13	+13%	0%	0%	+ 8%	+	+ 8%	+13%	— 4%	+ 3%	+	—	+	—	+	—
14	+ 4%	— 7%	— 4%	+10%	+	—14%	— 3%	+ 1%	+20%	—	+	—	—	—	—
15	— 5%	+ 1%	— 5%	+ 4%	+	+ 5%	+ 5%	— 1%	+21%	+	—	—	—	—	—
16	+ 1%	+10%	— 1%	—12%	+	— 8%	— 4%	— 7%	—12%	+	—	—	—	—	+
17	—21%	0%	—18%	+ 2%	+	+ 3%	+ 6%	— 1%	— 6%	+	—	+	—	—	—

Table II.

Abbreviations:

No S : the number of the test subject.
la pr : labyrinth predominance.
di pr : directional preponderance.
ss : preponderance in the speed of the slow phase.
d : preponderance in the duration.
ho ny : horizontal nystagmus.
ve ny : vertical nystagmus.
pe mo : pendular movements.
je : jerks (amplitude of 10°-15°).
P.O.S. : paroxysmal ocular state.
S.P.N. : secondary phase nystagmus.

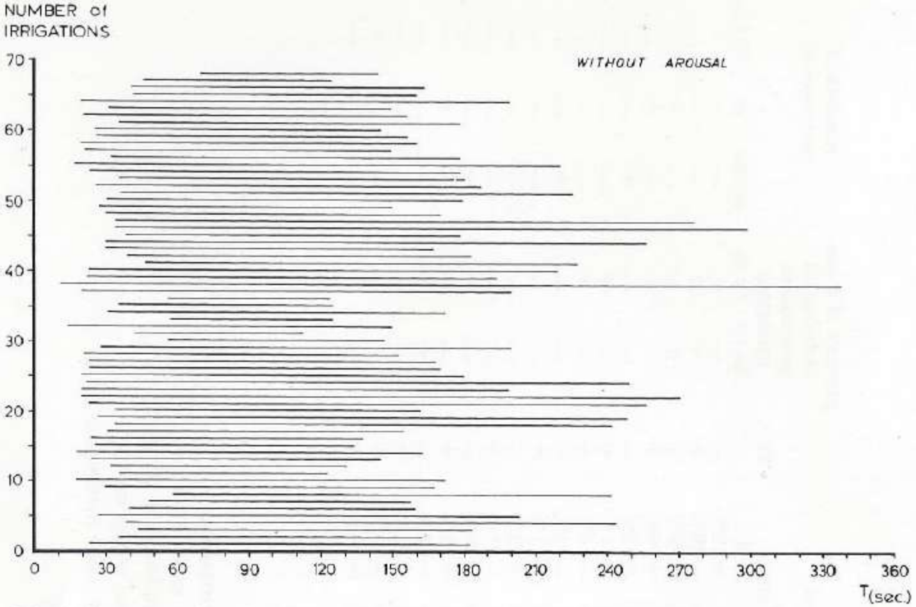


Fig. 2. Duration of each caloric reaction (17 × 4 = 68).

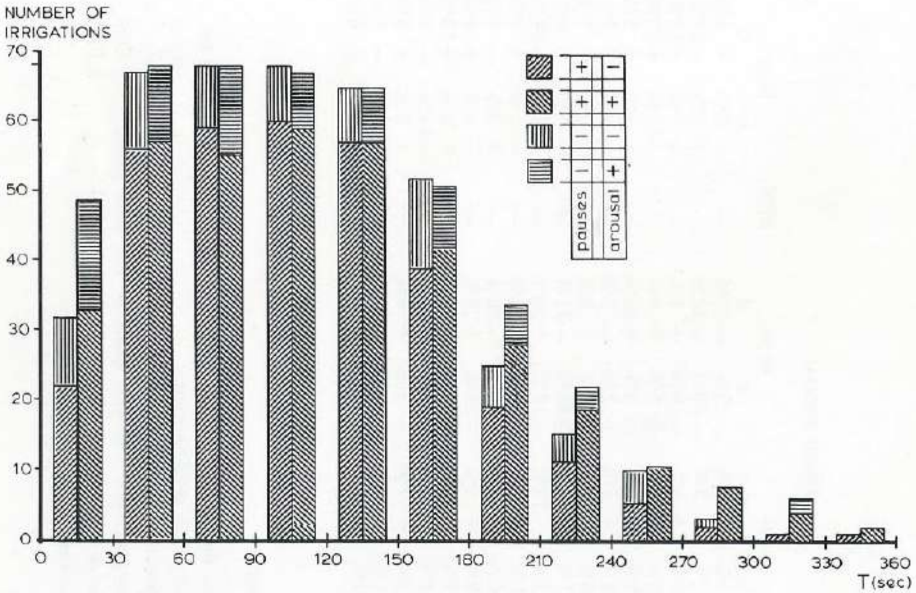


Fig. 3. Number of irrigations still showing a nystagmus for different periods of 30 seconds.

II. QUALITATIVE FINDINGS

A. Dysrhythmia

As stated before, we shall use the term dysrhythmia for every rhythm-deviation occurring during caloric nystagmus.
If we apply the definition of Riesco-MacClure, we find evidence of dysrhythmia as pathological. Only few describe it in normal subjects and others in normal as well as in pathological cases.
If we apply the definition of Riesco MacClure, we find evidence of dysrhythmia in each recording from our normal subjects.
We therefore consider it premature to make a diagnosis or even to accept pathology on the basis of dysrhythmia. In fact an E.N.G. is not an E.E.G. or an E.C.G. By means of E.E.G. we measure the electric activity of the brain and with E.C.G. the electric activity of the heart. In E.N.G. we only measure one of the responses of a labyrinth: the vestibulo-ocular reflex or nystagmus. There are many more effects e.g. vestibulo-spinal reflexes, turning sensation, and vegetative reactions (Bárány and Wittmaack 1911). Therefore we agree with the conclusion of Kornhuber (1966): "An isolated dysrhythmia has to be assessed with restraint. I do not remember having seen a case in which dysrhythmia was conclusive for a diagnosis".
It may be preferable to consider dysrhythmia as a certain irregularity characteristic for each individual (Christian 1954).
Stahle (1958) noticed that marked degrees of dysrhythmia mostly accompanied with directional preponderance should probably be regarded as an expression of a disturbance in the central part of the vestibular apparatus. The presence of directional preponderance (in the velocity of the slow phase) would further support this assumption. However, the opinions concerning the causes of directional preponderance are very divergent (Jongkees 1969). In the first series (caloric test without arousal) we found four subjects with a pathological directional preponderance in the velocity of the slow phase (see table II, no. 2-8-12-17) and indeed all of them showed some dysrhythmia (Fig. A1). *)
During the second series (caloric test with arousal) the dysrhythmia persisted (Fig. A2), but the nystagmus preponderance disappeared. On the other hand we also found in our group subjects showing a dysrhythmia without directional preponderance (Fig. A7).
We found the dysrhythmia of Riesco-MacClure (1960, 1964), see page 14, in most of our recordings, but none of our subjects showed signs of an invasive process of the cerebello-vestibular pathways. Riesco-MacClure warned that slow pendular movements of the eye with superimposed nystagmus could look like dysrhythmia: these pendular movements can occur in natural sleep (Hamersma 1957) or after taking barbiturates (Aschan 1964). In fact, none of our subjects showed a sleeping rhythm in the E.E.G. lead, or had taken barbiturates.
Pendular movements were sometimes seen at the end of the caloric reac-

*) The "A" before the number means that the figures can be found in the appendix.

tion, but never together with superimposed nystagmus. In establishing the differential diagnosis, Riesco-MacClure instructs the patient to open the eyes when he suspects that the patient is falling asleep. If the dysrhythmia still persists, it should be a genuine dysrhythmia. However, from experience we have learned that vestibular nystagmus is suppressed when the eyes are opened (see page 11). Furthermore the recordings of Riesco-MacClure show that the speed of the slow phase is constant during dysrhythmia (Barber 1967) (see also Fig. 1). When pendular movements are superimposed on a nystagmus, the speed of the slow phase increases if the eye deviates in the direction of the slow phase and decreases if the eyes deviate in the opposite direction; in any event, the speed of the slow phase is influenced. Also Pfaltz (1965) could not bear out the theory of Riesco-MacClure that dysrhythmia indicates a tumour of the posterior cranial fossa.

Suzuki et al (1960) noticed that dysrhythmia increased at the end of a post-rotatory stimulation. This is hard to confirm with our caloric stimulation. In fact, dysrhythmia is a very complex fusion of different phenomena some of which — such as "crochetage" and groupformation — can only be seen during the culmination period, when the nystagmus is at its maximum. At the end of a caloric reaction, the frequency and the speed of the slow phase are very slow, and slow sinus-wave-like eyemovements, counter-beats etc., interfere so much with the nystagmus, that it becomes hard to distinguish the former phenomena. The irregularities occurring at the end of the nystagmus period mostly differ from those occurring during the culmination period.

Fernandez and Fredrickson (1964) obliterated the nodulus cerebelli in cats by inserting a radio-active palladium needle and found dysrhythmia, as defined by Riesco-MacClure, during the caloric nystagmus. This would prove the cerebello-vestibular interrelation theory. However, it appears from the histological studies that in all their cats not only the nodulus was obliterated, but also a part of the medial longitudinal fasciculus, the vestibular nuclei, and the reticular formation. Exactly these parts are vital to the production of the fast phase, as will be described later. Secondly it is not necessary to destroy the nodulus in cats in order to provoke dysrhythmia. Milojevic and Voots (1965) investigated nystagmic response decline in cats. In the majority they found large qualitative variations in the different recordings, and attributed them to the variations of the degree of arousal, i.e. one cat was asleep and the other was very active.

Tokunaga (1967) differentiated two types of dysrhythmia (see literature review). The first type was said to be typical for supratentorial lesions and the second type for infra-tentorial lesions. As we have already pointed out, both types also occur in normal subjects. Furthermore Kornhuber's statement should be considered here (see page 27).

In addition to the E.N.G., Karbowski et al (1963) recorded the E.E.G., the E.C.G., arterial blood pressure, ventilation, and O_2-uptake during the caloric test in 15 subjects. In the majority, labyrinth stimulation did neither change the E.E.G. nor lead to a non-specific activation of the cortical electro-genesis. Only in two cases a temporary asymmetrical des-

organization was induced on the side of the irrigated ear. Of all the other recordings only the arterial blood pressure showed a variation, i.e. during the irrigation it increased slightly, after which it decreased by 10 % for one or two minutes.

Furthermore the author (Karbowski 1965) points out the existence of a cortical projection of the turning sensation via the reticular formation and via specific corticopetal pathways. According to Gerebtzoff (1940) the latter would run parallel to the auditory pathway, but without crossing over (trapezoid body, lateral lemniscus, corpora quadrigemina, medial geniculate body).

According to Biemond (1961), however, there are cortical projections from the vestibular apparatus going to both cerebral hemispheres respectively, the contra-lateral projection being the most important. They run in two specific tracts: the vestibulo-thalamic tract and another joining the central auditory tract.

In a publication in 1967, Karbowski compared the E.N.G. with the E.E.G. of 44 epileptics and 22 normal subjects. In most of the cases a significant similarity was found between the regularity and the irregularity of E.N.G. and E.E.G. Real dysrhythmias in the E.E.G. of our subjects did not occur. We found that there was no correlation between the occurrence of dysrhythmia in the E.N.G. and the amount of alpha-rhythm present in the E.E.G.

In almost all our subjects the alpha-rhythm disappeared during the first seconds of the irrigation. If we compare the period between 30 and 60 seconds with that after 60 seconds, we observe that during the former the alpha-rhythm is present to a smaller extend. Test subject no. 16 is a good example. She underwent the caloric test for the first time in her life. During the first irrigation the alpha-rhythm disappeared not only during the irrigation, but also during 30 seconds following it (Fig. A3). But during the culmination period the alpha-rhythm returned. In the next three irrigations, the alpha-rhythm was only suppressed during the first 5 seconds of the syringing after which it reappeared quickly (see Fig. A4).

From this we conclude that the arousal effect was maximum during the first syringing. The subject experienced stimulation of the external auditory canal by syringing with water for the first time in her life. During the next irrigations the subject had become accustomed to this experience and the alpha-rhythm was only suppressed during the first 5 seconds, this is the period with the largest P.O.S. (paroxysmal ocular state, i.e. periods of extreme vertical eyemovements). It is curious that such a strong sensory and auditory stimulus as syringing, does not bring about a "réaction d'arrêt" of the E.E.G. Furthermore we observed that the alpha-rhythm did not decrease during the culmination period. Also the turning sensation, occurring mostly after the beginning of the caloric nystagmus and disappearing before the end of the nystagmus (Jongkees 1949), seems to affect the alpha-rhythm only very slightly. Since dysrhythmia is such a complex phenomenon we have divided it into two groups:

1. periods during the caloric nystagmus in which no nystagmus occurs, i.e. the pause phenomenon.
2. irregularities occurring during the active period or periods with abnormal nystagmus. This group can be subdivided into:

   a. arhythmia
   b. intermittent fast phase
   c. abnormal size of the amplitude
   d. varying amplitude
   e. secondary-phase nystagmus

Beside dysrhythmia we shall also discuss other phenomena, such as: vertical nystagmus, counter-beats, Kipp-nystagmus, P.O.S., pendular movements, aberrant apices, and blinking.

1. Pauses or the pause phenomenon

During caloric nystagmus, pauses can occur in which no nystagmus is recognizable (Fig. 4 no. 1: see page 46). For practical purposes we use the word nystagmus, but we mean caloric nystagmus. We use the definition of nystagmus according to Janeke (1968), i.e. a rhythmical eyemovement characterized by a fast and a slow phase.

Aschan et al (1956) who used the same caloric technique, described pauses, which occurred during the culmination period (at the 80th second). Stahle (1958) found pauses especially at the end of the reaction. From Fig. 3 (see page 26) follows that in our material pauses occur during the entire caloric reaction and the number of the pause-free recordings is lowest during culmination. Only 13 % and 12 % resp. of the total number of recordings demonstrate no pauses during respectively the periods between 60 and 90 seconds and the period between 90 and 120 seconds.

Table III.

caloric nystagmus in periods of 30 seconds	mean number of pauses per 30 seconds	mean total duration of the pauses in seconds	mean duration of a pause in seconds	mean duration of the longest pause in seconds
0"—30"	1	3	—	2,5
30"—60"	4	11	3	6,5
60"—90"	5	9,5	2	4
90"—120"	5	11	2	4,7
120"—150"	4	9	2,5	4
150"—180"	3	10,5	3,5	6
180"—210"	2	10,5	5	5,5
210"—240"	2	11	5,5	8
240"—270"	2	8	4	4

For each period of 30 seconds of each recording, we calculated each pause. If we consider Fig. 3 (i.e. for each period of 30 seconds the number of recordings with occurrence of pauses) we can determine for each of these periods the mean number of pauses, the mean total duration of the pauses, and the mean duration of the longest pause (table III). From this follows that during the culmination period the mean number of pauses is at its highest, (since at the end of a reaction the short pauses can no longer be observed, an account of the very small amplitude and speed of the slow phase), and the mean duration of a pause smallest. The longest pauses occur at the end of the reaction.

In our series the assertation of Rossberg (1956) that all pauses are of approximately equal duration is not true. The shortest pause that we can record amounts to approximately the duration of a nystagmus-beat (i.e.

Table IV.

FIRST SERIES WITHOUT AROUSAL

Results of the left hot irrigation:

No S	No I	Tr	V=V°	A°	F°	a	a°	t	t°	m	m°
1	5	205	25	469	98	17	9	322	102	75	30
2	1	183	49	310	76	36	19	914	321	180	36
3	2	99	80	1852	157	—	—	—	—	—	—
4	3	114	55	642	77	16	8	565	232	139	75
5	2	128	56	570	105	23	16	343	145	70	17
6	2	227	60	1467	127	10	—	246	—	44	—
7	5	132	65	1271	181	19	10	158	50	27	7
8	1	136	100	1802	119	17	4	420	53	70	33
9	1	68	13	54	13	12	9	517	488	155	155
10	4	196	41	920	91	10	3	307	33	84	19
11	1	225	32	712	107	20	6	324	144	47	47
12	5	140	62	421	57	66	32	668	345	56	53
13	3	166	50	1338	100	17	5	191	37	25	14
14	3	146	80	634	133	44	19	575	198	60	44
15	2	120	53	504	110	11	8	132	96	27	27
16	3	180	40	445	61	20	5	813	203	159	51
17	3	75	15	11	4	6	5	716	551	173	173

Results of the right hot irrigation:

1	2	140	25	232	64	19	9	603	128	210	29
2	2	110	24	112	43	21	12	694	341	138	138
3	1	87	64	1863	156	—	—	—	—	—	—
4	4	108	66	844	100	24	12	470	177	85	45
5	1	212	45	950	159	17	—	470	—	177	—
6	1	178	55	1117	117	5	—	264	—	94	—
7	4	145	52	1125	172	25	4	345	61	42	21
8	2	71	35	96	17	13	7	562	426	124	124
9	2	90	14	18	4	7	5	822	558	255	174
10	3	172	26	475	76	14	11	162	122	35	35
11	2	137	32	735	130	4	2	73	24	40	18
12	3	244	39	242	45	40	17	1130	399	212	103
13	4	189	39	1018	123	23	8	440	52	99	14
14	4	203	70	546	115	41	22	750	162	125	16
15	1	130	55	1082	129	4	1	77	17	24	17
16	4	118	42	444	63	12	7	678	410	215	215
17	4	79	18	76	15	11	9	625	278	300	136

Table IV, V, VI, VII.

Abbreviations:

No S : the number of the test subject.

No I : the number of the irrigation during the caloric test. Normally four irrigations are performed. A number 5 indicates that one irrigation failed and was repeated 20 minutes after the fourth irrigation.

Table V.

FIRST SERIES WITHOUT AROUSAL

Results of the left cold irrigation:

No S	No I	Tr	V=V°	A°	F°	a	a°	t	t°	m	m°
1	3	127	14	212	46	14	8	510	85	120	22
2	3	120	19	130	49	27	16	667	321	160	59
3	4	154	36	1376	114	1	—	5	—	5	—
4	1	145	32	722	74	16	7	483	88	120	33
5	4	208	34	570	127	18	2	582	29	85	16
6	4	250	34	677	94	22	6	453	52	57	16
7	1	147	37	670	123	35	19	387	157	37	19
8	3	91	20	140	33	14	12	722	604	232	232
9	3	141	11	146	27	15	10	735	360	120	95
10	5	326	40	977	90	10	4	203	55	58	26
11	3	144	27	782	101	10	6	192	69	56	21
12	2	264	30	434	55	38	14	699	236	55	35
13	1	149	38	982	95	19	8	247	90	30	17
14	1	155	52	904	143	34	11	587	106	84	22
15	4	141	35	572	93	2	2	39	39	23	23
16	1	136	50	523	73	16	9	459	162	116	29
17	1	123	24	54	9	23	9	1021	454	160	127

Results of the right cold irrigation:

1	4	159	20	320	82	30	15	707	185	220	33
2	4	176	29	286	71	31	12	755	205	106	80
3	3	138	40	1287	123	2	2	26	26	15	15
4	2	121	32	625	81	17	10	503	170	126	41
5	3	120	27	540	56	22	10	601	195	90	55
6	3	233	25	658	106	29	6	541	52	86	10
7	2	160	30	663	114	21	10	247	101	36	28
8	4	172	15	21	9	20	6	1498	418	332	142
9	4	68	24	155	30	11	10	490	470	136	136
10	1	180	40	609	83	19	6	459	81	147	18
11	5	181	28	704	84	13	—	471	—	101	—
12	1	140	35	382	44	46	29	608	347	57	43
13	2	123	28	596	76	10	9	191	179	97	97
14	2	145	52	710	118	35	15	557	114	98	24
15	3	128	45	538	109	10	5	185	37	82	14
16	2	143	45	854	80	15	3	362	43	59	31
17	2	119	45	174	37	31	18	826	469	160	83

Tr : duration of the caloric reaction in seconds.

V=V° : maximum velocity of the slow phase (in degrees per second).

A° : total amplitude (in degrees) during the culmination period (i.e. between the 60th and 120th second).

F° : number of nystagmus beats during the culmination period.

Table VI.

SECOND SERIES WITH AROUSAL

Results of the left hot irrigation:

No S	No I	Tr	V=V°	A°	F°	a	a°	t	t°	m	m°
1	3	102	110	263	41	25	16	517	352	96	96
2	3	198	45	725	154	31	7	495	63	74	20
3	4	148	70	1632	159	1	—	90	—	90	—
4	3	139	55	533	96	32	23	890	235	197	28
5	3	107	28	361	69	21	13	304	185	44	44
6	3	199	50	1092	120	19	5	259	28	50	7
7	3	160	55	1164	158	32	10	513	78	50	15
8	3	127	80	663	82	34	24	711	248	182	24
9	2	210	50	907	88	12	6	226	53	65	16
10	3	165	55	1246	101	24	6	212	35	40	10
11	2	315	57	1395	145	28	3	538	21	67	11
12	3	36	70	60	7	15	4	166	67	35	35
13	1	196	55	1830	105	13	—	416	—	103	—
14	3	106	35	316	66	30	21	401	248	38	38
15	1	125	56	1197	137	5	1	60	22	22	22
16	3	165	29	472	85	19	9	466	112	150	21
17	1	105	48	843	165	9	2	121	15	40	11

Results of the right hot irrigation:

1	4	115	77	138	9	17	5	744	513	157	157
2	4	133	30	409	96	30	21	446	175	70	18
3	3	166	51	1368	128	2	—	40	—	22	—
4	4	109	60	615	87	31	17	453	231	80	80
5	4	102	29	217	49	20	16	503	340	124	96
6	4	162	50	1295	111	4	2	93	17	58	9
7	4	158	60	749	131	37	17	630	151	155	14
8	4	111	53	505	76	31	17	642	304	81	41
9	1	147	60	1353	99	17	9	181	42	50	7
10	4	149	49	1445	111	5	—	53	—	17	—
11	1	216	42	1244	151	24	—	491	—	72	—
12	4	172	57	377	43	34	16	1148	404	90	60
13	2	116	45	1505	92	9	1	149	4	47	4
14	4	203	40	670	106	47	15	491	96	27	10
15	2	119	50	1008	122	11	5	129	36	49	12
16	4	139	30	450	78	20	10	370	139	45	25
17	2	80	45	758	113	20	13	173	93	15	14

a : total number of pauses during the caloric reaction.
a° : total number of pauses during the culmination period.
t : total duration of all the pauses during the caloric reaction (in tenths of a second).
t° : total duration of all the pauses during the culmination period (in tenths of a second).

Table VII.

SECOND SERIES WITH AROUSAL

Results of the left cold irrigation:

No S	No I	Tr	V=V°	A°	F°	a	a°	t	t°	m	m°
1	1	134	50	961	79	19	14	170	120	14	13
2	1	157	26	524	93	10	2	158	20	39	12
3	2	325	37	1427	109	3	—	193	—	95	—
4	1	266	35	441	46	40	11	1176	287	175	78
5	1	188	28	720	103	15	3	344	24	65	12
6	1	284	27	882	87	34	6	708	56	67	16
7	1	147	35	795	121	29	14	316	118	38	27
8	1	121	32	677	84	24	17	272	135	66	19
9	4	234	53	759	63	28	7	751	80	200	20
10	1	170	34	1028	106	15	1	175	3	33	3
11	4	175	36	845	90	17	7	249	56	65	12
12	1	329	40	917	104	65	10	1178	60	74	12
13	3	205	34	1158	94	12	2	253	8	64	5
14	1	307	44	728	125	67	15	1399	114	142	18
15	3	154	31	528	74	25	15	465	237	69	69
16	1	142	36	706	111	9	3	151	28	40	11
17	3	107	25	446	100	16	10	163	67	35	10

Results of the right cold irrigation:

1	2	101	60	314	43	18	12	447	318	75	75
2	2	318	26	533	97	51	11	1356	105	162	29
3	1	167	46	1540	103	6	—	161	—	42	—
4	2	134	55	750	81	29	13	678	224	128	74
5	2	173	20	372	76	19	11	778	100	185	19
6	2	257	31	730	86	15	—	442	—	90	—
7	2	200	45	905	92	35	8	443	56	76	11
8	2	128	35	239	31	35	19	973	427	135	135
9	3	175	45	786	76	28	17	333	131	82	11
10	5	143	44	1006	108	22	5	324	27	78	8
11	3	282	45	1115	95	33	7	656	70	74	14
12	2	165	52	905	69	30	13	615	142	242	40
13	4	148	31	972	94	17	6	287	47	63	13
14	2	234	56	819	156	49	12	693	77	57	23
15	4	134	28	502	76	12	8	240	134	85	41
16	2	195	47	879	92	20	5	408	43	98	12
17	4	108	24	284	78	26	17	439	235	93	34

m : duration of the longest pause during the caloric reaction (in tenths of a second).
m° : duration of the longest pause during the culmination period (in tenths of a second).

0,3 sec.). If we compare this with the mean duration of the longest pause (8 seconds), there is a great difference. Megighian et al (1961) state that the mean duration of a pause during the whole reaction is from 1 to 6 seconds. Mehra (1964) finds them between 5 and 10 seconds. These figures agree with our findings.

Mahoney e.a. (1957) found pauses in 10 out of 18 subjects (i.e. 55 %) and Mehra in 25 %. In each case (i.e. 100 %) of our 17 subjects we demonstrated pauses in at least one of the 4 irrigations; in only 2 of the 68 recordings (i.e. 3 %) (both in the same subject), no pauses were demonstrated. These differing figures are probably due to the different standards and to not well-defined criteria used in measuring pauses.

The examination of many records containing pauses led Coats (1966) to the conclusion that the number of pauses increased with an increasing nystagmus intensity. Thus the longest pauses were usually present during the period of maximum intensity of the caloric nystagmus and also patients with relatively intense caloric responses tended to show a greater degree of pause formation.

We have already pointed out that, during the culmination period only the frequency of the pauses is at its highest. Table IV and V (see page 32-33) show that the maximum number of pauses and maximum total duration of the pauses as well as the maximum duration of the longest pause during the culmination period is encountered in subjects with a low speed of the slow phase, small amplitude, low frequency, and short duration of nystagmus.

Statistics demonstrate these facts even more clearly as can be seen in the tables VIII and IX (see page 80-81). In these tables we have looked for a possible correlation between quantitative parameters and pauses (see chapter III). In the cases where there was a significant correlation coefficient, it was preceded by a minus which indicates that the variables are inversely proportional. This, therefore, clearly contradicts the opinion of Coats. We are of the opinion that Coats has allowed himself to be misled by the fact that pauses are more prominent during pronounced nystagmus.

Rossberg (1956) established that the speed of the slow phase immediately starts at maximum speed during an active period between two pauses and that this speed remains constant during the remainder of the active period. Of course, the amplitude of the nystagmus changes during this period. Aschan et al (1956) described an increase of nystagmus during the active period up to a maximum, followed by a decrease till the following pause. The study of the speed of the slow phase and the amplitude during an active period is only purposeful if both factors are obvious, i.e. during the culmination period. We made a graph of each recording between the 60th and 120th second, plotting out the speed of the slow phase against the corresponding amplitude of each nystagmus-beat. In some irrigations where no pauses occurred, we established that the speed of the slow phase showed less obvious variations than the amplitude (Fig. A5). This phenomenon was also described by Rossberg (1956). The larger, irregular fluctuations of the amplitude are due to the appearance of "crochetage", salvos etc. The fluctuations in speed of

the slow phase are more regular, lasting from 4 to 8 seconds. After the start of a pause the nystagmus is usually interrupted suddenly, and after the pause the speed of the slow phase continues at the same rate as before the pause (fig. A6 and A7). We did not observe an increase of the nystagmus to a maximum followed by a decrease during the active phase, except where the active phase coincidently occurred simultaneously with the periodical change in the speed of the slow phase. However, the difference between the maximum and the minimum is very small. The amplitude of the last or first nystagmus-beat during an active phase can either be large or small (Fig. A6 and A7).

Hamersma (1957), repeating the caloric test even after an interval of a few months, found an increase in the number of pauses. Lidvall (1961) studied the response decline. He performed 78 series of irrigations with an interval of 10 minutes. In 56 series (71 %) he could not find extreme increases or decreases. In 20 series (26 %) he found an increase, and in 2 series (3 %), a decrease in the number of pauses.

We had at our disposal recordings of 17 subjects, each having had four irrigations with intervals of 20 minutes between them. As regards the total duration of the pauses, we found one subject (table IV—V no. 8) with increasing total duration of the pauses and one subject (table IV—V no. 17) with decreasing total duration, and for the duration of the longest pause during the caloric reaction, we found one subject (table IV—V no. 8) with an obvious increase. *)

Christian (1954) described a tremor of the eyes during the pauses.

Megighian et al (1961) found that a pause begins after a fast phase and that at the end of a pause, the nystagmus begins again with a slow phase. However, this slow phase was not easy to recognize, since the investigator used a short time-constant (1,5 sec.).

Montandon and Dittrich (1962) noticed during the pause, eyemovements with an amplitude smaller than one degree. Sokolovski (1966) described the appearance of deformed nystagmus or total rest of the eyes during the pause. With the aid of a monocular vertical lead, Megighian e.a. (1961) at the beginning of a pause often found a downward-directed vertical eyemovement. Gabersek e.a. (1965) found a rapid vertical upward eyemovement shortly before the appearance of the pauses (the time-constant of his E.N.G. leads were: 0,1; 0,3 and 0,5 sec). From this he concludes that it is the upward eyemovement — spontaneous or voluntary — that induces the pause.

One can only make reliable recordings within the limits of the time-constant of the apparatus (in our case 10 seconds). Slower movements are either recorded partially or not at all.

We found large differences in the horizontal movements. During the pause most of the recordings either indicate the eye to be in a resting state (Fig. A8), or they show a pattern with a slide wave (Fig. A9). Oc-

*) The successive irrigations were performed in the following way: subject no. 8: left hot, right hot, left cold, right cold; and for subject no. 17: left cold, right cold, left hot and right hot.

casionally brusque movements of large amplitude occur or the eyes deviate rapidly in the direction of the fast phase (Fig. A7). On other occasions rounded nystagmoid movements occur with a considerably slowed-down fast phase (Fig. A10), or nystagmus-beats whose sharp apex is cut off (Fig. A11 and A12). Often restless eyemovements are found during pauses, occasionally even a tremor of the eyes (Fig. A13).
Our investigation pointed out that, in normal individuals, a pause can begin both after a fast and after a slow phase, and although it ends mostly with a slow phase this is not always the case.
The vertical eyemovements of large amplitude can be divided into two groups: some that gradually move up and down and some that appear suddenly. As a rule, the slow movements result in a suppression of the horizontal and vertical nystagmus only when a maximum upward deviation is reached (Fig. A14). The brisk vertical eyemovements nearly always abruptly suppress the horizontal and vertical nystagmus, and almost appear together with the fast phase of the horizontal and vertical nystagmus (Fig. A7). It also must be mentioned that we sometimes recorded vertical eyemovements of large amplitude which did not in the least influence the nystagmus.
We especially recorded the E.E.G. in order to get an impression of the degree of alertness of the subject during caloric nystagmus. Usually the alpha-rhythm is not disturbed during a pause (Fig. A8 and A13). We seldom observed that the pause was paired with a disturbed alpha-rhythm (Fig. A10).
Mahoney e.a. (1957) established that with closing the eyes pauses appeared, together with an alpha-rhythm in the E.E.G. By presenting the subjects with an arhythmetical problem they saw the pauses as well as the alpha-rhythm disappear again. From this they concluded that pauses are abolished by two factors which disturb the alpha-rhythm, namely opening the eyes and "alerting".
Megighian et al (1961) found that the alpha-rhythm remained unchanged during the pause and that by acoustic stimulation the pauses as well as the alpha-rhythm disappeared. However, they found that by repeated acoustic stimulation the pauses always disappeared, but that no further disturbance of the alpha-rhythm occurred.
Up to this point our findings are in agreement with those of both authors, i.e. that the alpha-rhythm usually is maintained during the pause. However, we also found some pauses in which the alpha-rhythm was disturbed. Therefore we can say that cortical influences usually facilitate the nystagmus, but can cometimes suppress the nystagmus (see later page 74).

Discussion

Rossberg (1956) has, as already mentioned, noticed rhythmic, periodical changes of the speed of the slow phase and of the amplitude. According to him these changes cannot be explained on a peripheral mechanical basis only. He finds it more probable that overlapping of one or more central excitatory states of different rhythm has occurred. If we accept that the amplitude varies more than the speed of the slow phase, the hypothesis holds that the amplitude is also more sensitive to central influences. He finds further support for his hypothesis in the "Kipp-model" of Bethe (1940), demonstrating that the loading phase (cf. slow phase) is a much simpler process than "Kipping" (cf. fast phase). According to the author the rhythmical interruptions of the nystagmus or pauses are an extreme result of these rhythmic periodical changes.
The conclusion of the author that the fast phase is more easily influenced than the slow phase, is not new. Already in 1907, Bárány found in a patient with a brainstem lesion at the level of the reticular formation, an absence of the fast phase, paired with a deviation of both eyes in the direction of the slow phase. This phenomenon was later described in patients with brain lesions, coma, and anaesthesia by Bárány e.a. 1911, Lorento de Nó (1928), Alfandary (1937), Kornhuber (1966) etc.
Later on the hypothesis of Rossberg (that fluctuations of both above-mentioned parameters, are not of labyrinthine origin) was confirmed by Ledoux (1949) and Groen (1961). These authors plugged one semicircular canal with the cupula in a deviated position in an isolated frog preparation, and could not demonstrate any fluctuation in the electric activity of the afferent nerve.
From our recordings we see, however, that fluctuations in the speed of the slow phase and in the amplitude are not synchronous and that the occurrence of pauses is not at all rhythmical. Pauses can occur at any given moment during the periodical changes of these two factors. Firstly, the fact that fluctuations of these factors are not synchronous, and secondly, that the pauses can appear at any given time during these fluctuations, shows that the occurrence of these fluctuations does not depend on the occurrence of pauses. The fluctuations in speed of the slow phase can be partially explained by the superimposing of slow pendular eyemovements on the slow phase of the nystagmus.
Buys (1924), Greiner e.a. (1967), Clemens and Festen (1970) also observed these pendular movements. The larger and more irregular fluctuations of the fast phase were already explained by the occurrence of "crochetage", groupformation, "petite écriture", salvos etc.
Mahoney e.a. (1957) believed the origin of the fast phase to be in the cortex. The fast phase would result from a compounding of influences involving the voluntary motor system of the eyes (frontal mechanism), the reflex-following system (occipital mechanism), and perhaps other cerebral influences concerning the orientation of the eye. They had two hypotheses. With eye-closure they observed that the frequency slowed down or a pause occurred. During a pause the eyes could no longer remain in the original position, since the extra-ocular motor nuclei where then under the influence of stimuli from brainstem nuclei, chiefly vestibular, and not controlled by inhibiting influences from the cortex. With alerting the latter influences dominate again and the eyes return to the primary position. Their alternative explanation is that eye-centering mechanisms of the cerebral cortex hold the caloric nystagmus in check as long as the attention of the person can be directed toward this end. Any diverting influence such as mental activity would interfere with this function of the cortex and make the nystagmus reappear.

The appearance of pauses and alpha-rhythm during eye-closure, and the reappearance of nystagmus after a cortical stimulation with suppression of the alpha-rhythm would prove their theory. They considered as a further proof the investigations of Spiegel and Aronson (1933) who found that eye-closure provoked pauses that interrupt a caloric nystagmus, and attributed this to fluctuations in the excitability of the reflex-arc itself.
It seems very unlikely to us that the fast phase has its origin in the cortex. The experiments of Bauer and Leidler (1912) proved this to be impossible. They were still able to induce nystagmus in animals, after having removed the hemispheres and the cerebellum, as well as a section of the brainstem just anterior to the abducens nuclei and behind the vestibular nuclei. This was also observed in humans. De Kleyn (1939) induced a vestibular nystagmus in an anencephalous monster with the same lesions as described in the investigations of Bauer and Leidler (1912).
Indeed we find that with eye-closure and alpha-rhythm occurs, it persists mostly during the pause, but after one acoustic stimulation the nystagmus reappears and the alpha-rhythm disappears. However, with repeated acoustic stimulation (see also Megighian et al 1961, Crampton 1964) the alpha-rhythm does not disappear anymore, but the nystagmus continues to reappear each time. All these findings point to the fact that the system involved in bringing the eyes back to the midline position should not be looked for in the cortex, but in the brainstem. This does not mean that the cortex does not influence the caloric nystagmus. On the contrary, in our recordings we could indicate a few pauses coupled with suppression of the alpha-rhythm. Also over-alerting will suppress the nystagmus, e.g. fixation with arithmetical problems (Sokolovski 1966).
It is difficult to compare the experiments of Spiegel and Aronson (1933) with the routine caloric test. They worked on cats kept under superficial ether anaesthesia, and irrigated the external auditory canal — in some cases they even perforated the ear drum — during hours with cold water from 23° C to 8° C. Furthermore it seems very unlikely to us that in these experiments the cats were examined with their eyes closed, since the investigators used a lever-kymograph attached to the sclera. We also disagree with the theory that closing the eyes would suppress the caloric nystagmus and find support in the literature (Aschan, Bergstedt and Stahle 1956, Jongkees 1960, Mahoney e.a. 1961, Jongkees and Philipszoon 1964, Sokolovski 1966).
Lidvall (1961) studied the response decline of the caloric nystagmus after repeated irrigations with short intervals. He assumed that a gradual increase of the pauses during the caloric nystagmus can be interpreted as an expression of progressive cerebral control of nystagmus (habituation). However, we found that the pauses do not increase during succeeding irrigations, but occur at random. There exist two mechanisms other than habituation that can be responsable for a response decline namely adaptation and fatigue. Though these are old conceptions (Breuer 1874), up to now one does disagree about their meaning and origin. According to Sharpless and Jasper (1956) and von Albert (1968) adaptation is the result of one stimulus of long duration, and habituation (or habit forming) is the result of a series of repeated stimuli. Adrian and Zotterman (1926) and Beerens (1969) are of the opinion that adaptation should be seen as a centrally occurring process at the level of the second-order neurons or as a peripherally occurring process at the level of the sensory epithelium, while habituation is the result of a more intricate activity at a higher level in the central nervous system.

However, none of these mechanisms explains the pause phenomenon. According to Singleton (1967) before they come into play the stimulus must be present for at least a few minutes (adaptation), for several hours (fatigue), or for several days (habituation). Furthermore, as a result of these mechanisms the suppressed reaction does not abruptly return to its original maximum value. The speed of the slow phase and the amplitude after the occurrence of a pause, however, do regain their original value at once.
Megighian e.a. (1961) were of the opinion that pauses are not of cortical origin, considering that the alpha-rhythm continues during pauses, but believe that there could be a difference in the activity of the vestibulo-ocular reflex arc at the level of the reticular formation. During acoustic stimulation there is an increased activity in the reticular formation, followed by a facilitation of the conduction of the vestibular stimulus and a change in the activity of the cortex. Repeated acoustic stimuli occur only in combination with a facilitated conduction of vestibular activity without any change in the activity of the cortex. Therefore there would be a conditioning of the reflex. At this point we must mention that excessive cortical activity with disturbance of the alpha-rhythm can result in the appearance of pauses.
Demetriades and Spiegel (1925) indicated the inhibiting influence of the cerebellum on nystagmus. After ablation of the cerebellar nodulus in cats, Fernandez and Fredrickson (1963) showed a prolonged vestibular reaction to caloric stimulation. In an other series of cats they stimulated the nodulus electrically and observed that by stimulating and during the whole period of each stimulation, the eyes showed a conjugate deviation in the direction of the slow phase. Considering that the nodulus has efferent pathways leading to all vestibular nuclei (fastigial nucleus, dorsal reticular formation and the medial longitudinal fasciculus), they assumed the hypothesis that the nodulus acts as an inhibitor upon the neural mechanism underlying the fast phase of the nystagmus. They emphasized the possibility that this effect could originate as a result of a spreading of electrical stimulation to other structures. We did not record this deviation of the eyes in the direction of the slow component or in other words; the occurrence of pauses cannot be explained only by an increased cerebellar activity.
Gabersek and Jobert (1965) stated as a result of their observations that an isolated voluntary eyemovement never occurred during the slow phase, this voluntary eyemovement is then a substitute for the fast component. They therefore do not believe in the existence of a nystagmogenic area for the fast component. The fast phase could be triggered by a stretch-reflex (myotatic reflex). They believe to find a confirmation of their hypothesis in patients with Friedreich ataxia paired with an

absence of the fast components of the nystagmus and of all tendon-reflexes and in which a "viscosity" of the gaze occurred (i.e. a slowing down of all voluntary eyemovements as though they were moving in a viscous suspension: Garcin and Man 1958).

According to this hypothesis, pauses are explained by a peripheral blockade. This last supposition is strengthened by the fact that the pause as a result of an eyemovement (previously Gabersek e.a. stated that an upward eyemovement causes a pause) is not accompanied by a pause of the other vestibular expressions (sensation, vestibulo-spinal reflex) which proves the continuation of the vestibular impulses. No fast phase occurs in the limbs, because the slow phase is subliminal, the stretching of the muscles too weak, and the movement of the limb too slow (in comparison with the speed of the slow phase of the eye) to elicit a myotatic reflex. It would be the speed of the slow phase, dependent on the sensitivity of the neuro-muscular end organs (Cooper and Daniel 1949, Christman and Kupfer 1963) that is responsable for the generation of a fast phase.

The absence of the fast phase in the vestibulo-spinal reflex in humans was clearly demonstrated by the recordings of Henriksson, Janeke, and Claussen (1969) who recorded nystagmus as well as latero-torsion of the head during routine examinations. Nevertheless, the existence of a fast component of the vestibulo-spinal reflex has been demonstrated: e.g. the laws of Ewald (1892) were formulated after the observation of a head nystagmus in pigeons and in patients a head nystagmus was observed by Fuite (1954). Even though the theory of Gabersek (a theory Bartels already put forwards in 1914), concerning the origin of the fast phase at the level of the extrinsic eyemuscles seems very attractive, it has been disproved on very solid grounds since decades.

In rabbits De Kleyn (1922—1939) observed that nystagmus continued to occur after he had cut through all extrinsic eyemuscles with the exception of one lateral rectus, into which novocaine was injected (an amount just short of causing paralysis, and so that the proprioceptive nerves were safely blocked, but the motor activity remained intact). At this stage, according to the theory of Bartels and Gabersek, one would expect a slow deviation of the eyes in the direction of the slow phase only. McCouch and Adler (1932) could not demonstrate a stretch-reflex in the eyemuscles of cats. At the level of the proximal end of the cut abducens nerve McIntyre (1939) observed a decreasing amplitude and frequency of the tonic discharges, corresponding to the slow phase followed by a loud burst of discharges corresponding to the fast phase. Kornhuber (1966) could not record a fast phase in humans when he moved their eyes by applying external pressure.

The publication of Garcin and Man (1958) deals with certain cases of cerebellar and spino-cerebellar degenerative diseases and one case of cerebro-vascular accident at brainstem level. They observed that the voluntary horizontal eyemovements were slowed down and laborious, but that the automatic reflexes were normal. Garcin and Man stated in their publication that the slow phase of the nystagmus was blocked (and not

the fast phase as Gabersek mentioned) as a result of lesions at the level of the medial longitudinal fasciculus.

The fact that during a pause other vestibular manifestations are not interrupted, is no proof that the fast phase does not have a central origin. The other vestibular manifestations probably follow other central pathways.

Long ago the cerebral and proprioceptive theories concerning the origin of the fast phase have already been rejected. The existence of a Bechterew nystagmus proves every localization of the fast phase at labyrinthine level to be impossible. At a later date similar theories were refuted by the experiments of Spiegel and Sato (1927) who, after cutting through both auditory nerves and by puncturing the vestibular nuclei, were still able to produce a nystagmus; and by the experiments of Dohlman (1938) who showed by studies of chonaxia and rheobase that the vestibular conduction system is too slow to conduct the fast phase.

Later the origin of the fast phase was placed at the level of the oculomotor nuclei by Wendt (1951) and Fluur (1962) who stressed that this was the natural point of convergence of both the optokinetic and the vestibular nystagmus. Spiegel (1930) cut the connections between the right vestibular and oculomotor nuclei, superior to the abducens nucleus in dogs, and detached the left eye muscles with the exception of the internal rectus. The absence of vestibular nystagmus after stimulation shows that the oculomotor nuclei with their internuclear connections are not able to produce a nystagmus by themselves.

According to the hypothesis of Bauer and Leidler (1912) and Spiegel and Sato (1927), the fast phase of the nystagmus would be created at the level of the vestibular nuclei. Lorente de Nó (1928) found that a lesion of the medial longitudinal fasciculus did not prevent a vestibular nystagmus, and was more inclined to believe that the fast phase originated at the level of the reticular formation. Blohmke (1929) provoked a nystagmus by stimulating reticular formation at the level of the mesencephalon. Spiegel (1930) confirmed the findings of Lorente de Nó. Yet, after he had produced large lesions of the reticular formation by puncturing the brainstem from the ventral aspect, on rotation he still observed nystagmus with both components. Lorente de Nó (1933) destroyed the reticular formation by dorsal puncture and established that after stimulation only deviation of the eyes without a fast component occurred. Spiegel and Price (1939) repeated these experiments and discovered that the fast phase returned after a few days. With these findings it would appear that the vestibular-nuclei theory could be correct.

In 1954, Eckel found rhythmical discharges in the vestibular nuclei during post-rotatory nystagmus. However, three years later with single neuron recordings, Duensing and Schaeffer found units in the reticular formation which discharge synchronously with the quick component and units synchronous with the slow component.

Similar units were also found in the medial longitudinal fasciculus that receives its fibres from the vestibular nuclei. An interaction between some units of the vestibular nuclei and the reticular formation was demonstrated.

Lachmann, Bergmann and Monnier (1958) described a meso-diencephalic nystagmogenic area (D.N.A.) in rabbits. Oosterveld (1963) and P. Montandon (1964) found that by simultaneous stimulation of the D.N.A. and the labyrinth, subtraction or summation phenomena appear. In 1964 Gernandt showed that stimulation of the vestibular nerve elicits unit discharges in the reticular formation that are more complex than those recorded from the vestibular nuclei. Following Gernandt's findings, Spiegel (1964) ceased to refute that the reticular formation plays a part in the genesis of the fast component.

Kornhuber (1966) believes that the generative mechanism of the fast phase which is influenced by the cortex is formed by the pre-motor apparatus of the oculomotor nuclei, i.e. the gaze centres of the para-median reticular formation situated in the pons and the midbrain.

McCabe (1965, 1966) found an impaired slow phase of the vestibular nystagmus, subsequent to bilateral lesions of the vestibular nuclei. After the administration of drugs that mainly act on the reticular formation the speed of the fast phase was impaired. After damage to the reticular formation, especially in the vicinity of the fifth motor nucleus, the fast phase was eliminated without disturbance of the slow phase. According to McCabe, a nystagmus is generated in the following manner:

The vestibular nuclei receive afferent impulses via the vestibular nerve. The slow component is produced by impulses via fibres ascending in the medial longitudinal fasciculus and the reticular formation to the eye-muscle nuclei. Slow-component fibres send branches to high-threshold reticular-activating neurons which, when their threshold is reached, fire and produce the quick phase. As this neuron fires, it fires upon a reticular inhibitor-neuron which acts to cut off the flow of incoming slow component discharges. The first nystagmic cycle is thus born.

Cohen and Feldman (1968) found potentials in the paramedian zone of the pontine reticular formation that lead each rapid movement of the eyes by 10—20 msec. They were of the opinion that portions of these pontine eyemovement potentials probably reflect activity in neurons which generate quick phases of nystagmus or jerks. Following their investigation Horcholle and Tyc-Dumont (1968) assumed that the existence of ocular interneurones with opposing functions (inhibitory or re-excitatory) could explain the periodicity of the nystagmic discharges and also their progressive increase in amplitude by involving the action of short closed loops of interneurones.

We conclude that even though the reticular formation theory is considered more and more acceptable, all these hyphotheses indicate the complexity of the origin of the fast phase, and the obscurity of its real origin (Biemond 1961).

The occurrence of pauses can be explained in different ways, i.e. by an increased activity of an "inhibitor neuron", or by an increased contralateral D.N.A. activity, suppressing the ipsi-laterally generated nystagmus, or indirectly by an increased activity of the cerebellum, or of the cerebral cortex.

Szentagothai and Schab (1956) found that, during simultaneous electric stimulation of the posterior semi-circular canal and Darkschewitch's nucleus, the vertical nystagmus ceased immediately. Fluur (1962) was of the opinion that Darkschewitch's nucleus inhibits the interstitial nucleus of Cajal which receives its impulses from the posterior vertical canal. He thought that the inhibition was caused by an hyper-polarisation of the neurons, with a raised threshold for depolarisation-pressure. Unfortunately it has never been established whether Darkschewitch's nucleus or similar nuclei do indeed have any influence on horizontal nystagmus.

We should not forget the part of the efferents in suppressing labyrinthine activity (Engström 1958, Wersäll and Flock 1963). Gleisner and Henriksson (1963) by cutting the vestibular nerve in frogs demonstrated a distinct efferent activity in the proximal end, caused by utriculo-fugal acceleration (of the ipsi-lateral labyrinth), but only at an acceleration of $15^\circ/sec^2$. With a smaller stimulus no efferent activity was recorded.

Schmidt (1963) and Bertrand and Veenhof (1964) showed that also other than vestibular stimuli could produce an efferent activity in the eighth nerve e.g. passive movements of a limb, gently probing the belly or gently probing the posterior part of the eye in the area of the Gasserian ganglion.

The role of the efferents was clearly demonstrated by Goetmakers (1968) who found a large increase in the frequency of the afferent action potentials of the eighth nerve after the death of the test animal.

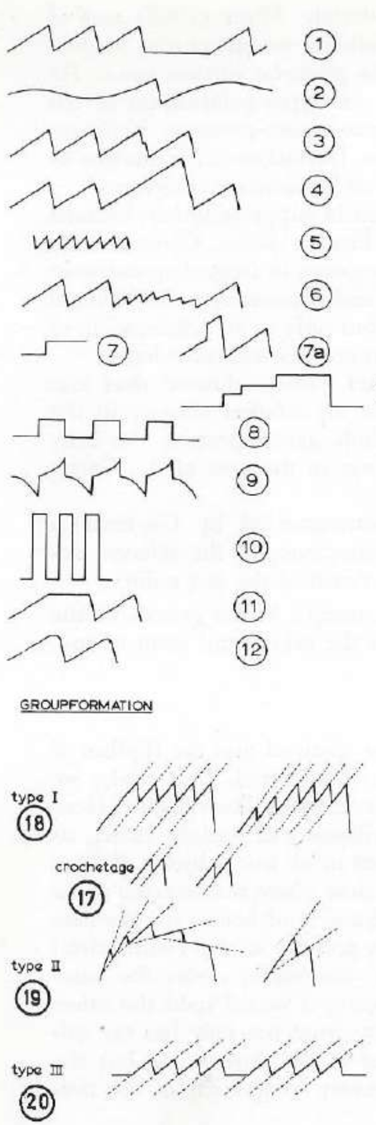
In conclusion: as long as we have so little insight in the genesis of the fast phase of nystagmus, we cannot explain the origin and form of nystagmus or consequently of its pauses.

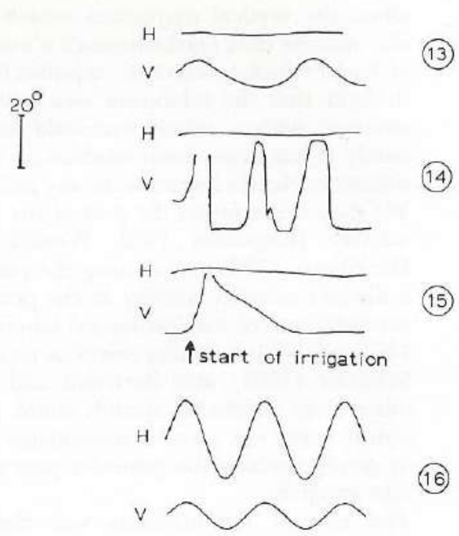
2. Anomalies during the active period

In the study of the normal nystagmogram we noticed that the rhythm of the nystagmus was not always as regular as we expected. This is why we recorded the "derived nystagmogram" according to Henriksson (Henriksson 1955a, 1955b, 1956, Dohlman, Henriksson and Andrén 1956), simultaneously with the routine nystagmogram in all our subjects. By this method the speed of the fast phase and the slow phase is measured as an amplitude above (in the case of the slow phase) and below (in the case of the fast phase) a base-line. Moreover it is possible to clip (with cristal diodes) the recording of the fast phase one centimeter under the base-line. The speed is so high that without clipping it would spoil the other recordings (Fig. A15). The derived nystagmogram not only has the advantage of giving informations on the speed of the slow phase, but the clipped amplitudes of the fast phase show every irregularity of the nystagmus-rhythm quite clearly.

We used the clipping regularly. Occasionally, at the end of the recording or during extreme arhythmia, the clipping mechanism was switched off. The speed of the slow phase and the amplitude of the nystagmus are then so small, that they are difficult to recognize (Fig. A19 and A16). The speed of the fast phase however remains relatively high and still gives a large downward deflection in the derived nystagmogram.

Also counter-jerks (i.e. fast component in the direction of the slow phase) are more easily detected in the derived nystagmogram (Fig. A17). If the speed of the slow phase is high during the culmination





1. pause
2. arhythmia
3. intermittent fast phase
4. dysmetria
5. "petite écriture"
6. salvo
7. jerk

7a. counter-jerk

7b. "staircase"-formation

8. square waves: real (DC- or long time-constant AC-recorder)
9. Kipp-nystagmus or nystagmus basculans: distorted (short time-constant AC-recorder)
10. Kipp-deviation
11. decapitated nystagmusbeat
12. rounding of apex
13. sinusoidal movements
14. spontaneous P.O.S.
15. irrigation P.O.S.
16. pendular movements
17. "crochetage"
18. groupformation type I
19. groupformation type II
20. groupformation type III

phenomenon, counter-jerks are often difficult to recognize. They are more easily recognized as a peak in the derived nystagmogram.

a. *Arhythmia*

Arhythmia is an extreme form of dysrhythmia (Fig. 4 no. 2). The nysmus is totally desorganised, i.e. atypical, aperiodic oculo-vestibular eyemovements in which here and there very irregular diphasic nystagmustagmus is totally desorganised, i.e. atypical, aperiodic oculo-vestibular eyemovements in which here and there very irregular diphasic nystagmusbeats are recognizable (Montandon e.a. 1962, Mounier-Kuhn e.a. 1966). In 41 of our 68 recordings we indeed demonstrated arhythmias.
We have divided them into three groups. In our first and largest group we could only demonstrate arhythmias at the beginning of the caloric nystagmus (Fig. A15, first part) after the appearance of the first nystagmus beat or at the end of the caloric nystagmus. In the second group arhythmias also occurred during the culmination period. In the third group the entire caloric-nystagmus reaction was arhythmical (Fig. A16).
An arhythmical response is characteristic of a particular subject. It does not occur at random. Neither is it found as a result of sleeping. In Fig. A16 we see an arhythmical response (together with a well developed alpha-rhythm in the E.E.G.) during the culmination period. There are no signs of pendular movements in the horizontal lead. In subject no. 8 we found a normal response during the first irrigation (Fig. A18). However, the following irrigations showed clear-cut arhythmia (Fig. A19). In this subject who experienced the vestibular test for the first time we can expect the presence of a strong arousal-effect during the first irrigation. Arhythmia in the horizontal lead is always accompanied by one in the vertical lead. Here arhythmia is even more often found since vertical nystagmus is mostly smaller in size than horizontal.
So we can conclude that in most cases the occurrence of arhythmia is due to a lack of alertness. However, it is no sign of sleep because the alpha-rhythm still exists.

b. *Intermittent fast phase*

An intermittent fast phase was described for the first time by Neveling and Poeck (1960) in patients suffering from Wallenberg syndrome (i.e. thrombosis of the posterior inferior cerebellar artery). They described a fast phase interrupted by short pauses in which case the duration of the fast phase, including the duration of the interruptions, is longer than that of one slow phase. They mentioned that this phenomenon did not appear in every case of Wallenberg syndrome. It depends on the extent of the vascular injury. Dufour e.a. (1966) described the same phenomenon as a "dentated fast phase". They also observed this dentation in the slow phase.
We see this phenomenon when a nystagmus is superimposed on a slow to and fro eyemovement, and therefore we will discuss it later together with groupformation. We accept to call this phenomenon "intermittent fast phase" with the restriction however, that the interruption may not be of

longer duration than 0.1 tot 0.2 seconds (Fig. 4, no. 3). We saw these intermittent fast phases in 47 of our recordings, in 31 of these cases we also saw this phenomenon during the period between 60 and 120 seconds. These very short interruptions are not explained by the fact that a nystagmus is superimposed on a pendular eyemovement. These intermittent fast phases appear to be induced by a change in the vertical lead. Every time a small interruption of the fast phase in the horizontal lead appeared, we observed a change in the polarity of the vertical lead (Fig. A17).
In the same recording (Fig. A17), however, we also detected a few intermittent fast phases as described by Neveling e.a. Here we see no changes in the polarity of the vertical lead.
In conclusion we can state that the occurrence of an intermittent fast phase in the horizontal lead, as we defined it, is due to a sudden change of polarity in the vertical lead and that this finding is not pathological.

c. *Amplitude of abnormal size*

Normal values of the amplitude of the nystagmus beat are between 5 and 15 degrees, but occasionally we see amplitudes of a greater and smaller size. We call the larger "dysmetria" and the smaller "petite écriture". The average amplitude, however, remains constant. This is in contrast to the following group in which there is a great variation in amplitude.
According to van de Calseyde et al (1969) the amplitude varies during life. In an investigation carried out on the torsion swing they found dysmetria especially in young people.
According to them the amplitude diminishes in older people, but the frequency increases.

a. Dysmetria

We define dysmetria as a nystagmus beat with an uninterrupted slow phase and a fast phase greater than 20° (Fig. 4, no. 4). Nystagmus-beats with an amplitude larger than 20° often appear but then the slow phase is interrupted by one or more fast phases. We shall later describe this latter type as "crochetage" or groupformation, type 1. In 12 of our recordings we found examples of dysmetria. In 3 of these cases dysmetria occurred outside the culmination period. However, the phenomenon usually showed itself during the culminationperiod (Fig. A20). A few of our recordings were entirely dysmetrical and showed a constant frequency. Dysmetria can be of high as well as of low frequency and does not influence the speed of the slow phase which follows its normal course.
In 8 of the 12 recordings dysmetria was not observed in the vertical lead: either there was no nystagmus, or there was nystagmus but no variation in amplitude. In one case blinking occurred which made it impossible to interpret the vertical component. In three cases there was an increase in amplitude of the vertical nystagmus without exceeding the 20° limit.
Montandon e.a. (1962) described dysmetria during a post-rotatory nystagmus in which the amplitude was alternately large and small without a change in frequency. According to their theory the speed of the slow phase must change radically as soon as the amplitude changes and not the frequency. This does not agree with our findings and seems very unlikely. From the recordings they published it appears that "crochetage" in our terminology is the phenomenon they discussed.

b. "Petite écriture"

A nystagmus of small amplitude and a frequency of 3 tot 5 beats per second (Fig. 4, no. 5) is called "petite écriture" (i.e. small script). Each series of nystagmus-beats which has an amplitude smaller than 5 degrees per beat we regard as "petite écriture", provided it does not deviate from the base-line and appears for short periods only, in which case we name this special type of "petite écriture" salvos. "Petite écriture" always occurs in combination with an increase in frequency. It does not only occur during the whole duration of the caloric reaction, but also for shorter periods during an otherwise normal nystagmogram (Fig. A21 and A22). Occasionally we even see "petite écriture" as one beat between nystagmus-beats of normal amplitude (Fig. A8, A20), though one might discuss the legitimacy of the name in this case.
In the vertical lead we usually observe a nystagmus that is difficult to recognize as such, since the amplitude is so small, or no nystagmus at all.
In 11 recordings we demonstrated a temporary "petite écriture". Furthermore it appeared that in the recordings of one and the same person some recordings showed "petite écriture" and others a normal amplitude.

c. Salvos

A special form of "petite écriture" during caloric reaction is a salvo (Aboulker e.a. 1967). The nystagmus has an amplitude smaller than 5°, has a fast rhythm and the needle deviates from the base-line during the salvo (Fig. 4, no. 6). A salvo usually does not last longer than two seconds (Fig. A7, A18). During a salvo the vertical component is small or absent since also the amplitude of the horizontal lead is already small. In 34 recordings we saw salvos appear. In the same subject salvos can occur in one, more or all irrigations, mostly during the culmination period. During the salvo the speed of the slow phase sometimes remains constant but usually diminishes.

It seems logical to conclude from our experiments that in normal individuals dysmetria, "petite écriture", and salvos are common findings and that we must be extremely careful in attributing a diagnostic value to these phenomena.

d. *Varying amplitude or groupformation*

We have already discussed (under the heading: pauses) the slight periodic variations of amplitude and speed of the slow phase which occur

during the period between the 60th and 120th second. Here we noticed that the variations in amplitude were greater and more irregular. This is primarly due to a phenomenon we call groupformation.
During a caloric reaction the nystagmus recording does not remain on the same level. Nystagmus occurs sometimes at one and then at another level, forming as it were groups. We have attempted to classify these groups according to their appearance.
In the literature attempts have already been made to classify these groups, but these classifications are rather confusing (see page 13-19).
Neveling and Poeck 1960, Gabersek e.a. 1965, Dufour e.a. 1966, Aboulker e.a. 1967, and Jongkees 1969).
Against this confusion we feel justified to make our own classification in order to avoid misunderstanding. It is evident that any classification is artificial and that in reality the different subdivisions overlap. Fig. 4, no. 17-18-19-20 gives the various subdivisions in their ideal form. We differentiate three types:

a. type I: groups in which the speed of the slow phase remains constant. (Fig. 4, no. 17-18).
The nystagmus deviates in the direction of the slow phase by consecutive incomplete corrections of the fast phase or by a long lasting slow phase. After this deviation the base-line continues at another level and finally the nystagmus returns to the previous level with a fast phase of large amplitude. The speed of the slow phase remains constant (Fig. A18, A20, A22) or is slightly reduced (Fig. A23) while the frequency of the nystagmus remains the same or increases slightly. When this type of group is formed by only two nystagmus-beats we call it "crochetage" or notched nystagmus-beat (Fig. A1, A14, A17, A18). Aboulker defined the latter as a "bifid" nystagmus-beat.

b. type II: groups where the speed of the slow phase does not remain constant. (Fig. 4, no. 19).
In this group the nystagmus also deviates in the direction of the slow phase. Initially the speed of the slow phase increases gradually and when it returns to its previous level the speed of the slow phase gradually decreases (Fig. A8, A17, A20, A23). It probably represents a nystagmus superimposed on a slow sinusoidal eyemovement; Aschan, Bergstedt and Stahle (1956) already described this phenomenon.

According to the law of Alexander (1911) the speed of the slow phase will show striking changes in both the first and the second group when the deviation becomes too large.

c. type III: groups divided by pauses (Fig. 4, no. 20).
In this type the nystagmus is interrupted by many pauses and does not necessarily show variations in amplitude. Because the formation of groups is so striking we have classified it here. It is due in fact to the pause-phenomenon. When the pauses are frequent enough, they divide the nystagmus into groups. In this case the frequency and the speed of the slow phase do not change (Fig. A11).

The greatest difference between type I and II is seen in the vertical lead. In type I the recordings of the vertical and the horizontal lead differ. The beats are not synchronous. "Crochetage" can occur in the vertical lead but less frequent and asynchronous to the horizontal lead. In type II the vertical eyemovements occur synchronously to the horizontal. It is in fact a slow pendular oblique eyemovement on which a predominantly horizontal nystagmus is superimposed. If these slow pendular movements are of small amplitude this phenomenon creates the rhythmic periodical changes of the speed of the slow phase in the period between 60 and 120 seconds which we have already discussed (page 39), and then we shall only be able to detect it by observing the changes of the speed of the slow phase. Type II is formed (Fig. A20, A23) when the amplitude of these movements becomes too large (larger than the amplitude of the nystagmus-beat) and when they are of short duration.
We have already discussed the vertical eyemovements during type III, together with the pause phenomenon (page 38).
Though it is difficult to study these types separately since they often overlap, we can conclude that in the recording they mostly form the typical findings characteristic for each individual.

e. *Secondary-phase nystagmus*

Aubry e.a. (1965) described inversion of the caloric nystagmus in a patient with a tumour of the fourth ventricle. Aboulker e.a. (1967) found in 3,5 % of their patients suffering from head injury a change in direction of the nystagmus during the caloric test, and this occurred in one or two irrigations of the same ear. According to these authors the inversion occurred two minutes after the beginning of the irrigation and in one of the irrigations the direction of the vertical nystagmus also inverted. From the descriptions the authors give, it appears to be a secondary-phase nystagmus (Thornvall 1932, Hamersma 1957), not to be confused with nystagmus alternans (a nystagmus which repeatedly changes in direction; Kornhuber 1966) and perverted nystagmus (a nystagmus having a vertical polarity after stimulation of the horizontal semi-circular canal; Riesco-MacClure 1960).
The secondary-phase nystagmus was already found by Bárány (1907) after a rotation chair investigation, and was called: "Nach Nach Nystagmus". There is as yet no agreement about the explanation of secondary-phase nystagmus and its cause. In the caloric induced nystagmus, Thornvall (1932) sometimes noticed a secondary-phase nystagmus, mostly two minutes after an irrigation. According to the author a change in the position of the head did not change the direction of the secondary-phase nystagmus, so that he presumed a central origin. Dohlman (1938) demonstrated that during labyrinthine stimulation the cupula does not only deviate but is also distorted. Jongkees and Groen (1946) assumed that a secondary-phase nystagmus and secondary after-sensation might also be caused by elastic after-effects (hysteresis) inside the structures of the cupula itself (i.e. might have a peripheral origin). According to Jongkees (1948) the temperature wave during a prolonged caloric

stimulation, eventually reaches the medial part of the lateral semi-circular canal while the lateral part is already regaining bodytemperature and may cause there the nystagmus to change in direction. Aschan and Bergstedt (1955) noticed a secondary-phase nystagmus after rotatory, as well as after optokinetic and caloric (simultaneous hot-cold irrigation) stimulation, and therefore believed them to be of central origin. According to Hamersma (1957) a secondary-phase nystagmus is always accompanied by a directional preponderance (in the duration) and therefore is one of the expressions of a latent nystagmus. McLay et al (1957) named the secondary-phase nystagmus following optokinetic stimulation "contranystagmus" and they often obtained it in individuals who show some directional preponderance to caloric testing. In 1961 Jongkees mentioned that changes in the position of the patient after the end of the caloric reaction provoke secondary-phase nystagmus during a long period (influence of otoliths?). Milojevic and Allen (1967) attributed it to an efferent feed-back mechanism, producing a transient imbalance of the vestibular nuclei. They found the occurrence of the secondary-phase nystagmus more frequently in normal subjects than in patients.

In 8 of our 17 subjects we found a secondary-phase nystagmus (in 13 of 68 recordings). The intermittent period between the end of the primary-phase nystagmus and the start of the secondary-phase nystagmus proved to be very variable, varying from one half to two minutes. These quantitative findings correspond rather well with those of Hamersma. The secondary-phase nystagmus is usually slow and can be either continuous, or appears in groups interrupted by pauses.

Two of our eight subjects showed secondary-phase nystagmus to the left, three to the right and three a secondary phase-nystagmus in both directions. We were able to confirm the simultaneous occurrence of secondary-phase nystagmus in one direction with a directional preponderance (in the duration) in that same direction (Hamersma 1957). We were, however, not able to confirm the fact that directional preponderance was more pronounced in the test-subjects who had a secondary-phase caloric nystagmus; e.g. table II (see page 25). According to the criteria of Maas (1960) test subject no. 11 shows the most pathological value concerning the directional preponderance (in duration) but without the occurrence of secondary-phase nystagmus; according to the criteria of Jongkees (1948) we find 6 subjects (no. 2-5-8-9-11-12) showing a pathological directional preponderance but only one of them shows a secondary-phase nystagmus. Therefore we cannot agree with the conclusion that the secondary-phase nystagmus is an expression of a latent nystagmus. We can hardly believe that a latent nystagmus can occur in both directions as we sometimes see in the secondary-phase nystagmus. We are of the opinion that in general there must be some central genesis because of its occurrence in optokinetic nystagmus.

In 10 recordings there is also a vertical component present during the secondary-phase nystagmus (Fig. A24). In three cases the vertical nystagmus had already changed in polarity. In one recording alternate

beats in both directions occur (Fig. A25). In the others the vertical nystagmus persists in the same direction or is extinguished. Usually the horizontal secondary-phase nystagmus does not run synchronously with the vertical. The vertical nystagmus is usually more frequent.
We are of the opinion that caloric secondary-phase nystagmus in itself has no diagnostic value.

B. Some other phenomena

1. *Vertical nystagmus*

With E. N. G. Fluur and Ericksson (1961) found a vertical nystagmus in 80 % of normal people. Also Bos, Oosterveld, Philipszoon, Vozza and Zelig (1963) and Jongkees and Philipszoon (1964) found a spontaneous vertical nystagmus in 80 % of their normal subjects. In the majority of cases the vertical nystagmus was directed upwards, in a few downwards or varying in direction. The authors concluded therefore that a vertical nystagmus in the E.N.G. of a subject with closed eyes is not necessarily pathological. Kornhuber (1966) also found that a slight spontaneous vertical nystagmus behind closed eyelids is physiological. Later in his publications this author established that every vertical nystagmus is of central origin. In 6 subjects with a spontaneous vertical nystagmus, Fluur (1962) found no change of its parameters during caloric stimulation. In subjects without spontaneous vertical nystagmus he did not find a vertical component during the caloric test either. In the first group at the beginning of the stimulation he observed a synchronous nystagmus in both leads. Later on the vertical persisted with the same frequency while the frequency of the horizontal decreased. However, the fast phases remained synchronous. In 1967 Aboulker and Laurent found a vertical component during caloric stimulation in a great number of patients after head injury. According to these authors this component can be present after all four stimulations or only after one of these. Depending on the kind of stimulation the polarity can change or remain the same. They found that the vertical component mostly follows the rhythm of the horizontal component, also passes through a culmination point, and sometimes persists a few seconds after the end of the horizontal component.
In 22 % they found a vertical component directed upwards and in 31 % downwards. According to them the vertical component is not always synchronous with the horizontal lead and sometimes precedes it by a fraction of a second, especially if its average amplitude is larger than that of the horizontal component. In a group of subjects composed of normal individuals and patients Gabersek and Jobert (1968), investigated recordings of caloric nystagmus, paying special attention to the vertical lead. In 80 % of their recordings they found an upward-vertical component following hot irrigation. They assumed these findings to be normal. As pathological they considered the occurrence of a spontaneous vertical nystagmus, a vertical component which differs in direction to that described above, the absence of a vertical component during one or two ir-

rigations at different temperatures, preponderant amplitude or longer duration of the vertical component.
Six of our 17 subjects showed a vertical nystagmus, in the position we used for the caloric test (i.e. in the supine position with the head flexed forward over an angle of 30°). In all these cases the vertical nystagmus was directed upwards. In the sitting position 2 of our subjects showed a downward nystagmus which disappeared in the supine position. In those with a vertical nystagmus in the supine position the speed of the slow phase varied between 2° and 22° per second (Fig. A26). In the 2 subjects with the highest speed of the slow phase, groupformation of the vertical nystagmus occurred (type III). In 2 other subjects we recorded vertical eyemovements which we did not identify as nystagmus. Our findings do not exactly agree with those of Fluur (1962). In most of our subjects with an upward vertical nystagmus the polarity changed following hot stimulation. When caloric nystagmus appears in the horizontal and vertical lead, most fast phases occur synchronously in both. The frequency of the nystagmus in both leads, however, does not always remain the same. Sometimes nystagmus-beats occur in the vertical lead, and not in the horizontal or vice versa.
During the culmination period 22 ($^1/_3$) of our 68 recordings showed a downward, and 30 (nearly ½) an upward nystagmus. Thus our percentages do not correspond with those of Aboulker e.a. (1967). The speed of the slow phase and the amplitude of the vertical nystagmus are often hard to determine when they are disturbed by blinking (Fig. A27). In 9 recordings we found the amplitude of the vertical component as large or larger than that of the horizontal. The subjects showing a vertical component of larger amplitude than that of the horizontal also showed a vertical spontaneous nystagmus when examined in the supine position. Sometimes the culmination point of the vertical nystagmus coincided with that of the horizontal, but in most cases it occurred later. Following caloric stimulation the vertical component can begin before or after the horizontal, irrespective whether or not a spontaneous vertical nystagmus was already present in the subject in the supine position, and in most cases it remains longer.
We were not able to confirm the findings of Aboulker e.a. (1967), that the horizontal nystagmus is preceded by the vertical by a fraction of a second. As we already mentioned, we found that the fast phases occur synchronously. We already rejected the theory of Gabersek that every spontaneous vertical nystagmus must be considered an anomaly, and agree with Fluur e.a. 1961, Bos e.a. 1963, Jongkees e.a. 1964, and Kornhuber 1966. During the caloric nystagmus all our subjects showed a vertical component in one or more recordings.
In our series of experiments we found only 2 examples (subject no. 6 and no. 8) of what Gabersek e.a. (1968) considered to be the anticipated direction of the vertical component. Besides subject no. 6 shows an upward directed spontaneous nystagmus in supine position. Gabersek's opinion that a vertical component is pathological if its direction does not correspond with the anticipated one seems very unlikely to us. Fuur's hypothesis that the fast phases of the horizontal and vertical nystagmus

are created by the same centre, seems a little rash. In spite of the fact that a slow phase is present in both the leads, we often see that one lead triggers off a fast phase and the other not. Furthermore we observed that the duration of the fast phases in both leads can differ (see intermittent fast phase). These findings do not correspond with the existence of a common centre that triggers off fast phases of both the horizontal and vertical nystagmus.
So we can conclude that sometimes following stimulation of the horizontal semi-circular canals, the vertical component can be more important (as well in amplitude as in the speed of the slow phase) than the horizontal component. In normal subjects this can only occur when a spontaneous vertical nystagmus already existed before the irrigation.

2. *Jerks, Kipp-nystagmus, square waves*

Jung and Mittermaier (1939) described the occurrence of "quadratisches Hin- und Herrücken der Augen" in optokinetic nystagmus. Hamersma (1957) discussed certain to and fro movements of the eye (which can also occur repeatedly), in which case he called it "table-mountain phenomenon".
McLay e.a. (1957) saw the appearence of square-waves afther the caloric nystagmus and before the secondary-phase nystagmus.
Kornhuber (1966) described the spontaneous occurrence of jerks with an alternating horizontal direction in a patient with his eyes closed. This is a normally occurring phenomenon that according to the author increases during psychological stress and decreases with relaxation. And, as can be expected, in most cases he saw the occurrence of jerks simultaneously with a pronounced vertibular reaction. The appearance of lively jerks with a diminished vestibular reaction is therefore a more significant symptom than a decreased irritability with pendular deviations of the eyes. According to the author the amplitude in normal jerks was usually smaller than 10° (Fig. 4, no. 7). Very occasionally he observed a pathological diminished inhibition of the jerk-function with an amplitude from 20 tot 50° (Fig. 4, no. 10) in alternate directions; this he called "Kipp-deviation". This phenomenon occurred mostly in cases of spinocerebellar atrophy and in residual lesions following head injury. In 13 % of their patients with head injury Aboulker e.a. (1967) found nystagmus-beats of varying direction (nystagmus basculans or Kipp-nystagmus: Fig. 4, no. 9), appearing spontaneously at the end or during the reaction. Van de Calseyde e.a. (1969) noticed the appearance of this phenomenon especially at the end of the postrotatory nystagmus.
In supine position two of our subject (Fig. A28) showed large frequent jerks from 10° to 15° (subjects no. 3 and no. 13). In the others there were either no jerks or jerks smaller than 5°. The two above-mentioned subjects, also, had a strong caloric reaction, which corresponds with the findings of Kornhuber, as far as speed, frequency, and amplitude are concerned. However, this does not exclude the fact that strong reactions can occur in other subjects who, nevertheless do not show jerks. During the caloric reaction jerks can appear as counter-jerks (Fig. 4, no. 7a, Fig.

A17), i.e. jerks in a direction opposite to the anticipated fast phase of the nystagmus. When these jerks are consecutive and in the same direction, and when the speed of the slow phase between the jerks is very low, the recording shows the form of a staircase (Fig. 4, no. 7b, Fig. A25). The counter-jerks can alternate with jerks, resulting in a square pattern (Fig. 4, no. 8, Fig. A29, A30). When the amplitude of this pattern is greater than 15° to 20°, and a long time-constant of the apparatus is used as in our case, one speaks of a Kipp-deviation (Fig. 4, no. 10, Fig. A29, A30); Kipp-nystagmus or nystagmus basculans (Fig. 4, no. 9) are the names for the same phenomenon when a short time-constant is used. Jerks occur far less frequently in the vertical lead. This is also a reason why we consider it unlikely that the centre of the fast phase is the same for the horizontal as for the vertical nystagmus (Fluur 1962). We see jerks as an expression of the fast phase mechanism. During most of the recordings the jerks do not exceed 10°. In the two subject who already had jerks ranging from 10° to 15° in the suppine position, their amplitude increased from 20° to 50° after caloric stimulation (Fig. A29, A30). We assume that caloric stimulation facilitates the jerk-function of the eyes. Mostly jerks occur at the end of, or just after the caloric nystagmus, and can be observed up to two minutes after the last nystagmus-beat.

When jerks are very frequent they occur either continuously or in groups, when they are infrequent they are isolated. They often occur just before caloric nystagmus starts and seldom during the culmination period.

To conclude we can state that jerks are a normal finding. When they occur spontaneously, we shall in most cases find strong caloric reactions.

3. *Paroxysmal ocular state*

In 1966 Gabersek described the occurrence of eyemovements which form a recognizable entity (i.e. "état oculaire paroxystique" or paroxysmal ocular state).

According to the author each entity in general consisted of four consecutive up- ("mouvement de libération ou inhibition": inhibits the nystagmus), and down eyemovements ("mouvement de facilitation" which again initiates nystagmus; Gabersek and Jobert 1965) ending with an upward movement. The whole entity had a constant duration of about 20 seconds. He assumed that, in view of the constant duration of the phenomenon, this could be an expression of an autonomous alternating activity of the vestibular nuclei. This autonomous alternating activity in turn would be initiated by a dysfunction of a cortical or subcortical area with a vestibular or oculomotor projection. If there was a large dysfunction, the author noticed that this P.O.S. was present, even if the patient kept his eyes open. Gabersek (1966) and Gabersek and Scherrer (1967) observed these P.O.S. especially in central pathology, such as congenital nystagmus, Parkinsonism, head injury etc.

In 1967 Gabersek defined P.O.S. as recurring groups of spontaneous oblique (predominantly vertical) eyemovements of great amplitude each group having a constant duration of 20 seconds. He noticed that each

caloric stimulation also initiated a P.O.S. (i.e. irrigation P.O.S.). This irrigation P.O.S. occurred at the moment the irrigation was started or after a period varying from 10 msec. to a few seconds. It was characterized by an upward eyemovement which could recur several times. The author often observed that the patient withdrew his head and sometimes recorded electrical activity of the peri-ocular muscles in the electronystagmogram, these phenomena occurring simultaneously with the irrigation P.O.S. According to the author the actual ocular response (i.e. nystagmus) can only occur at the end of such a P.O.S. and at least 20 seconds after it had begun. If a spontaneous P.O.S. existed, it can interfere with the irrigation P.O.S. and so delay the nystagmic response. The author noticed that when there was a spontaneous P.O.S., it continued during the reaction. Gabersek (1966) and Gabersek and Jobert (1968) observed that there was practically no latent period during torsion swing, rotatory- and galvanic stimulation, and so hypothesized that the selective stimulation of one labyrinth in caloric nystagmus causes the irrigation P.O.S. (the duration of the P.O.S. corresponds with that of the latent period). According to the author the first nystagmus-beat of each caloric reaction is always preceded by an oblique eyemovement that occurred 20 seconds previously.

When investigating our subjects in order to find position nystagmus we mostly observe that in the supine position the vertical eyemovements are more restless than the horizontal ones (Fig. A31). Only in two subjects the horizontal eyemovements were more restless since in one case violent jerks or square-waves (Fig. A28) occurred and in the other pendular movements (Fig. A33). Mostly sinusoidal movements with a maximum amplitude of 30° (Fig. 4, no. 13) or a vertical nystagmus or a mixture of both occur in the vertical lead (Fig. A26).

However, two subjects (Fig. A31 and Fig. A32) show P.O.S. (as described by Gabersek: Fig. 4, no. 14) in the supine position. These P.O.S. occur in groups with a duration varying from 20 tot 30 seconds or isolated with a duration of several seconds. So we were not able to confirm the observation of Gabersek that the intervals between the P.O.S. as well as the duration of the P.O.S. are regular. Their amplitude is larger than 30°, mostly between 40° and 60°. We should like to draw attention to the fact that different positions can change the P.O.S. especially as far as the frequency is concerned.

We cannot see the P.O.S. and the slow sinusoidal movements of a maximum amplitude of 30° as an expression of a central lesion, but rather as the absence of a visual fixation point with as a result a drift of the eyes.

During calibration we do not see these movements, (except now and then blinking movements), not even in subjects who had spontaneous P.O.S. with closed eyes.

We mostly see a more or less pronounced upward eyemovement followed by a slow return to the mid-position at the beginning of an irrigation (without any latent period: Fig. 4, no. 15). This movement can be followed by sinusoidal movements. Sometimes the irrigation P.O.S. can be very insignificant or absent. Its duration can be longer or shorter than 20 seconds (Fig. A3, A4). Usually no nystagmus occurs before the 20th second (only in 22 of the 68 recordings the nystagmus begins between the 15th and 25th second), but in most cases it begins between the 30th and 50th second (Fig. 2). Our opinion is that the beginning of the nystagmus is not due to the cessation of the P.O.S. but rather due to the temperature gradient wave, that at that moment reaches the lateral semi-circular canal (Schmalz 1931, Cawthorne e.a. 1954). We also see that P.O.S. occur during the caloric reaction (Fig. A7, A21, A22) in subjects who have no spontaneous P.O.S. We assume that this is due to drifting of the eyes induced by the labyrinthine stimulation. From our observation it follows that the P.O.S. occurred mainly in the vertical plane.

The occurrence of the irrigation P.O.S. can in no way be provoked by labyrinthine stimuli, since there is no latent period. We are inclined to explain the irrigation P.O.S. which occurs mainly in the vertical plane as a certain defence mechanism which consist of tightly closing the eyes followed by an upward turning of the eyes due to the presence of an unpleasant stimulus.

One must remember that the external auditory meatus is partially innervated by the trigeminal nerve (the auriculo-temporal nerve: Burger 1938).

So there exists a great morphological difference between the irrigation P.O.S. and the spontaneous P.O.S. The irrigation P.O.S. consists mainly of a fast upward eyemovement of very large amplitude followed by a slow return to the midposition. The spontaneous P.O.S. mainly consists of consecutive up and down eyemovements of large amplitude.

We conclude that the sinusoidal movements, the spontaneous P.O.S. and the irrigation P.O.S. are normal findings that have no vestibular cause.

4. *Sinus waves or pendular movements*

Hamersma (1957) described sinus-shaped recordings at the end of a caloric nystagmus as a result of slow and fairly regular pendulumlike movements of the eye. The author attributed this phenomenon to the relaxed condition of the test subject. Kornhuber (1966) described rhythmical eyemovements in the horizontal plane, in some cases with a slight vertical component and at a frequency ranging from 0.25 to 0.3 per second, and an amplitude between 5° and 60°, especially after an extensive and tedious nystagmographic investigation. On the strength of their frequency-characteristic analysis of the eyemovements during sinusoidal stimulation, Trincker, Sieber and Bartual (1961) and Trincker (1961) believed in the existence of a programming system for the eyes with its own inborn frequency of 0,3 seconds. The frequency corresponds very well with the frequency of the pendular movements. These sinus waves might occur as a result of a facilitation of the action of this programming system. Kornhuber pointed out that pendular movements should not be confused with the galvanic skin reflex (Fig. A5) that occurs especially in the vertical lead and is usually of greater amplitude and slower frequency. Kornhuber's galvanic skin reflex resembles a P.O.S. of large amplitude. The main difference between a P.O.S. and a galvanic skin reflex is that only the former influences the horizontal recording. According to

Lange and Kornhuber (1962) these horizontal pendular movements occur more frequently after head injury. Aschan (1967) described sinus waves caused by a decreased state of alertness in patients under hypnosis and in a woman with a metastasized breast carcinoma. We shall use the terms pendular movements or sinus waves in describing the regular sinusoidal recording of an amplitude of about 40° in the horizontal lead (Fig. 4 no. 16).

Three of our subjects (table no. II: see page 25) showed pendular eyemovements in the supine position (Fig. A33) and four recordings (three different subjects: no. 1-5-11) showed these movements after the end of the caloric nystagmus (Fig. A34). The frequency of the sinuswaves is in agreement with the values found by Kornhuber. The amplitude of horizontal pendular movements was usually about 40°, the amplitude of the vertical lead never exceeded 10°.

Why should there be more pendular movements in the horizontal than in the vertical lead? It must not be forgotten that the centres concerned with horizontal and vertical gaze-movements are more or less separate and their projections run in separate pathways (Biemond 1936, Crosby 1953).

All pendular movements indeed occurred after cessation of caloric nystagmus. Of the four recordings that showed pendular movements, there were two which had a slow speed of the slow phase, and two which had a fast speed of the slow phase during the culmination period of the nystagmus. Thus the speed of the slow phase did not influence the appearance of pendular deviations. The occurrence of these movements at the end of the reaction and the fact that the patients felt asleep during his period, show that they are caused by sleepiness.

5. *Aberrant apices*

In cases of partial reticular lesions Kornhuber (1966) described a slowing down of the fast phase in the vestibular as well as in the optokinetic nystagmus, paired with a rounding of the normally clear-cut apex of the return point between the fast and slow phase. Dufour e.a. (1966) found the same rounding of the nystagmic apex in central lesions of the bulbopontine region. In the horizontal lead in all our recordings we found that the transition between fast and slow phases were sharp. We very rarely saw a slight rounding of the apex (Fig. 4, no. 12). This frequently occurred in the vertical lead (Fig. A1, A11, A20, A23, A24, A27).

In the horizontal lead we noticed a different phenomenon, i.e. the absence of the apex of the nystagmus-beat, i.e. a decapitated nystagmus-beat (Fig. 4, no. 11). At a certain point the speed of the slow phase changed suddenly. At the same time we observed the occurrence of a fast phase in the vertical lead (Fig. A11, A12). Once more we see that we must have at least both a vertical and a horizontal lead if a qualitative analysis of the nystagmogram is to be obtained.

6. *Blinking*

We often recorded blinking of the eyelids in the vertical lead. This most-

ly coincides — but not always — with the horizontal and vertical fast phase (Fig. A27). Blinking occurs more often during the fast phase of the horizontal component than of the vertical (Fig. A35).
Occasionally they also occur during the slow phase of both (Fig. A36). Blinking as seen in the recording of the vertical component is often paired with a jerk in the horizontal lead. If the vertical component is directed downward, blinking can cause a deformation of the latter, so that it is impossible to determine its direction. (Fig. A37, last part). We do not consider blinking to be pathological, even when it occurs very frequently.

B. The caloric test with arousal

1. *Literature review*

Previously we already mentioned the influence of arousal or alerting techniques on caloric nystagmus.
Grahe (1923) pointed out that certain tactile stimuli produced an inhibiting effect on the caloric nystagmus.
Mowrer (1934) found in pigeons that bodily rotation which was preceded by a "rest" period (during which the birds were immobilized and hooded) usually elicited a nystagmus of significantly shorter duration than did rotation which was not preceded by such a period.
Hennebert (1946) found inhibition in an induced as well as in a pathological spontaneous nystagmus following: massage of the external auditory meatus, and after rubbing the same area with alcohol.
In 1948 Hulk and Jongkees were of the opinion that the frequency of the postrotatory nystagmus could be influenced by making the patient concentrate on some or other subject and in 1949 Jongkees thought that it was possible to influence the frequency of the caloric nystagmus by causing the patient to concentrate or by rubbing the cheek with alcohol.
Mahoney, Harlan and Bickford (1957) observed the effect of fixation, vision, eyeclosure and mental activity on caloric nystagmus. After the influence of fixation was abolished, there was an increase of the amplitude as well as a moderate increase of the duration and a decrease in frequency and amplitude stability. According to the authors eyeclosure increased the foregoing changes or often completely inhibited the caloric nystagmus. When this last phenomenon occurred mental activity or alerting frequently elicited brief bursts of nystagmus.
Anderson, Diamond, Bergman and Nathanson (1958) usually found that eyeclosure generated the maximum amplidude and the minimum frequency of the caloric nystagmus. Only in three subjects they noticed that eyeclosure provoked an immediate inhibition of the caloric nystagmus, which occasionally could be reestablished by a sudden loud noise or an unpleasant stimulus.
In cats Crampton and Schwam (1961) noticed a reduction in nystagmus (i.e. duration and amplitude of the slow phase) after repeated rotation. But alerting the animal by sounds occasioned only a temporary and partial recovery of the nystagmus.

Lidvall (1961) observed that a sudden verbal presentation of an arithmetical problem promptly elicited a regular nystagmus from a state of severe dysrhythmia.
Collins, Crampton, and Posner (1961) studied the influence of various tasks on rotatory nystagmus. These different tasks were: daydreaming, making key pressing-signals to indicate the sensation, and mental arithmetics. The two latter tasks increased the nystagmus output.
During the rotation test with open eyes Collins and Guedry (1962) observed qualitative as well as quantitative differences of the nystagmus as a function of arousal level. During mentally active states they found no evident decline of nystagmus during stimulation. During states of mental relaxation they observed a reduced nystagmic response (i.e. reduced duration and slow-phase velocity). They also described several eyemovement patterns, such as:

a. normal nystagmus.
b. reduced displacement and velocity of slow phase, with fast phase approximately normal.
c. large amplitude, low frequency nystagmus (less regular and more rounded) with slow- and fast-phase velocity greatly reduced.
d. large amplitude, low velocity sinusoidal oscillation with very-low amplitude nystagmus superimposed.
e. large amplitude, low frequency sinusoidal oscillations and no discernable nystagmus.

Collins, Guedry, and Posner (1962) studied the influence of mental activity on nystagmus following caloric stimulation. They found that the alert state produced nystagmus of greater amplitude and longer duration and yielded a smoother recording. During reverie-trials they never found eyemovement patterns c, d, and e. They believed that caloric stimulation is probably not conducive to full relaxation.
During rotatory stimulation Collins and Poe (1962) found no difference between the responses of a "naive" group (i.e. subjects with no previous turn table or caloric stimulation experience) and an "experienced" group, both groups showed a significant increase in nystagmus while solving arithmetical problems, and no significant effect of amphetamine on the total output of slow-phase nystagmus, but a significant effect on the form of a longer vestibular reaction.
Aschan, Finer, and Hagbarth (1962) observed that suggestion — e.g. making the patient believe that his rotatory sensation increased or decreased — influenced the nystagmic response as well as the eye speed of the slow phase during rotatory experiments whereas in the caloric experiments only the eye speed was affected.
In normal subjects Sokolovski (1963) investigated the influence of different mental states on the rotatory nystagmus. These were: normal test (i.e. dark room, eyes lightly closed, in silence), question test (i.e. same test conditions but the subject has to answer questions), drops test (i.e. the subject has to count silently to himself drops falling in front of him at the level of his eyes), writing test (i.e. the test-subject has to write a

dictated test on paper), dim-light-spot test (i.e. fixation of a lightspot or oculogyral illusion). The author concluded that during the question-test all quantitative parameters were increased and that a marked stability of frequency and amplitude existed.

In a patient Aschan (1964) observed that opening the eyes and fixing on a point a few meters away can make a well defined caloric nystagmus disappear. Sometimes caloric nystagmus did not appear behind closed eyelids, but if the patient had some kind of arousal effect such as solving a mathematical problem, a normal nystagmus was recorded. The author sometimes noticed irregular pendular eyemovements without nystagmus, but various arousal-stimuli caused this slow sinuswave-like recording to change to a nystagmus.

Ormerod (1964) found that the presence of a noise in the testing room interrupted the rhythm of the nystagmus. Conversation among third persons and any sudden movement or noise inhibited nystagmus. The author noticed that in complete silence some individuals responded with no or a poor nystagmus, but when subjected to simple conversational matter, better or more regular responses were obtained. In some subjects the playing of music led to a reduction in the amplitude or in the duration of nystagmus.

Crampton (1964) studied the habituation process during the rotatory test. In cats he found that arousal measures can produce brief periods of increased nystagmus activity. The author noticed that habituation could not wholly be recovered with arousal measures since auditory stimuli appeared to become less and less effective in eliciting nystagmus. He found that frequent cutaneous electric shocks and interperitoneal administration of d-amphetamine, attenuated the response-decline but did not prevent it. In man he noticed that mental arithmics produced more nystagmus than the lesser task of psychophysical judging. The author pointed to the fact that there are procedural difficulties in the study of habituation in man. Modern-day experimental subjects all have a history of exposure to a wide variety of acceleration experiences and therefore the greatest part of habituation may already have occurred before the experiment was started.

In cats Milojevic and Voots (1965) found that a lack of mental activity inhibited nystagmus and blindfolding increased the duration of nystagmus. In 1966 the same authors pointed out that the otolith system may act as a controling or regulatory mechanism over the duration of post-caloric nystagmus.

Sokolovski (1966) investigated experimental subjects in three different mental states. These were as follows:

— when visual fixation was absent and mental activity reduced
— when both visual fixation and mental activity were present
— when mental activity was present but visual fixation was absent.

Since in the sequence of these three tests six different combinations are possible, he used six subjects. He did this in order to avoid any possible influence of response-decline or habituation. In the third above mentioned mental condition he found the strongest reaction and the least dysrhythmia.

Gabersek and Jobert (1966) distinguished two totally different results of arousal. In subjects undergoing a first labyrinthine test (i.e. "naive"), nystagmus was suppressed by conversation during the first irrigation. In patients who had undergone repeated tests (i.e. "experienced") conversation resulted in an increase of the nystagmus. For this reason the authors advised the performance of one irrigation without recording, one week prior to the first investigation, or to conduct the first test without speaking to the "naive" patient. In the second group they gave simple tasks, e.g. saying the alphabeth or counting.

Coats (1966) remarked that when a subject solves mathematical problems his nystagmus becomes intenser and the occurrence of pauses decreases.

Barber and Wright (1967) pointed to a number of variables which could influence the nystagmus and called these the three "E"s:

1. Emotional factors:
   a. general emotional states of the patient: motivation, apprehension, stability.
   b. patient reaction to a "fearsome" test situation and atmosphere.
   c. familiarity with test procedures.
   d. patient-technician report.
2. Environmental factors:
   a. abolitions of visual cues: *most important*.
   b. unexpected noises (e.g. subway, observers conversation, footsteps).
3. Endogenous factors:
   a. state of wakefulness: *most important*.
   b. endogenous stimuli (e.g. headache, other troublesome body sensations).
   c. intellectual capacity.
   d. severe bilateral deafness.
   e. blindness, extra-ocular-muscle defect.
   f. drugintake, e.g. barbiturates, alcohol.

In order to avoid these variables as much as possible the authors suggested various methods. Before actual recording began, the technician chatted with the patient discussing such matters as the weather, occupation, family in order to try to relax the patient. During the investigation sample questions were asked such as: "Are you married" etc. Even though this method caused an arousal effect, the authors found it difficult to standardize. A second method consisted in the giving of "set tasks". Before the investigation, the patient was given an order which he had to work out during the investigation, like: "count backwards by 7 from 100" or "name as many words as you can that starts with the letter t". These tasks, however, have the disadvantage of being too frustrating to the illeducated. Therefore the authors tried to compromise. Several questions of varying difficulty were recorded on tape e.g. "What to do when you are hungry?" and "What is the square root of 625?". The patient is instructed to listen carefully to each question and to do his best to answer. He is also reassured that some questions are most difficult and that he should guess if he does not know the answer. The list of questions was offered to the patient through a speaker mounted near the patient's head.

Henriksson, Janeke, and Claussen (1969) indicated two possible techniques for arousal: i.e. either to record with open eyes in the dark or to present the patient with a mathematical problem.

Jongkees (1969) stated that the best results were obtained by recording the nystagmus in the dark from a mentally active patient with the eyes closed.

During torsion-swing tests van de Calseyde, Ampe, and Depondt (1969) often found the occurrence of pauses due to the lack of arousal. The suggested several arousal techniques like: encouraging the patient during the investigation, presenting him a set task (mathematical- or language problem).

To conclude we can state that the degree of mental activity plays an important part concerning both the quantitative parameters and the regularity of the nystagmus.

Many investigators have to keep the degree of mental activity at a constant level in order to avoid wrong clinical conclusions. Keeping the mental activity at a constant level was obtained in several different ways: e.g.

1. tactile stimulation: rubbing the cheek with a brush (Grahe 1923) or with a piece of cotton soaked in alcohol (Hennebert 1946, Jongkees 1949).
2. concentrating the attention: (Hulk e.a. 1948, Jongkees 1949), encouraging the patient (van de Calseyde e.a. 1969), pushing buttons according to the sensation (Collins e.a. 1961, 1962).
3. applying electric stimulation to cats (Crampton 1961, 1964).
4. drugs like amphetamine (Collins 1962, Crampton 1964).
5. hypnosis with or without the suggestion of a turning sensation (Aschan e.a. 1962).
6. degree of fixation: the eyes open or closed in the dark (Mahoney e.a. 1957, Anderson e.a. 1958, Collins e.a. 1962, Sokolovski 1963 and 1966, Aschan 1964, Henriksson e.a. 1969, Jongkees e.a. 1969).
7. auditory stimuli: noises (Anderson e.a. 1958, Ormerod 1964), conversation among third persons (Ormerod 1964), music (Ormerod 1964), bell or buzzer (Crampton 1961, 1964), conversation with the test subject, tasks, or mathematical problem (Lidvall 1961, Collins e.a. 1961, 1962, 1962, Sokolovski 1963, Aschan 1964, Coats 1966, Barber e.a. 1967, Jongkees 1969, van de Calseyde e.a. 1969).
8. writing: (Sokolovski 1963).

All these techniques were used both separately and together. These arousal techniques also have different results, depending on whether they are used in animals or in man (Crampton 1964) and when they are used in human beings, the experience with vestibular testing plays an impor-

tant part (Collins e.a. 1962, Gabersek e.a. 1966), as well as the kind of vestibular stimulation (rotation or calorization: Collins e.a. 1962).

2. *Choice reaction task*

We indicated before (page 11) that the eyes-closed technique in a dark room was used in the majority of our cases and caused the most regular nystagmus. One has to see to it that the eyes are not closed too tightly, for in that case other phenomena interfere, like Bell's phenomenon (Aschan e.a. 1956, Anderson e.a. 1958). Recording with closed eyes also has the advantage of producing an alpha-rhythm on the E.E.G. recording. When arousal techniques are used their influence on this alpha-rhythm can be traced.

Since in literature auditory stimuli have been the most often described stimuli to obtain a state of arousal, we too applied this technique. Noises alone are not enough to keep the test subject awake. He can easily fall asleep even with the noise produced by the waterpumps in the background. Together with the noise one must stimulate the attention of the patient. Since, however, intellectual problems may be frustrating, we decided to stimulate the attention of the test subject by presenting him a very simple mental task e.g. making a choice. Giving a simple mental task has the advantage that talking to the patient during the investigation is not necessary, so every possible suggestion can be avoided (Aschan e.a. 1962). An other advantage is the easy dosage of this technique. For this reason we choose the binary-choice generator (BCG), described by Ettema (1967). Through a bone conductor on the forehead (to avoid an eye deviation in the direction of the noise) two bursts of white noise in succession — one with 50 dB SL and another with 60 dB SL intensity — are offered in random order. One should not use too strong noises, for they cause an inhibition of the nystagmus (Burgeat, Freyss, Fontelle and Burgeat-Menguy 1966). The test subject has two buttons at his disposal. If the first noise is the loudest, he should push one button and if the first noise is the weakest, he should push the other. With the aid of separate counters we recorded: the total number of pairs of noises offered, the total number of pairs with the loudest noise first, the total number of correct answers and the total number of wrong answers. The apparatus was fixed at a set speed of offering noises ("paced") i.e. 30 pairs of noises a minute. The BCG presents a simple task which is easy to dose. This technique represents an aspect of mental activity i.e. the making of a choice. If one does not make a choice and pushes the same button all the time, there is still a 50 % chance of correct answer.

Before starting the actual caloric test, each test subject had to practice for a couple of minutes in order to be able to perform his set task as well as possible. After practicing with the BCG, the actual caloric test was started. The caloric stimulation was performed in the same way as during the first series. The BCG came into action at the end of the irrigation, i.e. between the 30th and the 35th second, because the noise produced by the syringing of the water would interfere with the noise offered by the BCG.

3. *Results*

I. THE EFFECT OF AROUSAL ON THE QUANTITATIVE FINDINGS

During the investigation with the BCG we obtained an average score of 91 % correct answers. This indicates that none of the test subjects had pushed the buttons at random and therefore we can state that a definite mental activity was present.

The first series without mental activation gave 151 seconds as the mean value of the duration of the caloric reaction. During the period between the 60th and 120th second we found the following mean values:

maximum speed of the slow phase: 39°/sec.
total amplitude: 623°
frequency: 85 beats/minute.

With the use of the BCG all these mean values increased. The mean duration of the reaction was 169 seconds i.e. an increase of about 10 %. During the period between the 60th and 120th second we found as mean values:

maximum speed of the slow phase: 45°/sec, i.e. an increase of 15 %
total amplitude: 808°, i.e. an increase of about 30 %
frequency: 94°/minute, i.e. an increase of 10 %.

If we calculate the mean amplitude per nystagmus beat for both series in the period between the 60th and the 120th second we get:

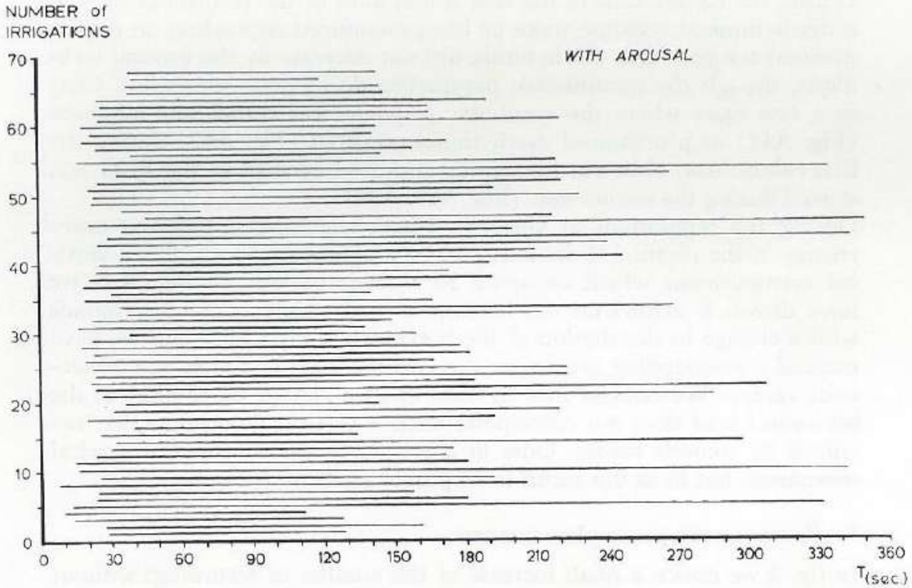
without arousal: $623° : 85 = 7{,}3°$ per beat
with arousal: $808° : 94 = 8{,}5°$ per beat, or an increase of about 15 %.

In other words, the mean amplitude per beat increases 15 %. Regarding the increase of the amplitude per beat of 15 %, and the simultaneous increase of the frequency of 10 % the total amplitude must have a minimum increase of 25 %. The further increase of the total amplitude is due to a decrease of the total duration of the pauses.

In the first series we saw that the maximum speed of the slow phase occurred at the 76th second. During the second series it occurred at the 71th second. So no significant change in the moment of occurrence of the culminationpoint appeared. In the first series the mean labyrinth-predominance (in the speed of the slow phase) was 11 % and the mean directional preponderance (in the speed of the slow phase) 9 %. With the use of the BCG, the mean labyrinth predominance is 6 % and the mean directional preponderance 7 %. Also the pathological labyrinth predominances and nystagmus preponderances (in the speed of the slow phase) of the first series disappeared (see table II: page 25, test subjects no. 2, 8, 9, 12, and 17).

In Fig. 5 (see page 67) the beginning and the endpoint of each caloric reaction is plotted. We read from this figure that in the periods between the 30th and 40th second (where we started the BCG) only one reaction had not started yet. The same figure of the first series (Fig. 2: page 26) shows us that in the same period, without the BCG, 13 reactions had not yet started. Fig. 3 (see page 26) shows that the number of reactions which stop between 180th and 360th seconds decrease more gradually than during the first series.

In general the sensation of rotation was weaker during the experiments with mental activation than during those without BCG.



Bij fig. 5. duration of each caloric reaction

II. THE EFFECT OF AROUSAL ON THE E.E.G.

The changes of the alpha-rhythm during the actual irrigation are the same as those in the first series. As said we started the BCG in the period between the 30th and 40th second. From that moment on we observed an interruption of the alpha-rhythm or a decrease of its amplitude. This change lasted from seconds to minutes, depending on the number of the irrigation (Fig. A38, A39) and the test subject (Fig. A40). At the end the alpha-rhythm always came back to normal (Megighian et al 1961). We also saw that the presentation of repeated auditory stimulation together with the choice test did not disturb the alpha-rhythm permanently. We might assume that after a "learning"-period the process of perceiving noises and the making of a choice, changes to a lower level in which the cortex is no longer involved as actively as before. We also noticed the occurrence of strong individual differences in the duration of the so called "learning"-period and found that every time again, with the starting of the BCG, the alpha-rhythm was disturbed for a shorter or longer time.

III. THE EFFECT OF AROUSAL ON THE QUALITATIVE FINDINGS

A. Dysrhythmia

During the caloric tests in the first series, most of the recordings showed a dysrhythmical response more or less pronounced depending on the individual subject. This dysrhythmia did not decrease by the arousal technique, though the quantitative parameters do increase somewhat. Only in a few cases where the vestibular response was completely inhibited (Fig. A41) or pronounced dysrhythmia occurred (Fig. A42) during the first caloric test, clear-cut nystagmus appeared as soon as the BCG was started during the second test. (Fig. A38, A39).
During the symposium at Geneva (1969) Gabersek stated that every change in the rhythm of the horizontal nystagmus is preceded by a vertical eyemovement which occurred 20 seconds earlier. In Fig. A43 we have drawn 8 arrows on the horizontal lead (each arrow corresponds with a change in the rhythm of the horizontal component) and we have marked corresponding arrows on the vertical lead, but each one 20 seconds earlier. We noticed that in most cases a change of rhythm in the horizontal lead does not correspond with a vertical movement that occurred 20 seconds before. Only in a few cases we do notice a vertical movement, but to us this seems to be purely accidental.

1. Pauses or the pause phenomenon

In fig. 3 we notice a small increase of the number of recordings without pauses during the culmination period, in other words arousal decreases the occurrence of pauses. This strikes us even more when we look at table X. If we compare the latter with table III (see page 31) we see that the frequency of pauses remains about the same for all periods of 30 seconds. We notice, however, that for the periods between 30 and 60 seconds, 60 and 90 seconds, and 90 and 120 seconds, the mean total duration of pauses, the mean duration of a pause as well as the mean duration of the longest pause have decreased significantly. This clearly

Table X.

caloric nystagmus in periods of 30 seconds	mean number of pauses per 30 seconds	mean total duration of the pauses in seconds	mean duration of a pause in seconds	mean duration of the longest pause in seconds
0"—30"	1	5,1	—	3,8
30"—60"	4	6,5	1,6	2,7
60"—90"	5	7,2	1,4	2,4
90"—120"	5	7,2	1,4	2,5
120"—150"	3	8,2	2,7	4,1
150"—180"	3	9,5	3,1	5
180"—210"	3	10,8	3,6	5,9
210"—240"	3	9,7	3,2	5
240"—270"	3	14,4	4,8	7,2
270"—300"	3	14	4,6	7,7

demonstrates the effect of the BCG on the pause-phenomenon. Furthermore, we can state that other findings regarding the pauses remain the same (i.e. highest frequency of the pauses during the culmination period, the longest lasting pause at the end of the reaction etc.).
We were not able to judge Hamersma's statement about the increase of the occurrence of pauses following repeated investigations, despite a few months' interval between the tests. There was an interval of a month between our two series, but the second series was performed with an arousal technique. We were able to state that the pauses decreased in duration by arousal. Therefore we are inclined to say that the increase of the pauses observed by Hamersma was probably due to the fact that, when the caloric test was repeated, the arousal effect of the caloric test itself had decreased.
In our series of 17 subjects we have one who shows a decrease in frequency of the nystagmus after the consecutive irrigations, three which show an increase in the total duration of the pauses during the consecutive irrigations, three who show an increase in the duration of the longest pause in the consecutive irrigations, and one who shows a gradual decrease of the duration of the longest pause. Even in this series we are not able to demonstrate a clear-cut response decline in the consecutive irrigations.
During the pauses the eyes make the same movements in the horizontal plane which occurred in the first series i.e. wave pattern (Fig. A44), rest (Fig. A43), tremor (Fig. A13). decapitated nystagmus (Fig. A45), rounding of the apices, nystagmoid movements (Fig. A46).
As far as the vertical movements are concerned, we can also differentiate between rapid up and down movements (Fig. A47), and slow movements (Fig. A48) which, in case the amplitude is large enough, causes the occurrence of a pause.
In conclusion we can state that the duration of the pauses decreased by arousal.

2. Anomalies during the active period

a. *Arhythmia*

During the second series arhythmia occurred less frequently. Only 20 recordings showed this phenomenon. In 11 recordings arhythmia appeared only before the 30th second. After we started the BCG the response became more regular (Fig. A39, A43). At the same time a downward movement occurred in the vertical lead ("mouvement de facilitation" of Gabersek).

b. *Intermittent fast phase*

With regard to the intermittent fast phase we do not find a significant difference with the first series. In 49 recordings they only occurred before or after the culmination period (Fig. A46). In 23 recordings they were observed during this period. We also noticed a change in the polarity of the vertical lead every time an intermittent fast phase occurred in the horizontal lead.

c. *Amplitude of abnormal size*

a. Dysmetria

In the arousal-series we find many more recordings which show dysmetria, this in accordance to our quantitative findings, where the mean amplitude per beat increased. In 30 recordings (during the first series only 12 recordings showed this phenomenon i.e. an increase of about 40 %) dysmetria is found. It also occurred before and after the culmination period (Fig. A39) but more frequently during this period (Fig. A39, A49) (26 recordings). In two cases it occurred only before and in two cases only after this period (Fig. A50). Dysmetria begins only after we started the BCG, (Fig. A39). This once more clearly demonstrates its effect.

b. "Petite écriture" and salvos

In 23 recordings we find short periods of "petite écriture" especially occurring during the culmination period and only rarely in the period after the 120th second. Salvos are seen in 28 recordings mostly during the culmination period. So we can state that the occurrence of "petite écriture" and salvos is not appreciably influenced by arousal.

d. *Varying amplitude or groupformation*

a. type I: groups in which the speed of the slow phase remains constant: The occurrence of type I is not influenced by arousal (compare Fig. A23 with Fig. A51). As far as this type of groupformation is concerned, we do not find a very clear synchronous course between the horizontal and vertical lead in any of the two series. Neither do we find a decrease in the occurrence of "crochetage".

b. type II: groups in which the speed of the slow phase does not remain the same. In the arousal series we also find the occurrence of groupformation type II. The amplitude of the deviation of the pendular movements is generally reduced and the groups occur especially at the end of a caloric reaction (Fig. A52). As we stated before, this type is an extreme form of the phenomenon responsable for the slow periodic changes in the speed of the slow phase. By our arousal technique these periodic changes in the speed of the slow phase more or less disappeared (Fig. A53 cfr Fig. A5).

c. type III: pause-groups. Under the head pause-phenomenon we already discussed this type (Fig. A43).

In this series too we mostly found a combination of the different types.

e. *Secondary-phase nystagmus*

The BCG caused a considerable increase in the occurrence of secondary-phase nystagmus. In 12 test subjects 20 recordings showed a secondary-phase nystagmus. Compared with the first series this represents an increase of 50 %. We also noticed that with the arousal technique some subjects showed a secondary-phase nystagmus that did not cause it in the first series.

We were not able to demonstrate a definite correlation between the appearance of a secondary-phase nystagmus and a directional predominance (in the duration). According to Jongkees (1948) a directional predominance (in the duration), worth its name, should at least show a difference of 20 % between the two sides and according to Maas (1960) the variation must be more than 30 % before it can be called pathological. Using the criteria of Maas we noticed that with arousal a pathological directional predominance decreases (as well in the duration as in the speed of the slow phase). Applying the criteria of Jongkees no decrease occurs for the appearance of 20 % directional preponderance (see table XI).

Table XI: Number of irrigations showing a directional preponderance:

	with arousal		without arousal	
	ss	d	ss	d
Maas	0 (0%)	1 (5%)	3 (17%)	2 (11%)
Jongkees		6 (35%)		6 (35%)

ss: speed of the slow phase
d: duration

In 1948 Jongkees observed nystagmus by means of Frenzel's spectacles and found that 17 % of his normal subjects showed a directional preponderance. This percentage is strikingly different to the 35 % we found. We are of the opinion that this difference is due to the fact that, by using E.N.G., we recorded nystagmus beats which would otherwise have been missed (Jongkees and Philipszoon 1964). One of the recordings showed a short-lasting inversion (three nystagmus beats before the end of the primary phase nystagmus: Fig. A54).

Both the latent period between the primary- and secondary-phase nystagmus and the duration of the secondary-phase nystagmus remained about the same with and without BCG.

B. Some other phenomena

1. *Vertical nystagmus*

During the culmination period we found 19 BCG recordings with a downward directed nystagmus and 39 with an upward directed nystagmus. In this series a downward directed nystagmus only occurred during irrigation with cold water. A spontaneous vertical upward nystagmus, if present, decreased after a cold stimulation, disappeared or sometimes changed its polarity. Most of the time the spontaneous vertical nystagmus was increased by a hot irrigation. When a spontaneous vertical nystagmus was not present the induced vertical component only occurred after the beginning of the horizontal nystagmus.

Sometimes both horizontal and vertical components begin the moment the BCG is started (Fig. A39, A43).

In conclusion we can state that the vertical component was not influenced by the BCG.

2. *Jerks, Kipp-nystagmus, square waves*

Also in the arousal series we noticed jerks during the entire reaction, mostly at the end, less frequently before the 60th second (Fig. A55) and sometimes during the culmination period (Fig. A56).
Although in two cases the number of jerks decreased, in every other case the number increased more or less. In general we also found an increase in amplitude of the jerks, under the influence of arousal.

3. *Paroxysmal ocular state*

With BCG the duration of the irrigation-P.O.S. and the amplitude remained almost identical. Again we found that most irrigation P.O.S. were shorter than 20 seconds (the duration that Gabersek indicated) and in most cases even less than 10 seconds (Fig. A43, A55). In a number of cases there existed practically no irrigation P.O.S. at all (Fig. A43). Sometimes the nystagmus had already started before the end of the irrigation P.O.S., when its duration was long; in most cases, however, it started a few seconds after the end of the irrigation P.O.S.
In the case of test person no. 1, who already showed a spontaneous P.O.S., we noticed that this P.O.S. became continuous on turning on the BCG, i.e. the vertical lead showed movements of larger amplitude, continuously going up and down. This continuous P.O.S. however, stopped before the end of the nystagmus (Fig. A47).
With the exception of one case, no influence of the BCG was noticed on the P.O.S.

4. *Sinus waves or pendular movements*

During the BCG test the pendular movements in two recordings (test person no. 1, Fig. A57) of one and the same test person had clearly increased. After the end of the caloric-induced nystagmus they were almost constantly present, at times interrupted by a short pause of a few seconds. The amplitude too had increased slightly (up to 50°-60°).
It is also remarkable that the arousal technique in these same recordings provoked a continuous P.O.S. in the beginning of the reaction. This continuous P.O.S. however, had already stopped before the pendular movements started. This shows once more that each of the two must have a different origin, since they occur at different moments of the reaction, have a different rhythm and a different form, and are accompanied by a different sensation.
In six other recordings we only found a beginning of pendular wave formation. However, the amplitude here was smaller (30° and less), the sinusoidal movement irregular and shorter (a few seconds). In spite of the mental activity during BCG, the test persons still felt sleepy at the end of the reaction. When asked for the reason of this drowsiness in spite of their appointed task, most of them answered that after some time the pushing of these buttons becomes an automatic movement. Yet we must not forget that in spite of this so called automatic movement, the answers were still correct or — in other words — the person was still paying

attention. Thus all these facts are in agreement with our E.E.G. findings viz. in the beginning the arousal effect is strongest (for the task must be learned) and the alpha-rhythm disturbed. After some time the "learning"-process is over, the buttons are being pushed automatically, the arousal effect decreases and the alpha-rhythm reappears on the E.E.G. In general, however, we may say that — with the exception of test person no. 1 — the pendular movements were absent. This proves the arousal effect of BCG. We must remember that during the BCG series the test persons had become more acquainted with the irrigations and that the time of recording was longer (6 minutes). These last two facts would rather facilitate the occurrence of pendular movements. In conclusion: we found a depressing effect of arousal on the pendular movements.

5. *Aberrant apices*

Once more we found no distinct rounding of the apices in the horizontal lead. In the vertical lead, however, rounding was frequently found, not only in the induced, but also in the spontaneous vertical nystagmus (Fig. A26). A nystagmus beat whose apex has disappeared was also repeatedly observed during the arousal series (Fig. A45). Both, the occurrence of rounding of the apices and decapitated nystagmusbeats were not influenced by the BCG.

6. *Blinking*

The occurrence of blinking in the vertical lead is not influenced by arousal (Fig. A46, A37).

Discussion

It has been known for some time that a simultaneous stimulation of other sensory organs together with the vestibular organ may strongly influence the induced nystagmus.
Grahe (1923) saw nystagmus disappear when rubbing a brush around the ear of the patient. He thought that this was caused by central suppression because the same effect occurred when he brushed the hand. The influence of sleep was shown by Mowrer (1934) who found that the duration of the nystagmic response was decreased by a preceding period of rest. This author put forward the following hypothesis: a preceding period of rest gives a decrease of the general nervous excitement and this phenomenon manifests itself by a diminished reactivity of the central mechanism which conditions the duration of the nystagmus.
In 1948 Hulk and Jongkees were the first to point out the importance of the attention paid by the test person himself and its influence on the nystagmus frequency. Thereafter much research has been done concerning the influence of all sorts of sensory stimuli on nystagmus (see literature review). As a result several techniques were found to stimulate the attention of test persons and animals. At the same time it was established that with a certain degree of alertness (provided it was kept at a constant level) the induced nystagmus was intensified and had a more regular

course. The best results were acquired with the help of acoustic stimuli. It is difficult however to give a physiological basis to the word "attention" — a notion which so far has only been used in psychology. Hubel, Henson, Rupert and Galambos (1959) demonstrated two kinds of units in the auditory cortex of a cat. One unit was stimulated by any sound, the other only when the cat was paying attention to the sound. Biemond (1961) pointed out the importance of the reticular formation which is directly or indirectly involved in a series of brain-stem reflexes and which moreover receives fibres from all sensory and sensible conduction systems which pass the brain-stem. Its significance lies, according to the author, in the fact that from the reticular formation a diffuse projection system is directed towards the entire cortex. This apparently has the object of bringing it in a condition of general "vigilance". And conversely, it is known that the cortex is projected on the thalamus and the brainstem by numerous efferent pathways which can facilitate the conduction of certain sensory stimuli and cause inhibition of others.
In what way can this arousal technique influence the induced nystagmus in our case? The discussion in the preceding chapter (vide pauses) showed that cortical influences (such as arousal) can affect the vestibulo-ocular reflex-arc at the following levels: i.e. (a) the labyrinths (via the efferents), (b) the oculomotor nuclei and (c) the complex of the vestibular nuclei and the reticular formation.
Both from our quantitative and from our qualitative data after caloric stimulation it follows that arousal leads to an increase of the velocity of the slow phase, an increase of the amplitude per beat, and to an increase of the frequency if compared to reactions following identical stimulation without arousal.
a. In recent years many authors have studied the vestibular efferents. Sala (1965) demonstrated that during electric square-wave stimulation at tetanic frequency of the vestibular efferent system both a reduction of the activity of the vestibular nerve and a hyperpolarisation of the vestibular direct current resting potentials surrounding the membranous labyrinth were recorded. Other functional studies (see page 45: Schmidt 1963, Bertrand and Veenhof 1964, Gleisner and Henriksson 1964, Goetmakers 1968) of the effect of the efferent nerve activation on the afferent flow impulses revealed that this control is inhibitory in action. In our case an increase in the velocity of the slow phase would implicate a decrease in the activity of the efferents. Following neuro-anatomical studies upon cats Petroff (1955), Rasmussen e.a. (1958), Gacek (1960) and Carpenter (1960) showed that the lateral vestibular nucleus of the same side, the medial, superior and parts of the decending vestibular nuclei and the fastigial nuclei on both sides send efferent fibres to the labyrinth (predominantly uncrossed). On the basis of histochemical and embryological studies upon guinea pig and rabbit Rossi e.a. (1963) were able to distinguish three bundles of efferent nerve fibres traceable from cells of different origin and location: (1) the direct ventral efferent vestibular bundle from the interposed vestibular nucleus, (2) the direct dorsal efferent vestibular bundle from the lateral vestibular nucleus and (3) the uncrossed efferent reticulo-vestibular bundle from the reticular formation near the median raphe. Primary efferents, however, to the labyrinths from cortical level were never found until now. So we can conclude that a direct cortical influence on the labyrinth by means of efferents is not very probable. If the efferents of the vestibular nerve play any part in the increase of the velocity of the slow phase, it can only be via a polysynaptic cortical pathway.
b. A direct cortical influence on the oculomotor nuclei is not probable either. This might explain the increase of the velocity of the slow phase but it does not explain the increase of the frequency, since the rhythm of the nystagmus is generated by the complex of vestibular nuclei and the reticular formation (see discussion of pauses).
c. We feel we must assume that the cortical influence mainly affects the reticular formation — an assumption that is in agreement with the ideas of most authors. The importance of the reticular formation is further demonstrated by the experiments of Duensing and Schaefer (1957) who discovered units in it that with an arousal stimulus showed an increased firing frequency simultaneously with an increase of the nystagmic response. According to Megighian e.a. (1961) an increase of the activity of the reticular formation causes a facilitation of the conduction of the vestibular stimulus. During a vestibular stimulus without arousal the vestibular sensory data — via the vestibular cortical projections area — are (according to Collins and Guedry 1962), a source of feed-back to the reticular formation which, in its turn, modulates vestibular nystagmus. According to the author it has been suggested that the experienced sensation depends upon cortico-fugal impulses (Jasper 1958), loss of subjective velocity may be an indicator of reduced cortico-fugal feed-back to the reticular system. Hence, as the subjective velocity declines during constant angular acceleration, the nystagmus would decline except for extraneous sources of arousal i.e. other sensory stimuli. Kornhuber (1966) noticed that the reticular formation is closely connected with the sleep-wake rhythm which also influences tht nystagmus. Goto, Takumasu and Cohen (1968) found that quick phases of nystagmus are initiated by a separate neural mechanism which is inoperative during drowsiness periods. From all these publications it appears that arousal facilitates the vestibular response. In recent years it has also been investigated which possible efferent routes exist for impulses from cortical (or subcortical) centres. Gernandt (1967) established that cortico-fugal fibres originating from the sensorimotor cortex, parietal, temporal, orbital and para-occapital areas project into reticular cells. Fibres going from these cells in the medullary and pontine regions are known to have intimate connections with the vestibular nuclei (Brodal e.a. 1962) and with the labyrinth via the efferents of the eighth nerve.
This facilitatory influence of the cortical activity via the complex of the vestibular nuclei and the reticular formation can explain the increase of the speed of the slow phase but does not explain the increase in frequency and amplitude.
The law of Mulder (1908) indicates that the threshold for rotatory nystagmus depends on the product of acceleration and stimulus duration which product has a constant value. We have no problems imagining

that there is a similar threshold with a constant value for the caloric stimulus. This threshold depends e.g. on the hour of the test, the age, psychologic factors of stress and fatigue (Clément, Jongkees and Oosterveld 1970). It can be influenced in three ways by arousal techniques.
In the first place the threshold value is lowered. In that case the threshold of the fast phase will be reached much sooner as a result of the increase of the velocity of the slow phase. The amplitude per beat will decrease. This, however, does not agree with our findings. Secondly the fast phase threshold remains the same but is reached sooner on account of an increase of the velocity of the slow phase, however, with an amplitude which remains constant. This does not agree with our findings either. If, however, the threshold rises slightly it will be reached sooner if only the speed of the slow phase increases also. This leads to an increase of the amplitude per beat and this does agree with our results.

We have already noticed that, in spite of the BCG, the dysrhythmia did not change appreciably and that the individual nystagmus patterns of the first series — typical for each test subject — remained unaltered. Pronounced dysrhythmia and arhythmia, however, did decrease strongly. This is in agreement with the findings of Collins and Guedry (1962), viz. that certain nystagmus patterns correspond with a certain degree of alertness. So far as the caloric induced nystagmus is concerned, we want to make slight changes in the division of the different levels of arousal proposed by Collins, Guedry, and Posner (1962).

1. Using an arousal technique we always found a nystagmus (the irregularity of this nystagmus in our opinion depends more on the individual than on the degree of mental activity).
2. Without arousal we noticed that a reduction of the quantitative parameters appeared. This, however, is so slight that it remains within the limits of the individual spread of these quantitative parameters and, therefore cannot be used as indicator for the degree of arousal.
3. At the same time the increase of groupformation type II is a sign of decreased alertness.
4. Complete suppression of the nystagmus indicates a very low level of mental activity.
5. The occurrence of pendular movements point to a test subject's falling asleep.

As we have already remarked all these conditions are accompanied by an alpha-rhythm and therefore we can add here to the conclusion of Collins, Crampton, and Posner (1961) and Collins and Guedry (1964) that under certain conditions the movements of the eye supply a better indication of the degree of alertness than E.E.G.-recordings. It would also be preferable to perform every routine caloric test on a subject who shows a simple mental activity for in that case one remains within the above mentioned stage number 1 or, at worst, one can fall back to stage number 2. When doing the routine caloric test without mental activity provocation one runs the risk of a quick fall-back to stage 4 and 5 and thus of getting entirely false results.

CHAPTER III

STATISTICAL ANALYSIS

Rank correlation tests of Spearman and Kendall

Maspétiol and Kéravec (1962) determined the product-moment-correlation between the different quantitative parameters and came to the conclusion that the maximum speed of the slow phase was the best parameter for measuring the vestibular reaction.

We used the rank correlation tests of Spearman and Kendall (table VIII, IX: see page 80-81) and not the product-moment-correlation because the random variables are not normally distributed.
Beside the correlation-coefficients also the corresponding tail probabilities p are calculated. A correlation now can only be regarded as significant if the corresponding twosided tailprobabilities p are not more than 0.05. Moreover, if the correlation-coefficient stated in absolute value is rather large, one can speak of a reasonable (positive or negative) significant correlation between both variables. A correlation-coefficient of 1 is only found in mechanical and physical phenomena. In physiology a coefficient of 0.7 points to a strong correlation between both variables; 0.4 to an average correlation and 0.2 to a weak correlation. The tail probability has in both cases been calculated by applying a normal approximation.
If the number of observations is rather small, e.g. 17 as in our case, theorically Spearman's test is preferable (de Jonge 1963).
We determined the correlation-coefficients between the following variables.

The first group of variables consists of:

1. Tr: the duration of the caloric reaction.
2. $V = V^\circ$: maximum speed of the slow phase.
3. A° : total amplitude during the culmination period.
4. F° : number of nystagmusbeats during the culmination period.
5. $Tn = Tr\text{-}t$: total duration of the active periods during the caloric reaction.
6. $Tn^\circ = 60\text{-}t^\circ$: total duration of the active periods during the culmination period.
7. $A^\circ/(60\text{-}t^\circ)$: mean amplitude per second during the culmination period.
8. $F^\circ/(60\text{-}t^\circ)$: mean number of beats per second during the culmination period or the effective frequency during this period.
9. A°/F° : mean amplitude per beat during the culmination period.

The second group of variables consists of:

1. a: number of pauses during the caloric reaction.
2. t: total duration of all pauses during the caloric reaction.

3. m: duration of the longest pause during the caloric reaction.
4. a°: number of pauses during the culmination period.
5. t°: total duration of all pauses during the culmination period.
6. m°: duration of the longest pause during the culmination period.

In table VIII all variables of the caloric reaction without arousal are tabulated and these with arousal in table IX. On top of each column we find three numbers. The first indicates the type of irrigation in the following way:

1 = left hot
2 = right hot
3 = left cold
4 = right cold

The second indicates the variables of the first above mentioned group and the third number concerns the variables of the second group, e.g. 323 is as follows: that we established in all 17 left cold irrigations (3) the correlation between the maximum speed of the slow phase (2) and the duration of the longest pause (3).
Our results were as follows:

1. Tr correlated with a, t, m.

Only a few correlation-coefficients indicate a positive correlation between a, t, and m paired with a tail probability <0.05. There is a slight increase in the number of significant tail probabilities during arousal. In general there is no definite correlation.

2. $V = V^\circ$ correlated with a, t, m.

The speed of the slow phase exhibits no correlation with a or t. We do find an average negative correlation value with m or in other words at a high speed of the slow phase no very long pauses will occur. With arousal we do not find this negative correlation value between the last two variables.

3. Tn correlated with a, t, m.

Obviously there exists an outspoken negative correlation value with t. In other words the duration of the active reaction will increase proportionally to the reduction of the total duration of the pauses.

4. $V = V^\circ$ correlated with a°, t°, m°.

In the absence of arousal we still find certain values which to a certain degree exhibit a negative correlation, especially between V° and m°. With arousal, however, we find a slight correlation value and a high tail probability or in other words unreliable results.

5. Tn° correlated with a°, t°, m°.

Obviously there exist unphysiologically high negative correlations for Tn° with t°. Also the negative correlations of Tn° with a° and m° are strong, both with and without arousal. From this it again clearly follows that the measuring during the culmination period was more accurate or in other words that the pauses are most obvious in those periods of the reaction where the speed of the slow phase is greatest.

6. $A^\circ/(60\text{-}t^\circ)$ correlated with a°, t°, m°.

With arousal we find an average negative correlation between the mean amplitude per second during the culmination period and the number of pauses during the same period. This applies to a smaller degree to t° and m°.

7. A° correlated with a°, t°, m°.

Here we find a strong negative correlation value with or without arousal, especially concerning t° and m°. It is normal that we find a reduced value with $A^\circ/(60\text{-}t^\circ)$. In fact in variable A° a certain influence of the pauses is already included.

8. F° correlated with a°, t°, m°.

A strong negative correlation exists between t° and m° with the number of pauses is not very clear. With arousal the correlation between both becomes less obvious.

9. $F^\circ/(60\text{-}t^\circ)$ correlated with a°, t°, m°.

Here is average negative correlation demonstrable between the effective frequency and the number of pauses, especially during arousal.

10. A°/F° correlated with a°, t°, m°.

The amplitude per nystagmusbeat exhibits an average negative correlation with a°, t°, and m° both with and without arousal.

Conclusion

1. The total duration of the reaction and the speed of the slow phase give little information about the occurrence of pauses.

2. The effective frequency — $F^\circ/(60\text{-}t^\circ)$ —, the effective amplitude — $A^\circ/(60\text{-}t^\circ)$ — and the amplitude per nystagmusbeat (A°/F°) exhibit an avarage negative correlation value regarding the number of pauses as well as the total duration of the pauses, and the longest duration of a pause.

3. The amplitude (A°) and the number of beats (F°) during the culmination period exhibit a strong correlation with a°, t° and m°.

Wilcoxon's test of symmetry

We wanted to know whether the quantitative changes which occurred after the caloric test with arousal were significant. For this reason we used Wilcoxon's test of symmetry (see table XII: page 81).
The hypothesis H_0 for Wilcoxon's test of symmetry (de Jonge 1963) is as follows: "both pairwise observed random variables have means of equal value". Depending on which way the test is to be used it is — in a sense — optimal against one of the following alternatives.

The tail probability has been calculated by applying a normal approximation.
Use was made of the same abreviations as above for the different variables.

Conclusions

1. During the test with arousal an increase of amplitude during the culmination period was demonstrated.

2. The number of pauses by the culmination period exhibits no increase or decrease by arousal.

3. The duration of the longest pause during the culmination period exhibits an obvious decrease by arousal.

The statistical procedures were provided by the statistical department of the Mathematical Centre in Amsterdam. The calculations were performed on the X8-Electrologica computer.

Table VIII. Explanation see Chapter III: without arousal

	111	211	311	411	112	212	312	412	113	213	313	413
S	+0,30	+0,50	+0,15	+0,22	—0,01	+0,05	—0,37	+0,08	—0,05	—0,02	—0,61	+0,02
p	0,24	0,04	0,56	0,40	0,96	0,85	0,15	0,76	0,84	0,93	0,01	0,93
	121	221	321	421	122	222	322	422	123	223	323	423
S	+0,18	+0,14	+0,07	+0,04	—0,26	—0,33	—0,55	—0,33	—0,55	—0,51	—0,57	—0,38
p	0,49	0,58	0,80	0,89	0,30	0,19	0,02	0,19	0,02	0,03	0,02	0,14
	151	251	351	451	152	252	352	452	153	253	353	453
S	—0,43	—0,49	—0,51	—0,70	—0,97	—0,99	—0,98	—0,98	—0,87	—0,87	—0,80	—0,67
p	0,09	0,04	0,04	10^{-2}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	10^{-5}	10^{-5}	10^{-3}	0,01
	124	224	324	424	125	225	325	425	126	226	326	426
S	+0,01	—0,17	—0,23	+0,02	—0,43	—0,48	—0,39	—0,36	—0,48	—0,61	—0,49	—0,36
p	0,97	0,50	0,38	0,95	0,08	0,05	0,12	0,16	0,05	0,01	0,04	0,16
	164	264	364	464	165	265	365	465	166	266	366	466
S	—0,60	—0,61	—0,86	—0,74	—1,00	—1,00	—1,00	—1,00	—0,90	—0,92	—0,82	—0,90
p	0,01	0,01	10^{-5}	10^{-3}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	10^{-4}	10^{-7}
	174	274	374	474	175	275	375	475	176	276	376	476
S	—0,45	—0,60	—0,38	—0,49	—0,38	—0,45	—0,28	—0,26	—0,32	—0,43	—0,01	—0,23
p	0,07	0,01	0,14	0,05	0,13	0,07	0,27	0,31	0,22	0,09	0,95	0,38
	134	234	334	434	135	235	335	435	136	236	336	436
S	—0,57	—0,53	—0,53	—0,48	—0,88	—0,84	—0,68	—0,83	—0,74	—0,81	—0,72	—0,73
p	0,02	0,03	0,03	0,05	10^{-5}	10^{-4}	10^{-2}	10^{-4}	10^{-3}	10^{-4}	10^{-2}	10^{-3}
	144	244	344	444	145	245	345	445	146	246	346	446
S	—0,19	—0,54	—0,35	—0,37	—0,76	—0,87	—0,66	—0,84	—0,76	—0,83	—0,83	—0,86
p	0,45	0,03	0,17	0,15	10^{-3}	10^{-5}	10^{-2}	10^{-4}	10^{-3}	10^{-4}	10^{-4}	10^{-4}
	184	284	384	484	185	285	385	485	186	286	386	486
S	—0,46	—0,58	—0,37	—0,50	—0,44	—0,45	—0,30	—0,31	—0,37	—0,41	—0,05	—0,27
p	0,06	0,01	0,14	0,04	0,08	0,07	0,24	0,23	0,15	0,10	0,85	0,29
	194	294	394	494	195	295	395	495	196	296	396	496
S	—0,58	—0,52	—0,46	—0,36	—0,68	—0,59	—0,44	—0,54	—0,49	—0,52	—0,37	—0,39
p	0,02	0,03	0,07	0,16	10^{-2}	0,01	0,08	0,03	0,04	0,03	0,14	0,12

Table IX. Explanation see Chapter III: with arousal

	111	211	311	411	112	212	312	412	113	213	313	413
S	+0,05	—0,00	+0,16	+0,52	+0,20	—0,10	+0,63	+0,32	+0,32	—0,15	+0,72	—0,18
p	0,86	1,00	0,54	0,03	0,45	0,71	0,01	0,22	0,21	0,57	10^{-3}	0,49
	121	221	321	421	122	222	322	422	123	223	323	423
S	+0,02	+0,09	+0,38	+0,05	+0,11	+0,25	+0,28	—0,10	+0,18	+0,38	+0,32	—0,27
p	0,93	0,74	0,13	0,84	0,69	0,33	0,27	0,70	0,49	0,13	0,22	0,30
	151	251	351	451	152	252	352	452	153	253	353	453
S	—0,86	—0,78	—0,88	—0,70	—0,94	—0,97	—0,92	—0,98	—0,63	—0,80	—0,62	—0,50
p	10^{-4}	10^{-3}	10^{-5}	10^{-2}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	0,01	10^{-3}	0,01	0,04
	124	224	324	424	125	225	325	425	126	226	326	426
S	—0,14	+0,09	+0,20	+0,04	—0,04	+0,26	+0,22	+0,01	—0,02	+0,17	+0,24	+0,03
p	0,60	0,72	0,43	0,88	0,87	0,31	0,40	0,96	0,93	0,50	0,35	0,90
	164	264	364	464	165	265	365	465	166	266	366	466
S	—0,95	—0,77	—0,94	—0,90	—1,00	—1,00	—1,00	—1,00	—0,85	—0,96	—0,91	—0,89
p	10^{-7}	10^{-3}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	10^{-7}	10^{-4}	10^{-7}	10^{-7}	10^{-5}
	174	274	374	474	175	275	375	475	176	276	376	476
S	—0,48	—0,65	—0,60	—0,46	—0,53	—0,47	—0,58	—0,40	—0,47	—0,46	—0,47	—0,32
p	0,05	10^{-2}	0,01	0,06	0,03	0,06	0,01	0,11	0,06	0,06	0,05	0,21
	134	234	334	434	135	235	335	435	136	236	336	436
S	—0,67	—0,68	—0,45	—0,65	—0,82	—0,92	—0,55	—0,78	—0,88	—0,92	—0,54	—0,75
p	10^{-2}	10^{-2}	0,07	0,01	10^{-4}	10^{-7}	0,02	10^{-3}	10^{-5}	10^{-7}	0,03	10^{-3}
	144	244	344	444	145	245	345	445	146	246	346	446
S	—0,55	—0,40	—0,19	—0,63	—0,72	—0,77	—0,41	—0,77	—0,75	—0,76	—0,40	—0,36
p	0,02	0,11	0,46	0,01	10^{-3}	10^{-3}	0,10	10^{3-}	10^{-3}	10^{-3}	0,11	0,01
	184	284	384	484	185	285	385	485	186	286	386	486
S	—0,51	—0,66	—0,57	—0,43	—0,56	—0,51	—0,53	—0,40	—0,46	—0,50	—0,40	—0,35
p	0,03	10^{-2}	0,02	0,09	0,02	0,04	0,03	0,11	0,06	0,04	0,11	0,16
	194	294	394	494	195	295	395	495	196	296	396	496
S	—0,56	—0,60	—0,36	—0,36	—0,59	—0,69	—0,25	—0,44	—0,61	—0,70	—0,24	—0,53
p	0,02	0,01	0,15	0,16	0,01	10^{-2}	0,33	0,08	0,01	10^{-2}	0,35	0,03

Table XII.

Wilcoxon's test of symmetry

H_0	H_1	left hot	right hot	left cold	right cold
$Tr_I - Tr_{II} = 0$	$Tr_I - Tr_{II} < 0$	$p_1 = 0{,}43$	$p_1 = 0{,}43$	$p_1 = 0{,}01$	$p_1 = 0{,}02$
$V^\circ_I - V^\circ_{II} = 0$	$V^\circ_I - V^\circ_{II} < 0$	$p_1 = 0{,}45$	$p_1 = 0{,}08$	$p_1 = 0{,}29$	$p_1 = 0{,}03$
$A^\circ_I - A^\circ_{II} = 0$	$A^\circ_I - A^\circ_{II} < 0$	$p_1 = 0{,}27$	$p_1 = 0{,}09$	$p_1 \; 10^{-2}$	$p_1 \; 10^{-2}$
$F^\circ_I - F^\circ_{II} = 0$	$F^\circ_I - F^\circ_{II} < 0$	$p_1 = 0{,}34$	$p_1 = 0{,}44$	$p_1 = 0{,}01$	$p_1 = 0{,}11$
$a_I - a_{II} = 0$	$a_I - a_{II} \neq 0$	$p_2 = 0{,}39$	$p_2 = 0{,}05$	$p_2 = 0{,}26$	$p_2 = 0{,}04$
$a^\circ_I - a^\circ_{II} = 0$	$a^\circ_I - a^\circ_{II} \neq 0$	$p_2 = 0{,}93$	$p_2 = 0{,}24$	$p_2 = 0{,}59$	$p_2 = 0{,}92$
$t_I - t_{II} = 0$	$t_I - t_{II} > 0$	$p_r = 0{,}31$	$p_r = 0{,}16$	$p_r = 0{,}41$	$p_r = 0{,}81$
$t^\circ_I - t^\circ_{II} = 0$	$t^\circ_I - t^\circ_{II} > 0$	$p_r = 0{,}13$	$p_r = 0{,}17$	$p_r = 0{,}02$	$p_r = 0{,}08$
$m_I - m_{II} = 0$	$m_I - m_{II} > 0$	$p_r = 0{,}43$	$p_r = 0{,}01$	$p_r = 0{,}41$	$p_r = 0{,}28$
$m^\circ_I - m^\circ_{II} = 0$	$m^\circ_I - m^\circ_{II} > 0$	$p_r = 0{,}03$	$p_r = 0{,}05$	$p_r = 0{,}02$	$p_r = 0{,}06$

SUMMARY

Since the introduction of electronystagmography we have at our disposal a method of obtaining a permanent recording of nystagmus. This recording not only enables us to obtain more quantitative results but also to make a detailed study of the nystagmus pattern. From the literature we see a definitely growing interest in the nystagmus pattern. Some authors attach much diagnostic value to it. There is, however, much difference of opinion, in the nomenclature as well as in the interpretation of specific types of nystagmuspatterns.

This thesis is a study of different types of nystagmus patterns occurring in the normal individual. After a strict selection we retained 17 normal test subjects. In these subjects a caloric nystagmus was induced with the Hamersma-technique (1957) and in this manner (17 x 4 =) 68 recordings were obtained. In each case the horizontal and vertical eyemovements were recorded simultaneously, together with the derived nystagmogram of the horizontal recording according to Henriksson and the electroencephalogram.

After one month a second caloric test was performed, but in this case the test was performed with the patient in a state of alertness, induced by the binary-choice generator. During the first test (without mental activation), some patients had a pathological directional preponderance or labyrinth predominance (Maas 1961). In all testsubjects a degree of dysrhythmia was found, furthermore the dysrhythmia found was characteristic of the recording of each individual test subject. By means of E.E.G. it was established that the first irrigation usually had the greatest arousal effect, especially in persons undergoing the test for the first time.

Pauses were demonstrated in all subjects. They were more frequent during the culmination period but their duration increased proportionately to the decrease in speed of the slow phase. During the pause various movement patterns of the eyes could occur. Vertical eyemovements were of great importance in the occurrence of pauses. In the E.E.G. the alpha-rhythm was not disturbed. After an extensive discussion on the origin of the fast phase we concluded that the reticular formation must play an important part in the origin of pauses.

The following were described as normally occurring patterns: arhythmia, intermittent fast phase, dysmetria, "petite écriture", salvos, different types of groupformation, and secondary-phase nystagmus. Other phenomena such as the occurrence of a vertical component, jerks, counterjerks, square-waves, P.O.S., sinus-waves, aberrant apices, and blinking were also discussed.

During the second test during mental activation we found no pathological labyrinthine predominance or directional preponderance in the same test subject. There was also an increase in the speed of the slow phase, the total amplitude, the frequency, and the mean amplitude per beat. After switching on the binary-choice generator during the first irrigation, we found in the E.E.G. a more protracted disturbance of the alpharhythm than during the following irrigations. Sooner or later the alpharhythm reappeared in spite of continuous mental activity. Dysrhythmia was not influenced by arousal; the number of pauses remained the same; there was, however, a significant decrease in the duration of the pauses, especially in the period following immediately after switching on the binary-choice generator. There was no obvious arhythmia during the second test series. The number of intermittent fast phases remained unchanged. There was a great increase in dysmetria; there was no obvious change in the occurrence of "petite écriture" and salvos. Groupformation (type II) was less outspoken. Secondary phase nystagmus increased 50 %. There was no change in the occurrence of the vertical nystagmus, jerks and counter-jerks increased, P.O.S. continued. Sinus-waves occurred less frequently. Aberrant apices and blinking were not influenced by arousal.

An attempt was made to explain "attention" on a neuro-physiological basis. On the basis of the literature we deduced that alertness goes together with "facilitation" of the stimulus-conduction together with a raised nystagmus-threshold. We also pointed out that certain eyemovements give more information on the degree of alertness than does the E.E.G. before falling asleep.

SAMENVATTING

Sinds de invoering van de electronystagmografie beschikt men over een methode van registratie en vastleggen van nystagmus. Deze registratie verschaft ons niet alleen de mogelijkheid om meer quantitatieve gegevens over de nystagmus te verkrijgen maar tevens om het nystagmuspatroon te bestuderen. Uit de literatuur blijkt duidelijk de groeiende interesse voor dit nystagmuspatroon. Sommige auteurs hechten er zelfs grote diagnostische waarde aan. Er bestaat echter veel onenigheid, zowel over de naamgeving als over de verklaring van bepaalde nystagmuspatronen.

Dit proefschrift heeft dan ook als doel verschillende nystagmuspatronen bij de normale mens te bestuderen. Na een strenge selectie bleven 17 proefpersonen over die aan de vooropgestelde eisen van normaal zijn voldeden. Bij deze proefpersonen werd een calorische nystagmus opgewekt volgens de Hamersma-techniek (1957) en aldus werden (17 x 4 =) 68 registraties verkregen. Telkens werden gelijktijdig de horizontale en vertikale oogbewegingen geregistreerd, samen met het afgeleide nystagmogram van de horizontale registratie volgens Henriksson en het electroencephalogram.

Na een maand werd voor de tweede maal een calorische test uitgevoerd, maar nu werd de proefpersoon tijdens de test een mentale belasting opgelegd (binaire keuze generator). Tijdens de eerste test zonder mentale belasting vertoonden enkele proefpersonen een pathologische richtingsvoorkeur of labyrinth-overheersing (Maas 1960). Bij alle proefpersonen werd in het nystagmogram enige dysrhythmie gevonden, en wel zodanig dat elke proefpersoon een voor hem karakteristieke registratie vertoonde. Door middel van het E.E.G. werd vastgesteld dat de eerste spoeling meestal het grootste wek-effect heeft, vooral bij proefpersonen die voor het eerst de test meemaken.

Bij elke proefpersoon werden pauzes aangetoond. Het aantal is frequenter tijdens de culminatieperiode, maar de duur neemt toe naarmate de snelheid van de langzame fase van de nystagmus afneemt. Tijdens de pauze zelf kan het oog nog verschillende bewegingspatronen vertonen. De vertikale oogbewegingen zijn van grote betekenis voor het voorkomen van de pauzes. Het alpha-rhythme in het E.E.G. wordt tijdens de pauzes niet verstoord. Na een uitgebreide discussie over de oorsprong van de snelle fase werd geconcludeerd dat de formatio reticularis een belangrijke rol moet spelen in het ontstaan van de pauzes.

Verder werden nog als normaal voorkomende patronen beschreven: arhythmie, intermittente snelle fase, dysmetrie, "petite écriture", salvo's. verschillende typen van groepsvorming en "secondary-phase nystagmus". Andere fenomenen zoals het voorkomen van een vertikale component, rukken, tegenrukken, blokvorming, P.O.S. , sinusgolven, afwijkende toppen en oogknipperen werden eveneens besproken.

Tijdens de tweede test met mentale belasting werd bij deze zelfde proefpersonen geen pathologische labyrinthoverheersing of richtings-voorkeur meer gevonden. Ook waren de snelheid van de langzame fase, de totale amplitude, de frequentie en de gemiddelde amplitude per slag toegenomen. Op het E.E.G. vonden wij bij het aanzetten van de binaire keuze-generator tijdens de eerste spoeling een langere verstoring van het alpha-rhythme dan tijdens de daaropvolgende spoelingen. Nochtans werd steeds na kortere of langere tijd, ondanks het goed uitvoeren van de mentale belasting, het alpha-rhythme opnieuw hersteld. Dysrhythmie werd door wekprikkels niet veranderd; het aantal pauzes bleef hetzelfde; wel werd een significante vermindering van de duur van de pauzes gevonden vooral in de periode direct na het aanzetten van de binaire keuze generator. Uitgesproken arhythmie kwam tijdens de tweede test-serie niet voor. Het aantal intermittente snelle fases bleef onveranderd. De dysmetrie nam sterk toe; het voorkomen van "petite écriture" en salvo's werd niet noemenswaardig veranderd. Groepsvorming (type II) was minder sterk vertegenwoordigd. "Secondary-phase nystagmus" nam toe met 50 %. De vertikale nystagmus was niet toegenomen, rukken en tegen-rukken wel. P.O.S. bleef bestaan. Sinusgolven kwamen veel minder voor. De afwijkende toppen en het oogknipperen werden door wekprikkels niet beïnvloed. Een poging werd gewaagd een neuro-fysiologische verklaring te geven voor het begrip "aandacht". Naar aanleiding van de gegevens uit de literatuur werd geconcludeerd dat wakkerheid gepaard gaat met "facilitatie" van de prikkel samen met een verhoging van de nystagmus-drempelwaarde. Ook werd er nog eens op gewezen dat vóór het inslapen bepaalde oogbewegingen meer inlichtingen geven over de graad van wakkerheid dan het E.E.G.

RÉSUMÉ

Depuis l'introduction de l'électronystagmographie on dispose d'une methode d'enregistrement qui nous permet d'obtenir un document permanent du nystagmus. Cet enregistrement nous donne non seulement la possibilité d'obtenir plus de données quantitatives concernant le nystagmus, mais nous permet de plus d'étudier le "pattern" nystagmique. Dans la littérature on s'intéresse de plus en plus à ce "pattern" nystagmique. Certains auteurs y attachent même une grande valeur diagnostique. Les opinions sont fort divisées, aussi bien pour la nomenclature que pour la signification de certains "patterns" nystagmiques.

Le but de cette thèse est d'étudier les différents "patterns" nystagmiques chez le sujet normal. 17 sujets normaux ont été scrupuleusement sélectionnés. Chez ces sujets on a produit un nystagmus calorique d'après la technique d'Hamersma (1957) et de cette façon on a obtenu (17 x 4 =) 68 inregistrements. Chaque fois on a enregistré en même temps les mouvements oculaires horizontaux et verticaux, aussi bien que le nystagmogramme dérivé de l'enregistrement horizontal selon Henriksson et l'électroencéphalogramme.

Un mois plus tard on a pratiqué pour une deuxième fois le test calorique, mais cette fois-ci le sujet devait accomplir un travail mental ("générateur binaire de choix"). Durant le premier test sans travail mental quelques sujets montraient une préponderance directionelle ou une prédominance labyrinthique pathologique (Maas 1960). Chez chaque sujet il existait un certain degré de dysrythmie, qui caractérisait son enregistrement. L'E.E.G. montrait que d'habitude la première irrigation provoquait l'effet d'éveil le plus marqué, surtout chez les sujets subissant le test pour la première fois.

Chaque enregistrement montrait des pauses. La fréquence de ces pauses était plus élevée pendant la période de culmination, mais la durée augmentait à mesure que la vitesse de la phase lente du nystagmus diminuait. Pendant les pauses l'oeil peut encore exécuter differents "patterns" de mouvement. L'apparition de pauses était fortement influencée par les mouvements oculaires verticaux. Le rhythme alpha n'est pas perturbé pendant les pauses. Après une longue discussion concernant l'origine de la phase rapide, ont peut conclure que la formation réticulée joue un rôle important dans l'origine des pauses.

Comme autres "patterns" on a encore décrit: arythmie, phase rapide intermittente, dysmétrie, petite écriture, salves, differents types de groupes et "secondary phase nystagmus". Autres phénomènes comme l'apparition d'une composante verticale, de saccades, de contre-saccades, de "square waves", d'E.O.P.'s, de mouvements pendulaires, de sommets nystagmiques aberrants et de clignements de l'oeil ont été discutés.

Pendant le deuxième test avec travail mental on n'a plus trouvé de prédominance labyrinthique ou prépondérance directionelle pathologique chez ces mêmes sujets. On a aussi constaté une augmentation de la vitesse de la phase lente, de l'amplitude totale, de la fréquence et de l'amplitude moyenne par secousse nystagmique. Pendant la première irrigation, après la mise en marche du générateur binaire de choix, le rythme alpha était perturbé plus longtemps que pendant les irrigations suivantes. Malgré l'exécution correcte du travail mental, le rythme alpha se réinstallait quand même toujours après un temps plus ou moins long. La dysrythmie n'était pas influencée par la technique d'éveil; le nombre de pauses restait égal; on constatait une diminution significative de la durée des pauses, surtout pendant la periode suivant la mise en marche du générateur binaire de choix. Pendant le deuxième test on n'observait pas d'arythmie prononcée. Le nombre de phases rapides intermittentes restait inchangé. La dysmétrie augmentait fortement, l'apparition de petite écriture et de salves n'était pratiquement pas changée. L'apparition de groupes type II était moins fréquente, le "secondary phase nystagmus" était augmenté de 50 %. La composante verticale n'était pas influencée, mais bien les saccades et contre-saccades. L'apparition de mouvements pendulaires était moins fréquente. Les sommets nystagmiques aberrants et les clignements de l'oeil n'étaient pas influencés par la technique d'éveil.

On a essayé de donner une explication neuro-physiologique pour la notion "attention". D'après les données de la littérature on a conclu que la vigilance entraine une "facilitation" de la conduction de la stimulation, accompagnée avec une augmentation du seuil nystagmique. On a constaté que, avant l'endormissement, les mouvements oculaires donnent plus d'information concernant l'état de vigilance que l'E.E.G.

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Appendix to the thesis:

DYSRHYTHMIA DURING THE CALORIC TEST

P. A. R. H. E. M. Clément

Abbreviations:

D	=	derived nystagmogram
H	=	horizontal lead
V	=	vertical lead
E	=	electroencephalogram
lc	=	left cold
rc	=	right cold
lh	=	left hot
no.	=	number of the subject
d	=	period of the recording represented
s	=	speed of the slow phase (measured as an amplitude above the base-line)
A	=	amplitude of the fast phase (measured as an amplitude below the base-line)
+a	=	with arousal
—a	=	without arousal
③	=	the number corresponds with the number of the phenomena represented in Fig. 4
↑	=	start of the choice task (BCG)

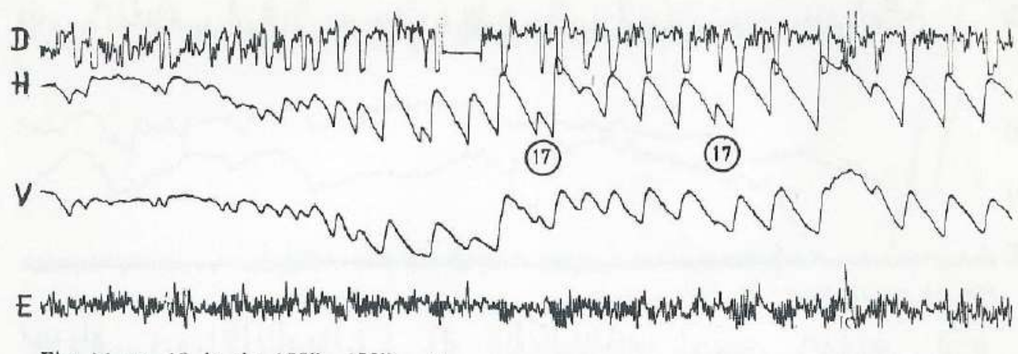
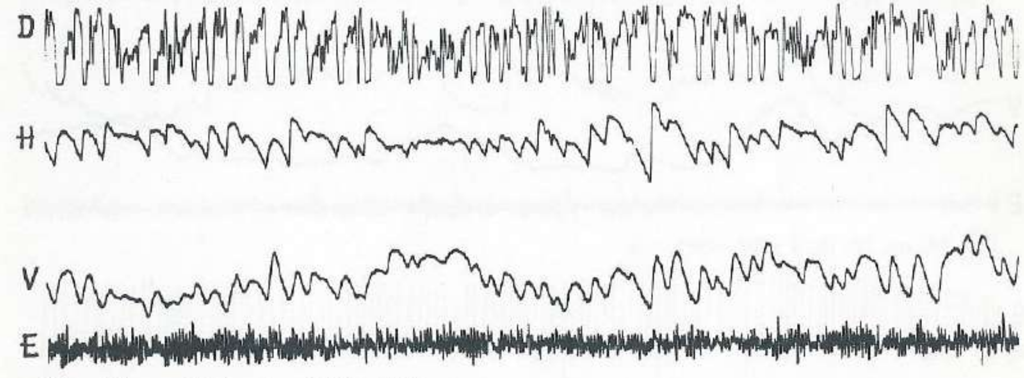


Fig. A1. no. 12, lc, d : 120"—150", —a



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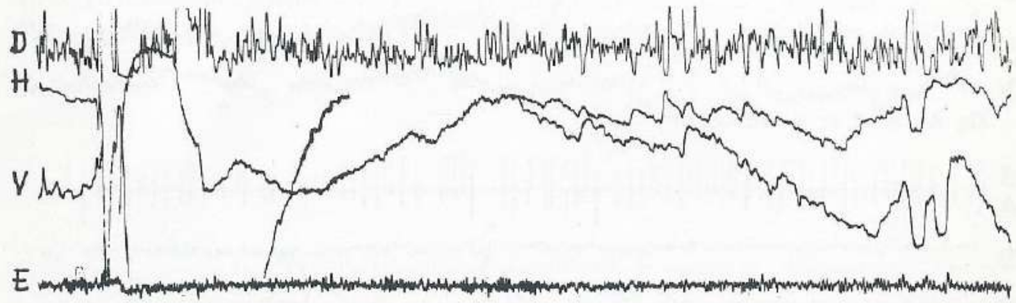

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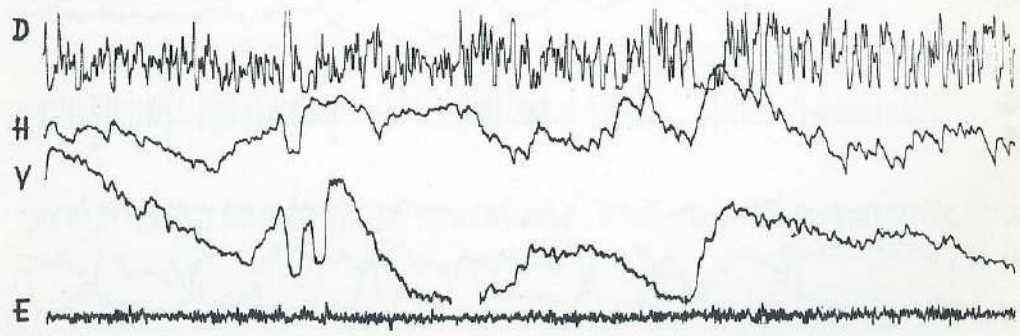

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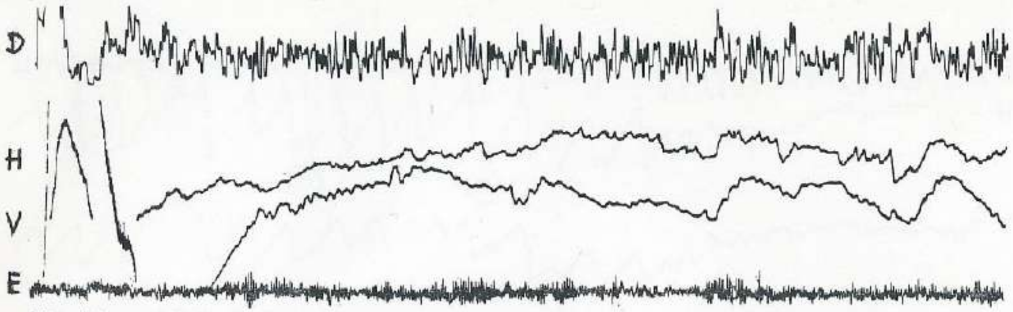

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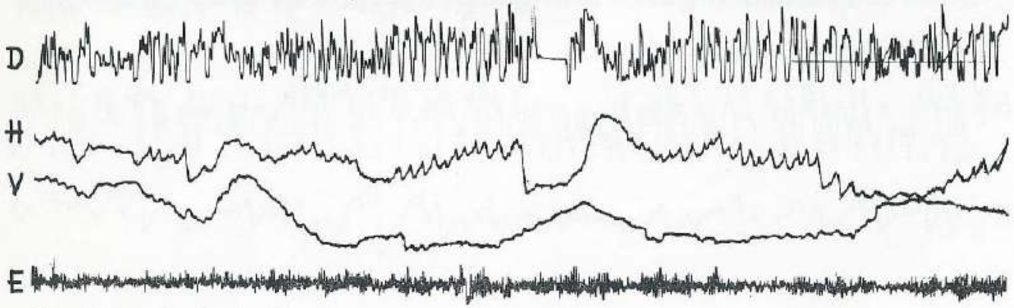

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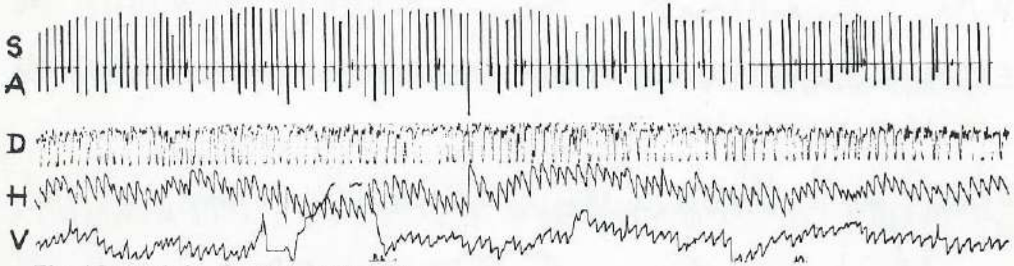

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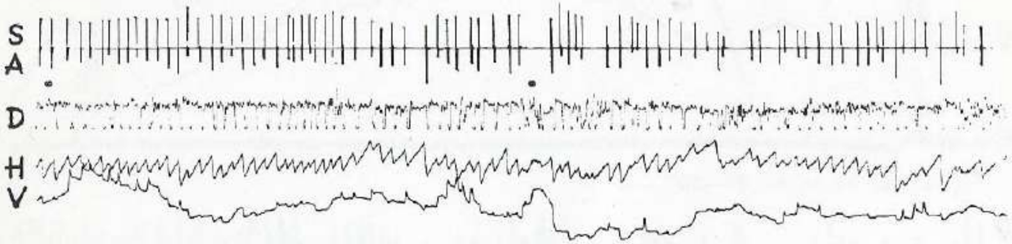

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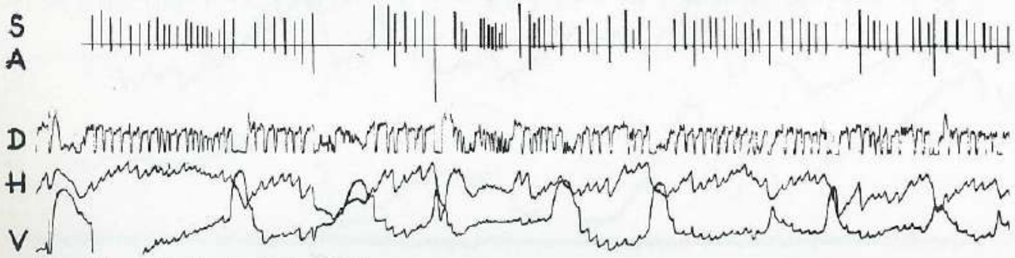

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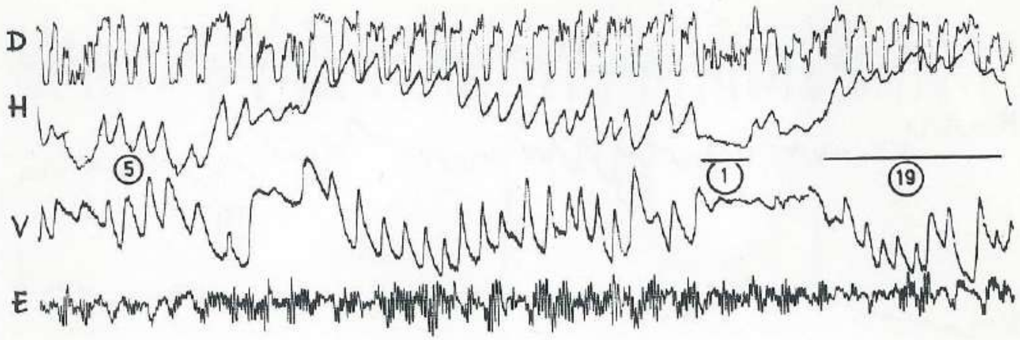
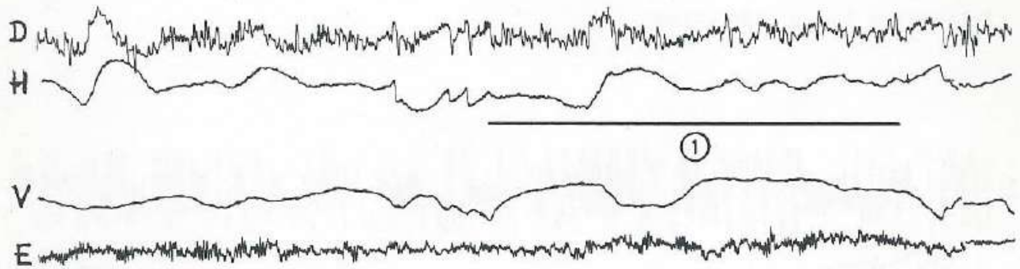


Fig. A8. no. 10, rc, d : 70"—100", —a



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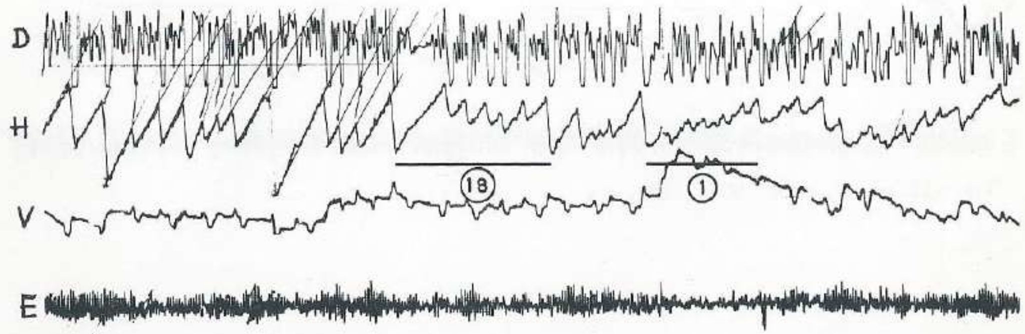
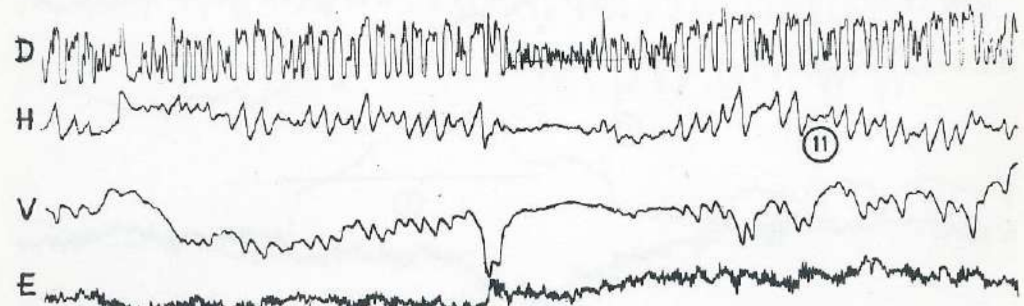


Fig. A10. no. 16, rc, d : 110"—130", —a



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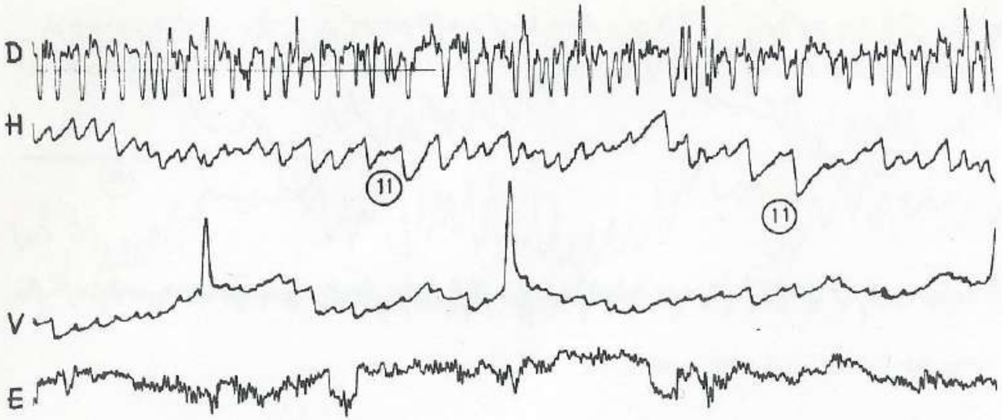

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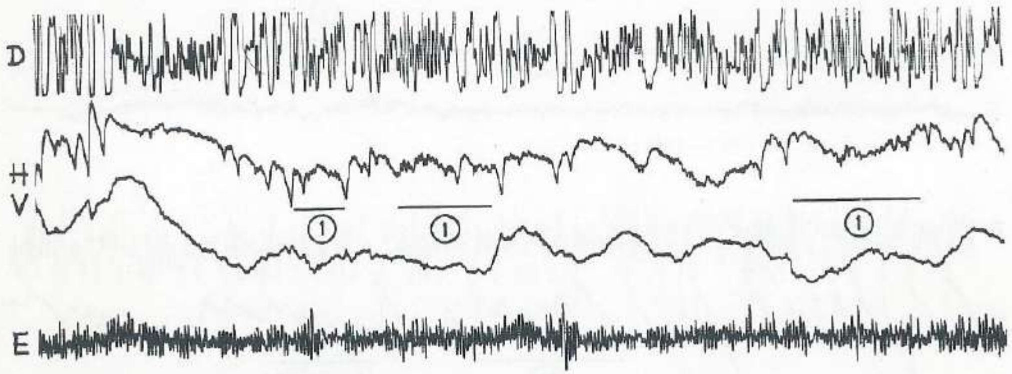

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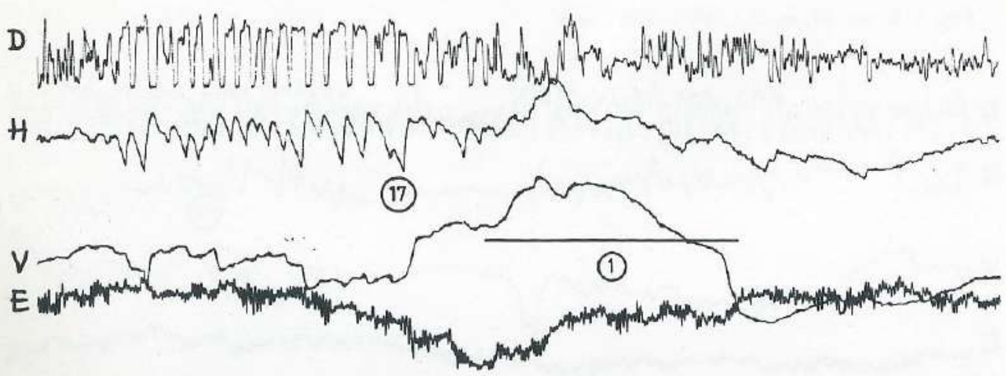

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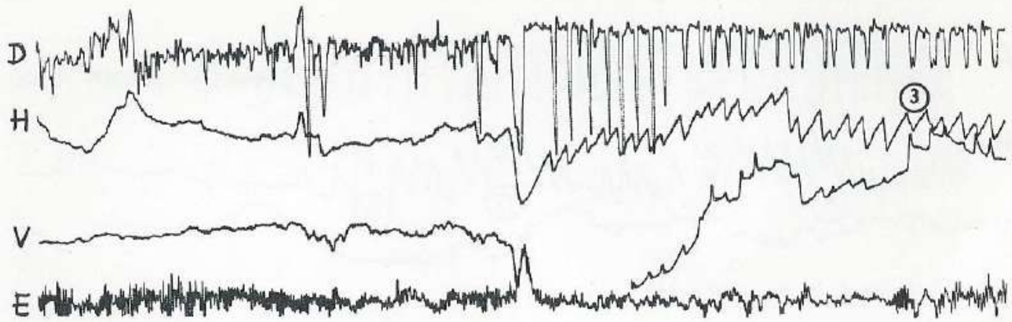

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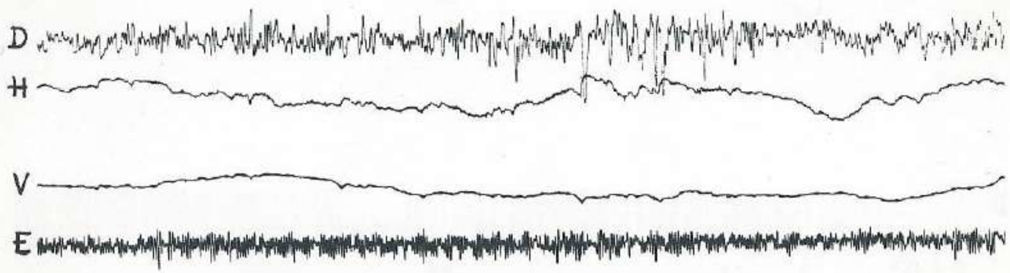
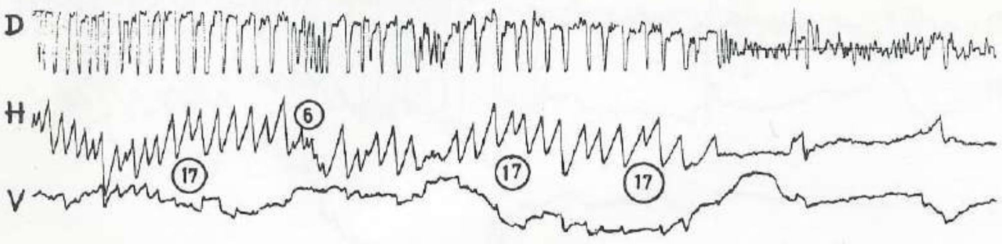


Fig. A16. no. 9, rh, d : 60"—90", —a

Fig. A17. no. 3, rc, d : 110"—150", —a



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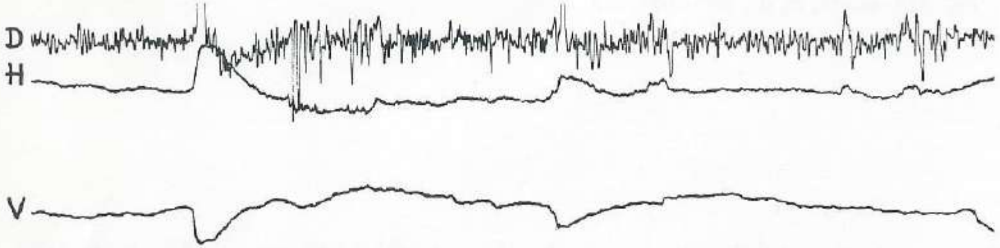

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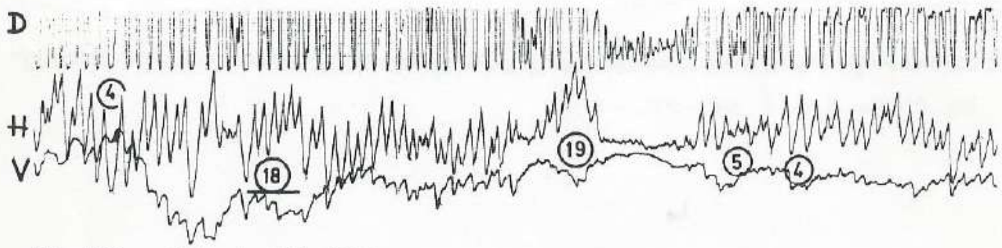

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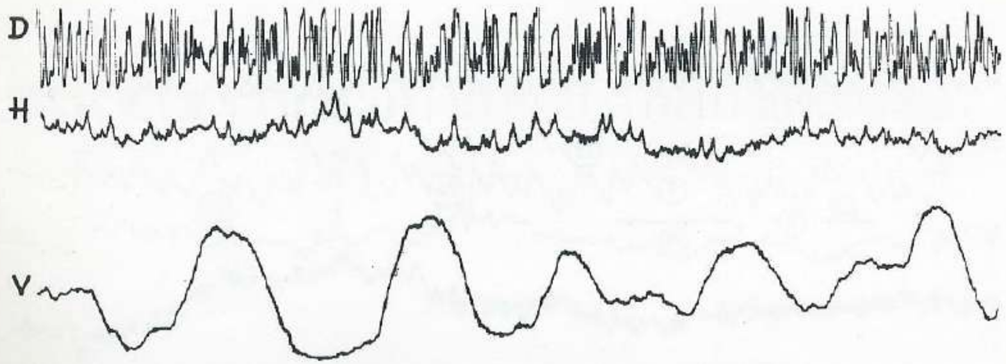

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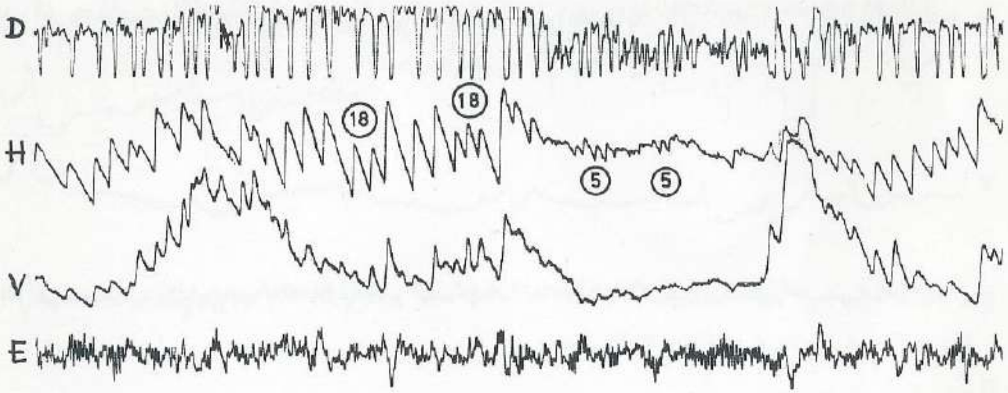

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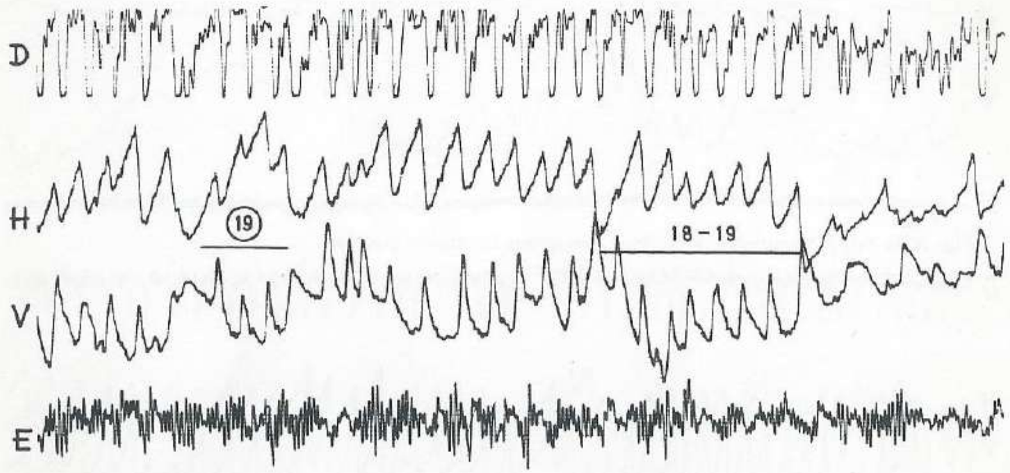

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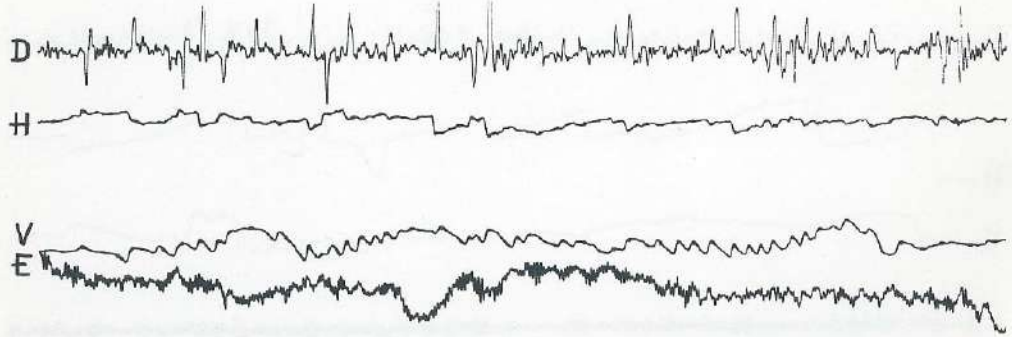

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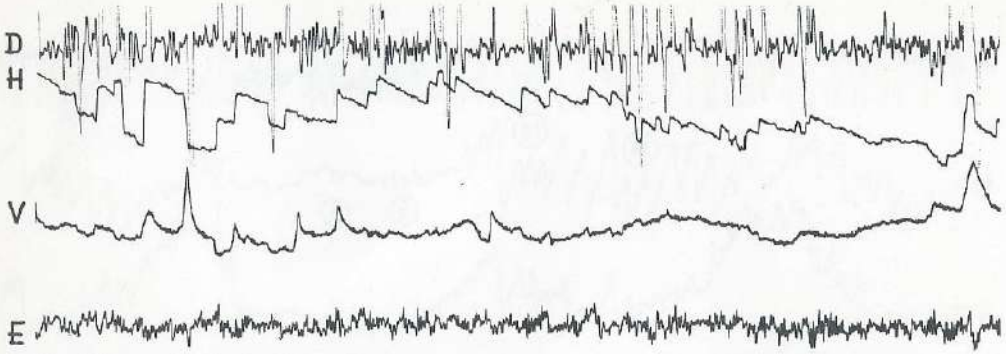

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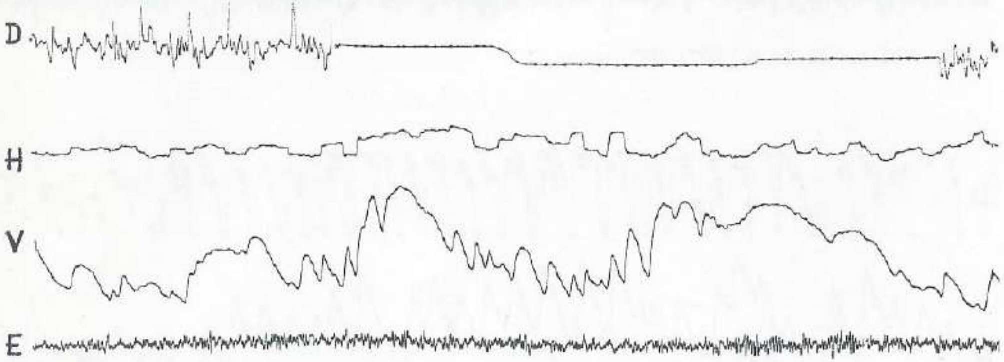
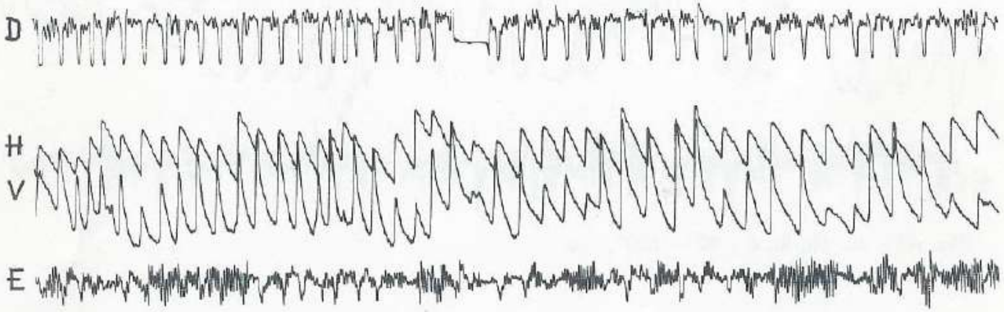


Fig. A26. no. 7, spontaneous vertical nystagmus in supine position



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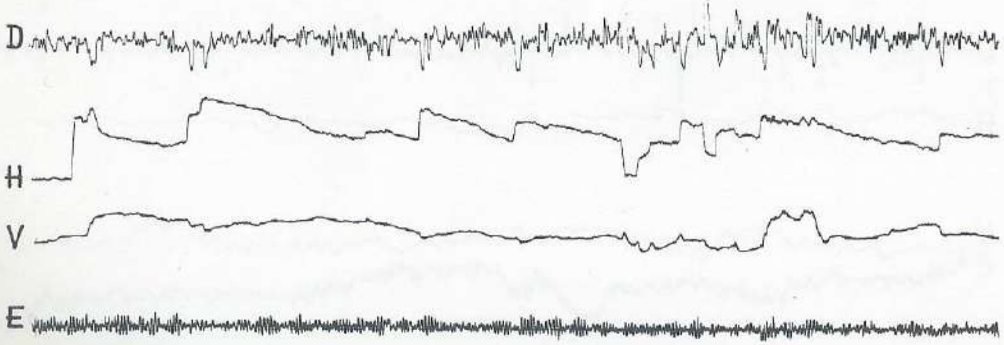
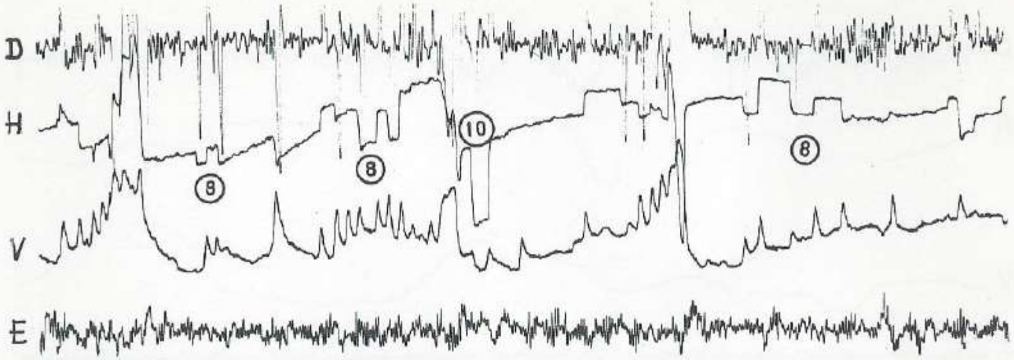


Fig. A28. no. 3, spontaneous jerks and square-waves in supine position



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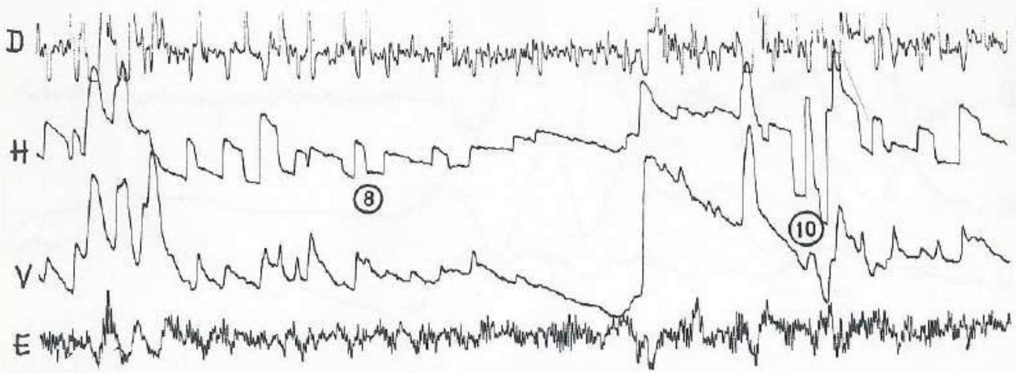

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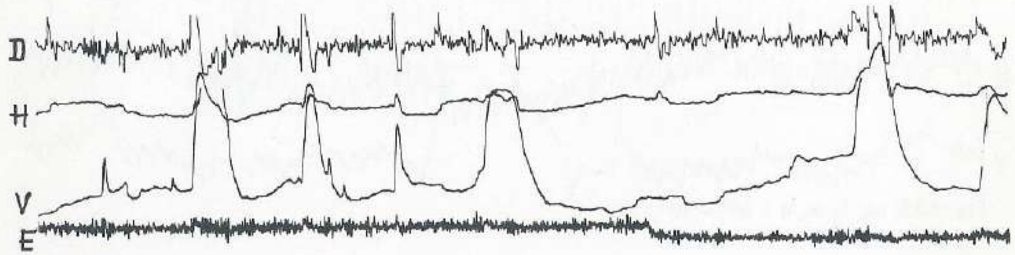

Fig. A31. no. 1, spontaneous P.O.S. in supine position

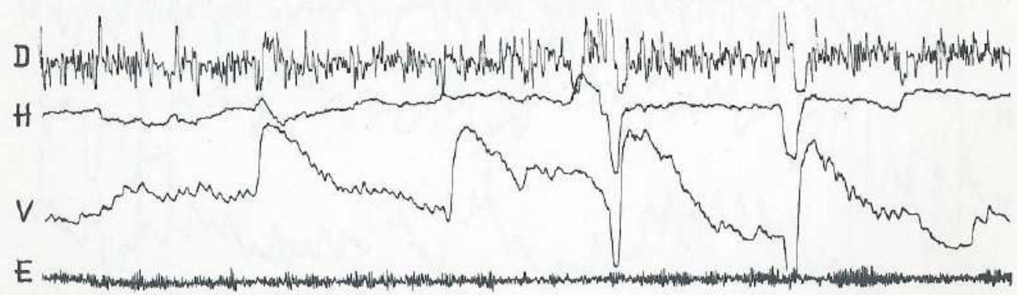

Fig. A32. no. 16, spontaneous P.O.S. in supine position

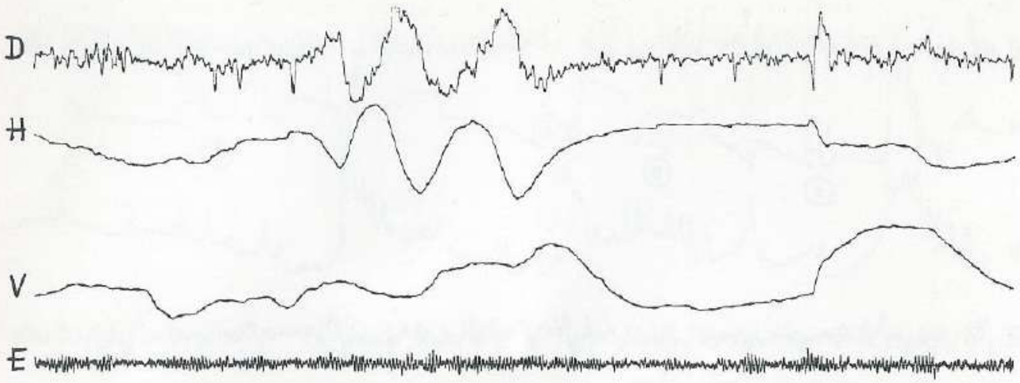

Fig. A33. no. 8, spontaneous pendular movements in supine position

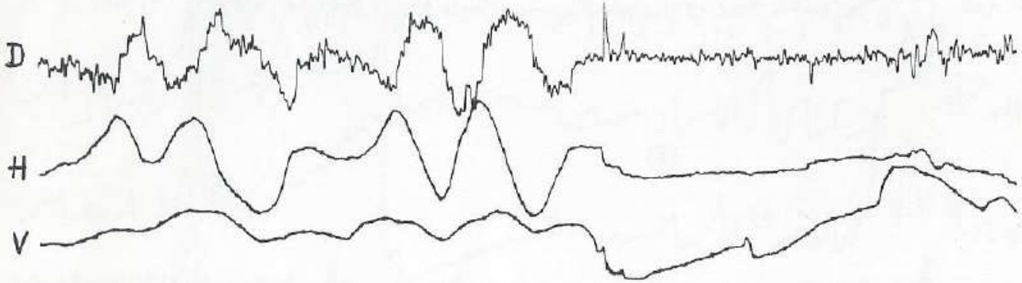

Fig. A34. no. 1, lc, d : 190"—210", —a

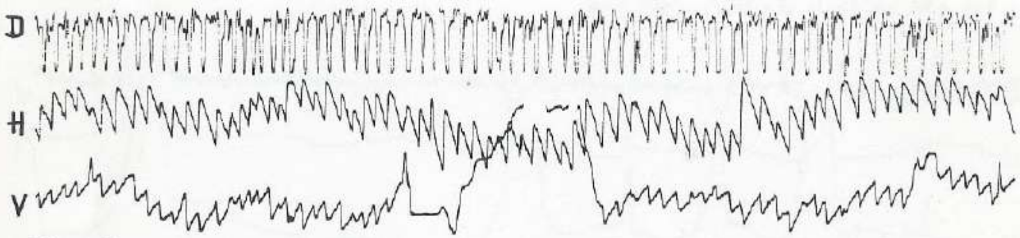

Fig. A35. no. 3, lc, d : 60"—90", —a

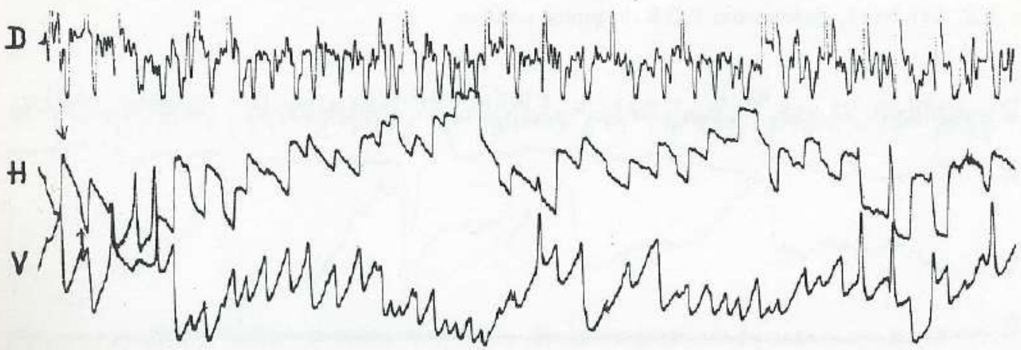

Fig. A36. no. 3, rh, d : 130"—150", —a

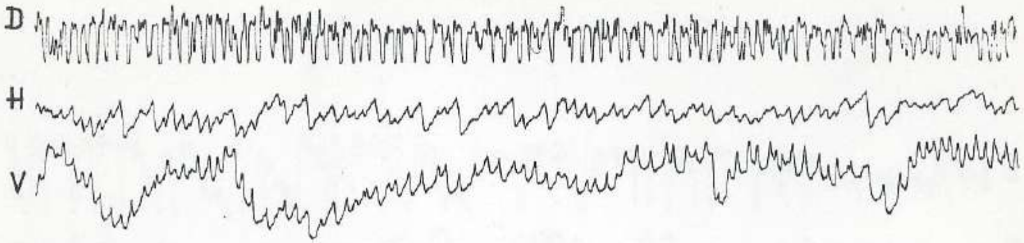

Fig. A37. no. 11, lh, d : 140"—160", —a

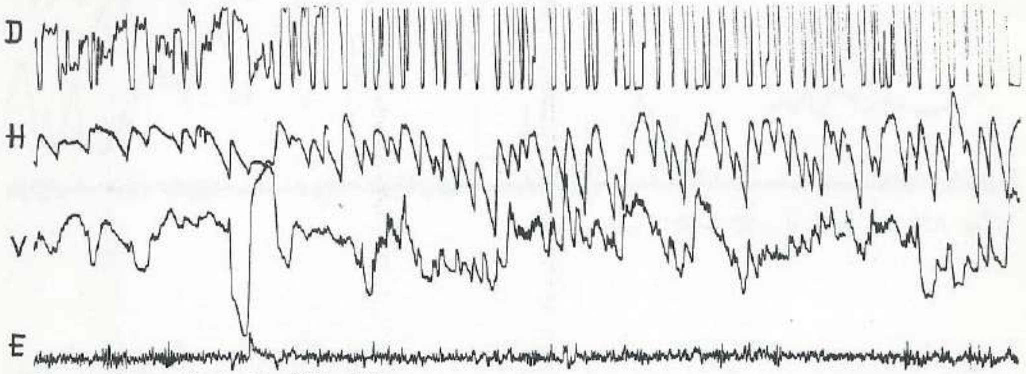

Fig. A 38. no. 9, rh, d : 30"—60", +a

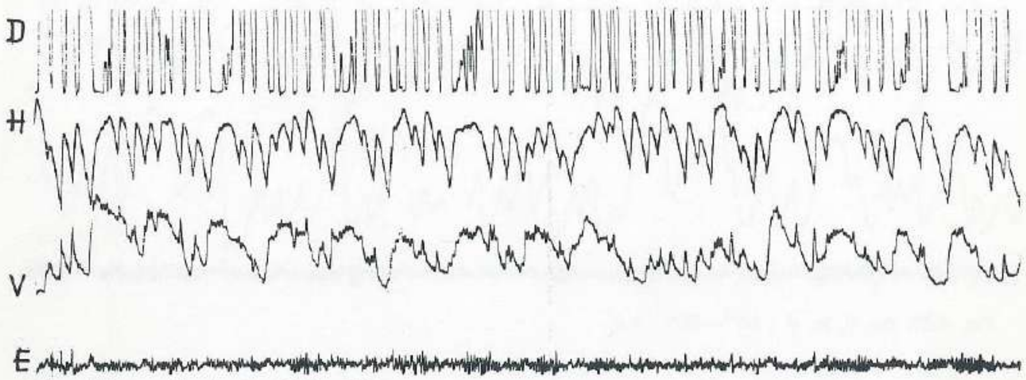

Fig. A38. no. 9, rh, d : 60"—90", +a

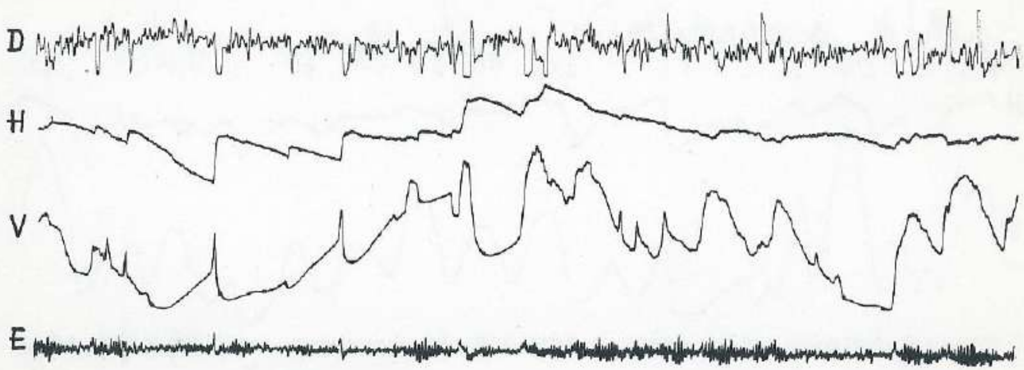

Fig. A38. no. 9, rh, d : 150"—180", +a

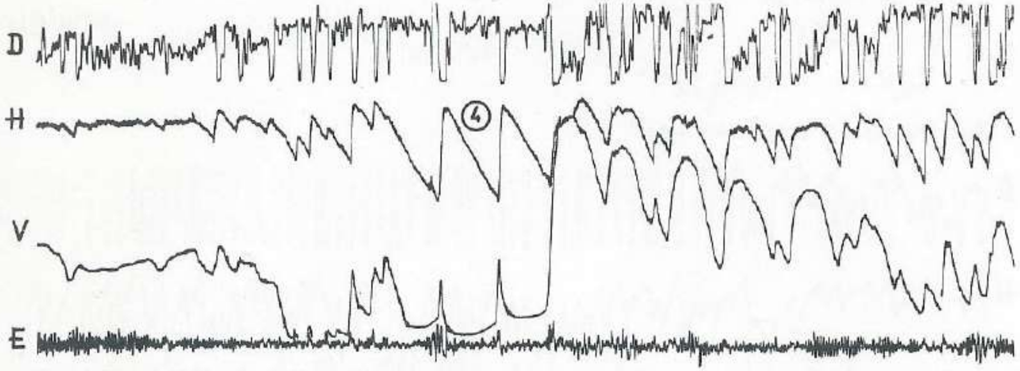

Fig. A39. no. 9, lc, d : 30"—60", +a

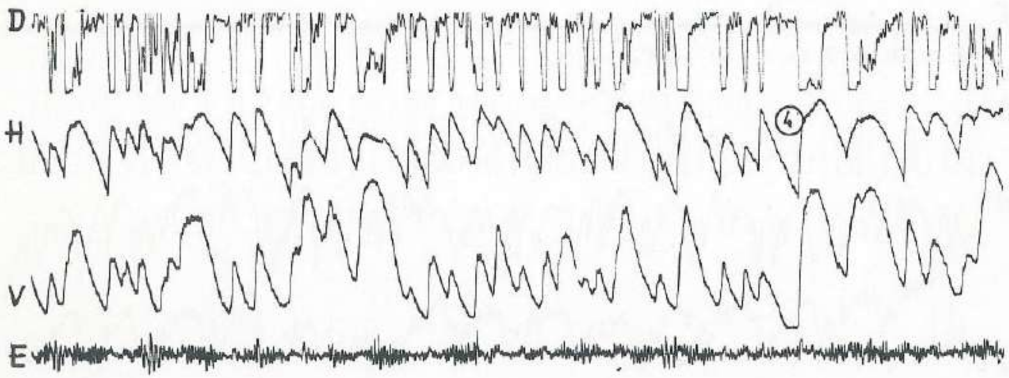

Fig. A39. no. 9, lc, d : 60"—90", +a

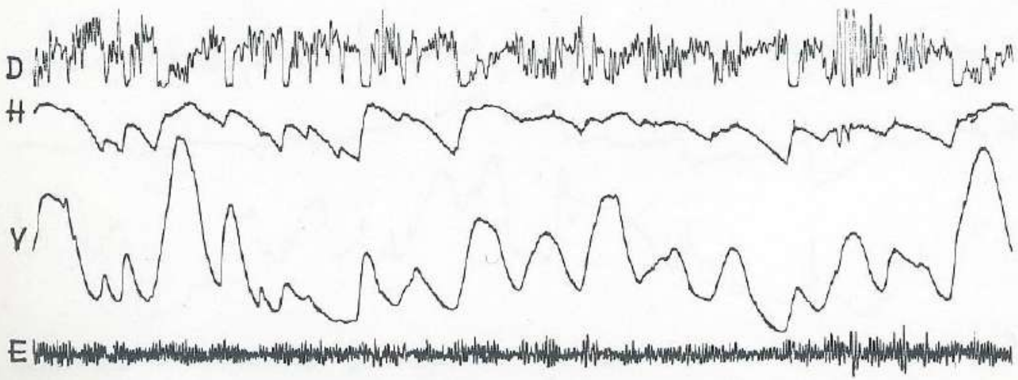

Fig. A39. no. 9, lc, d : 150"—180", +a

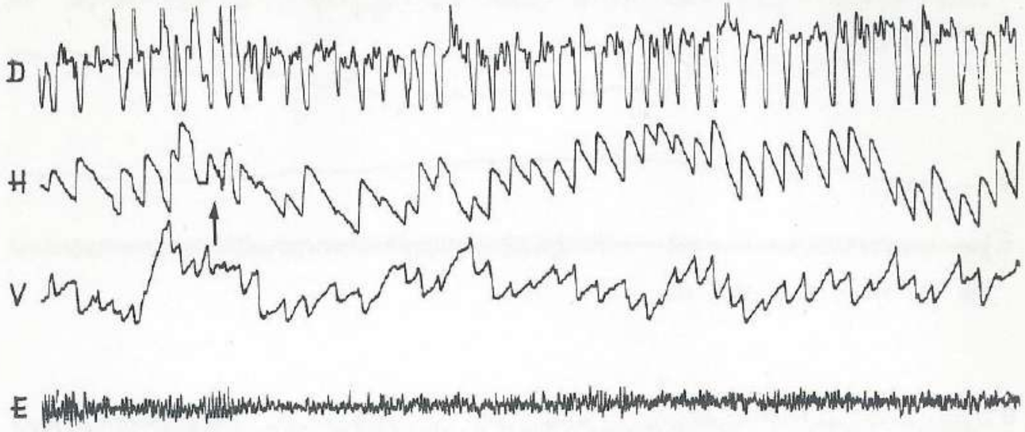

Fig. A40. no. 3, lc, d : 30"—60", +a

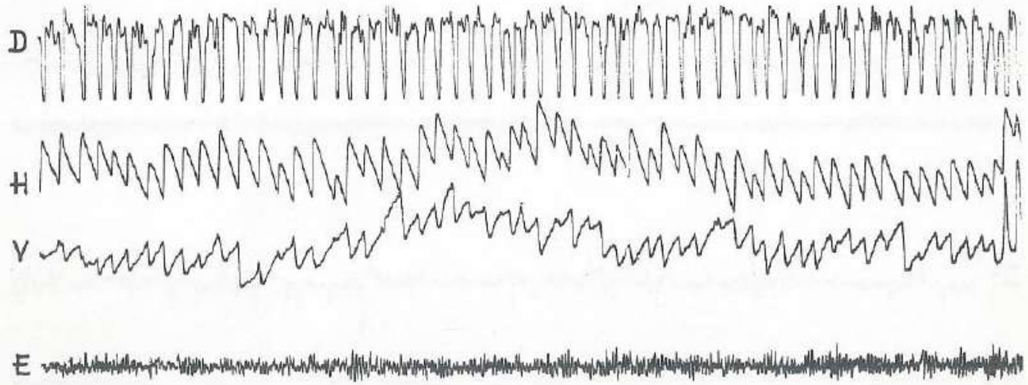

Fig. A40. no. 3, lc, d : 60"—90", +a

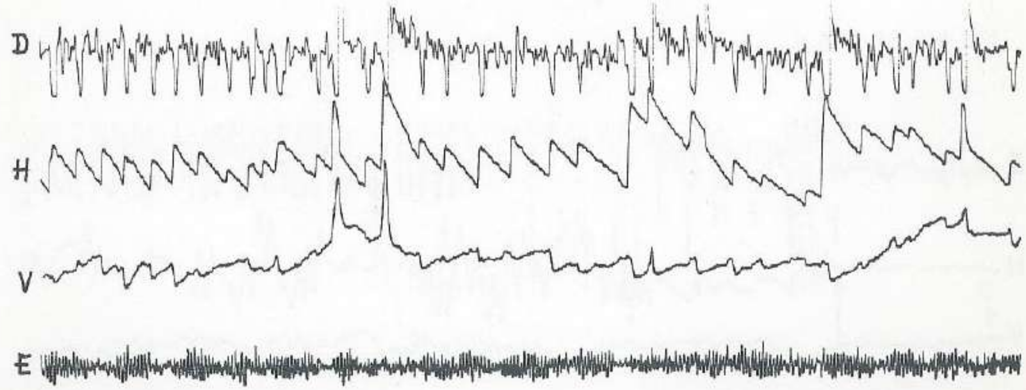

Fig. A40. no. 3, lc, d : 150"—180", +a

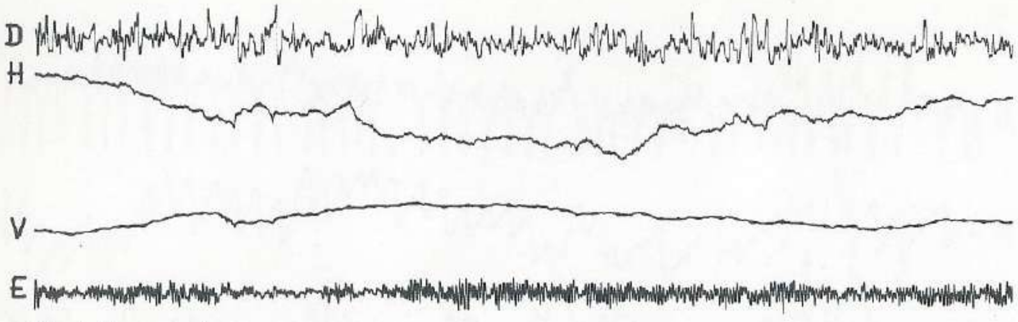

Fig. A41. no. 9, rh, d : 30"—60", —a

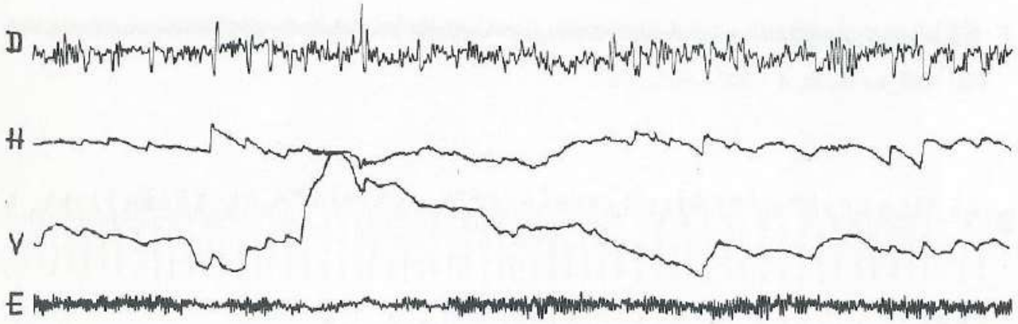

Fig. A42. no. 9, lc, d : 30"—60", —a

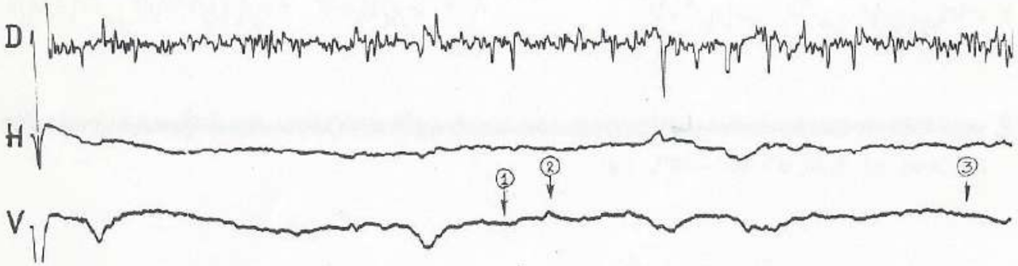

Fig. A43. no. 8, lh, d : 0"—30", +a

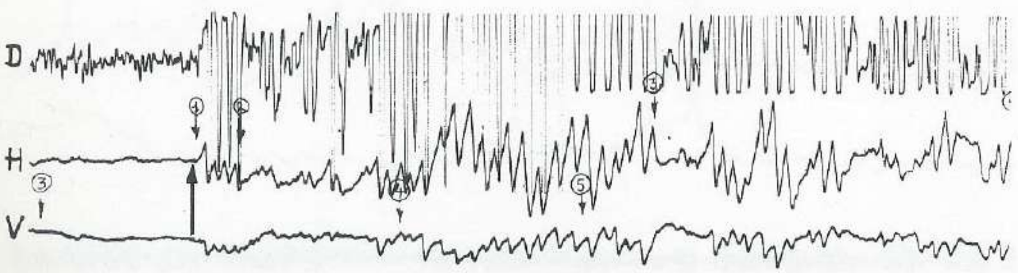

Fig. A43. no. 8, lh, d : 30"—60", +a

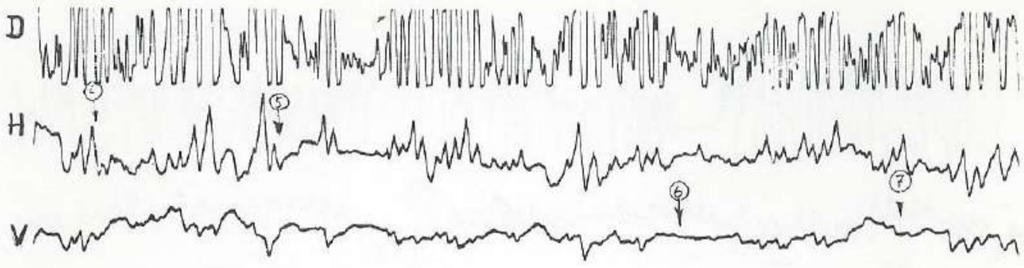

Fig. A43. no. 8, lh, d : 60"—90", +a

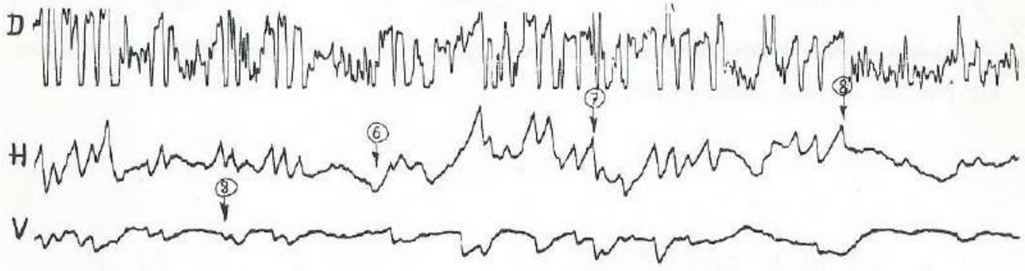

Fig. A43. no. 8, lh, d : 90"—120", +a

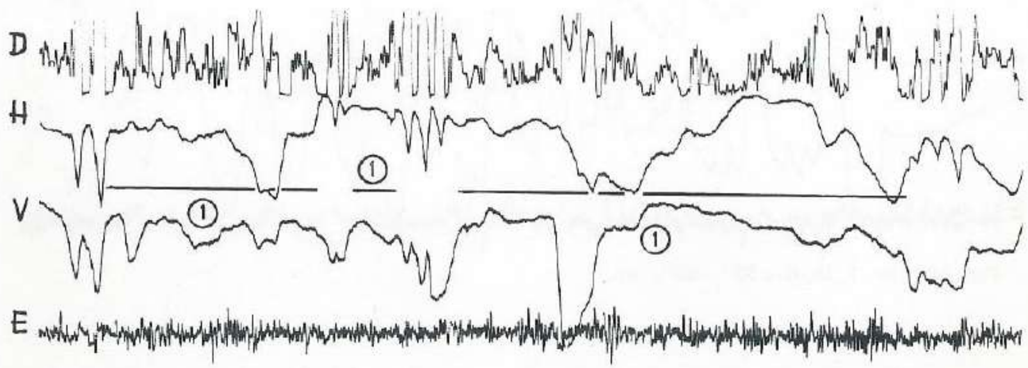

Fig. A44. no. 1, rh, d : 60"—90", +a

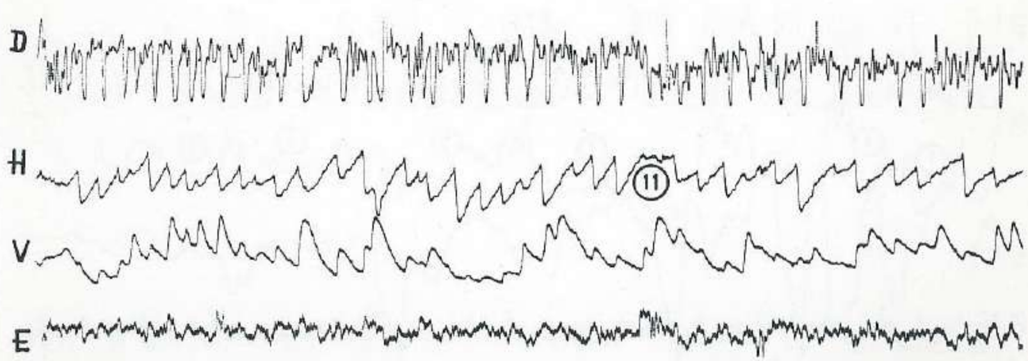

Fig. A45. no. 6, rc, d : 100"—130", +a

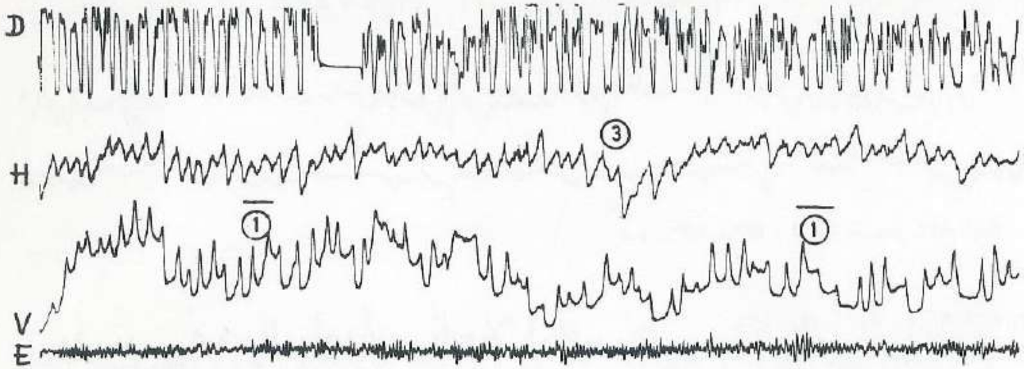

Fig. A46. no. 11, lh, d : 120"—150", +a

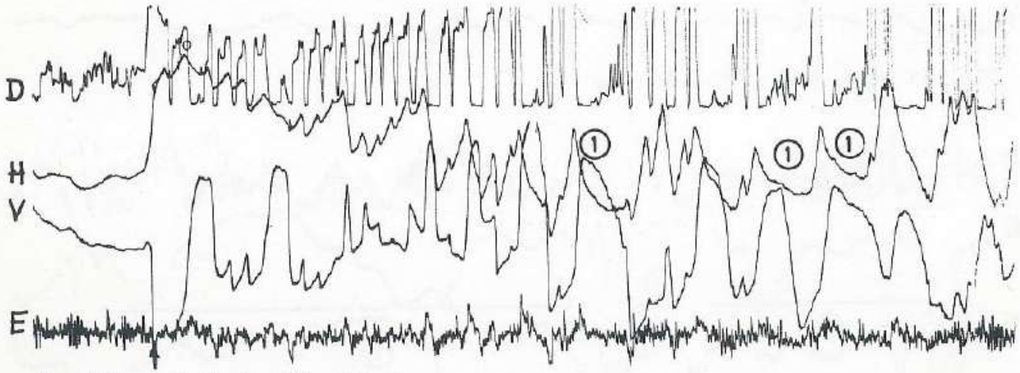

Fig. A47. no. 1, lh, d : 30"—60", +a

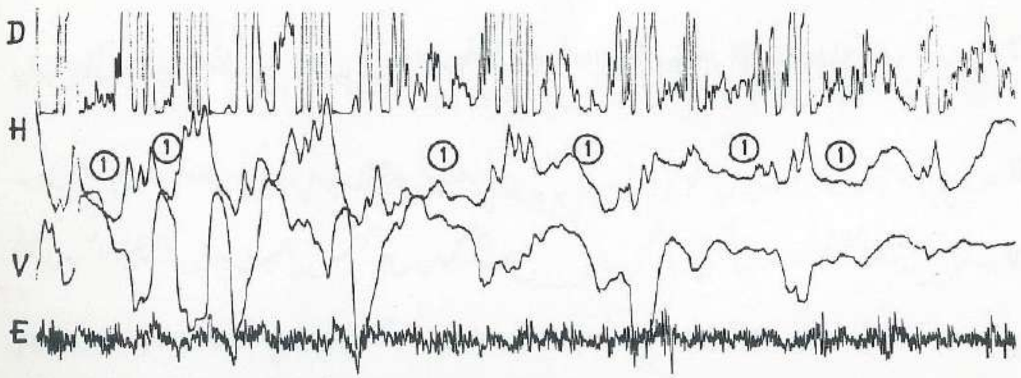

Fig. A47. no. 1, lh d : 60"—90" +a

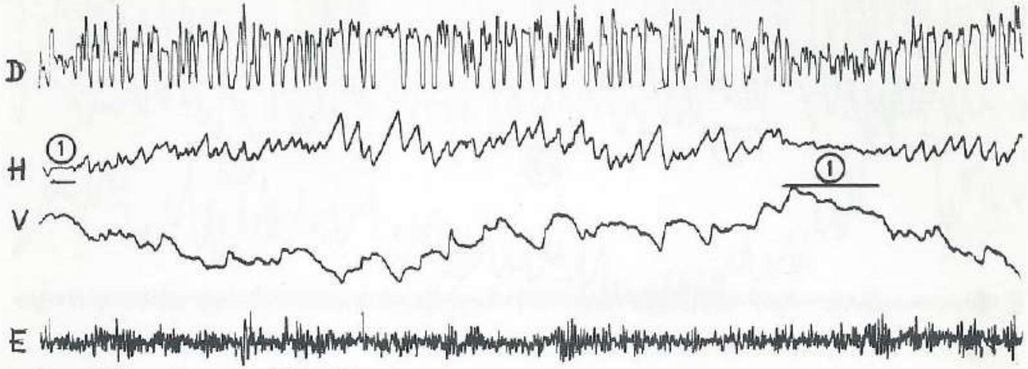

Fig. A48 no.. 2, rc, d : 60"—90", +a

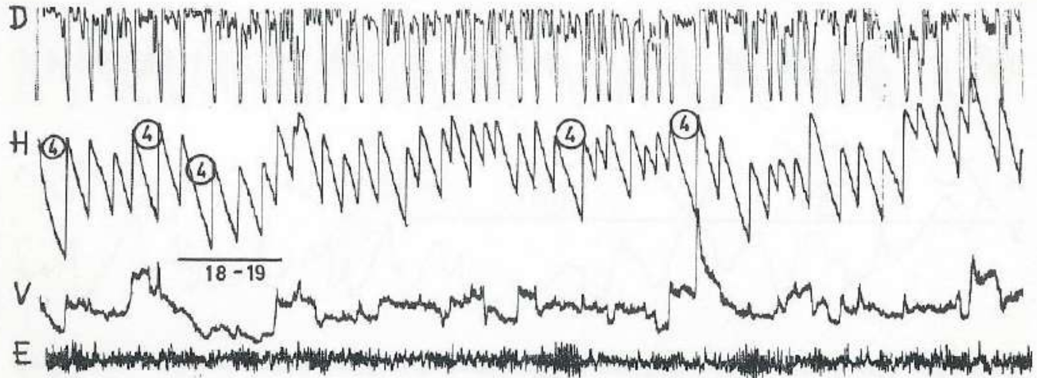

Fig. A49. no. 13, rh, d : 55"—85",+a

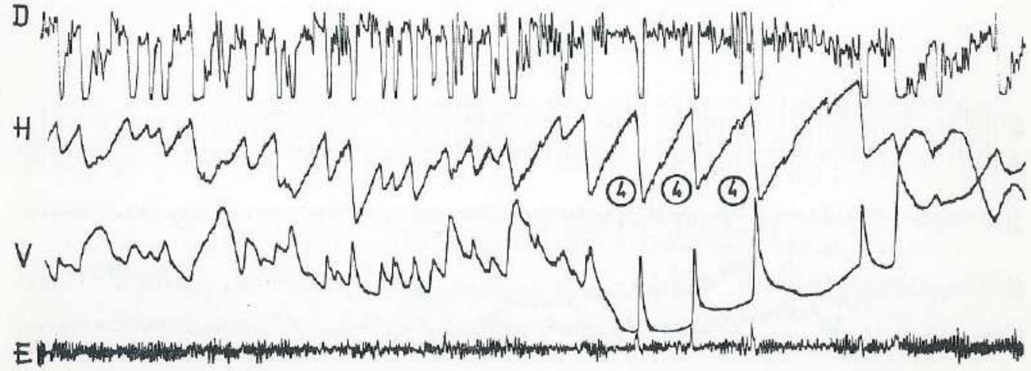

Fig. A50. no. 9, rc, d : 120"—150", +a

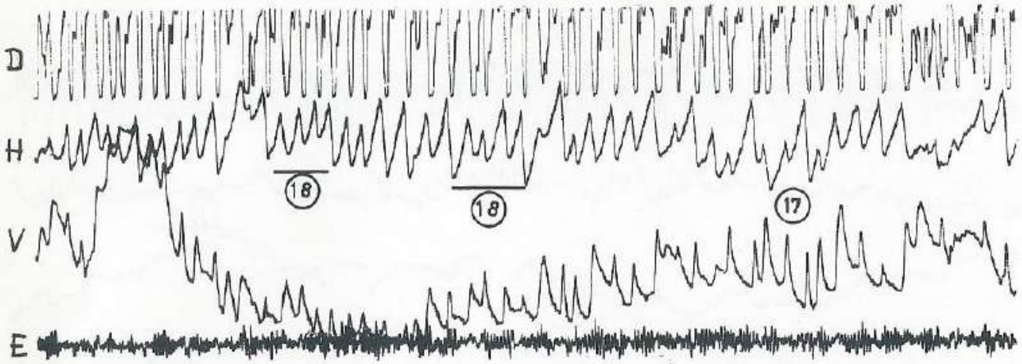

Fig. A51. no. 10, lh, d : 70"—100", +a

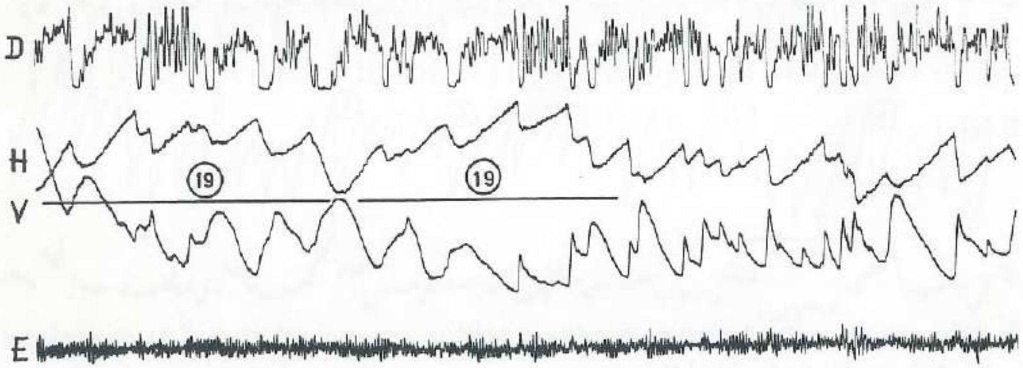

Fig. A52. no. 9, rc, d : 150"—180", +a

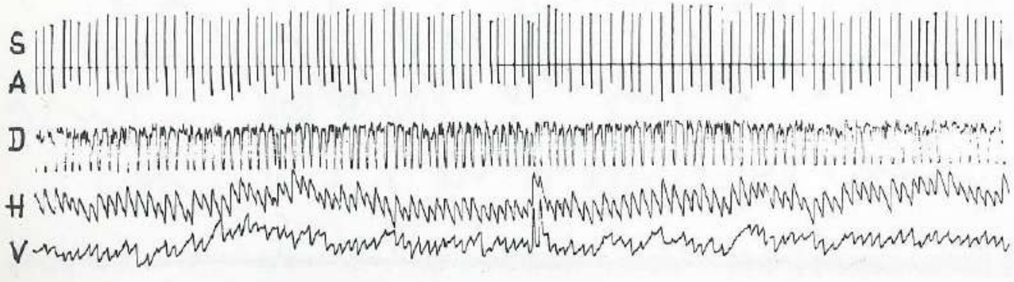

Fig. A53. no. 3, lc, d : 60"—120", +a

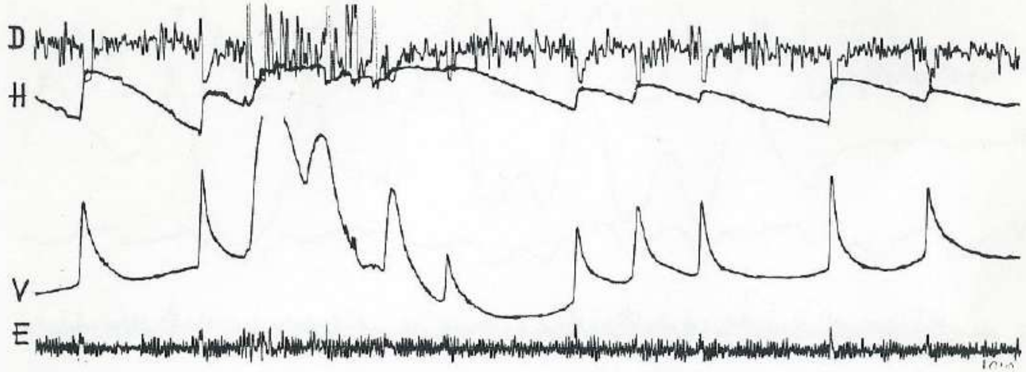

Fig. A54. no. 9, lc, d : 210"—240", +a

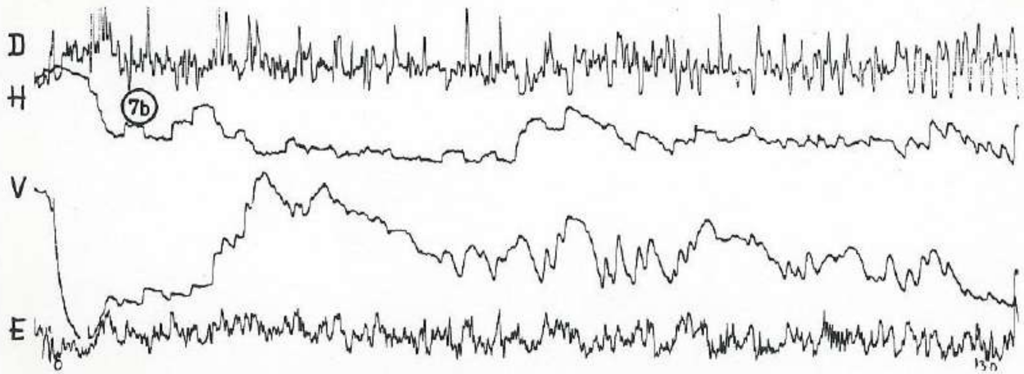

Fig. A55. no. 7, rh d : 0"—30" +a

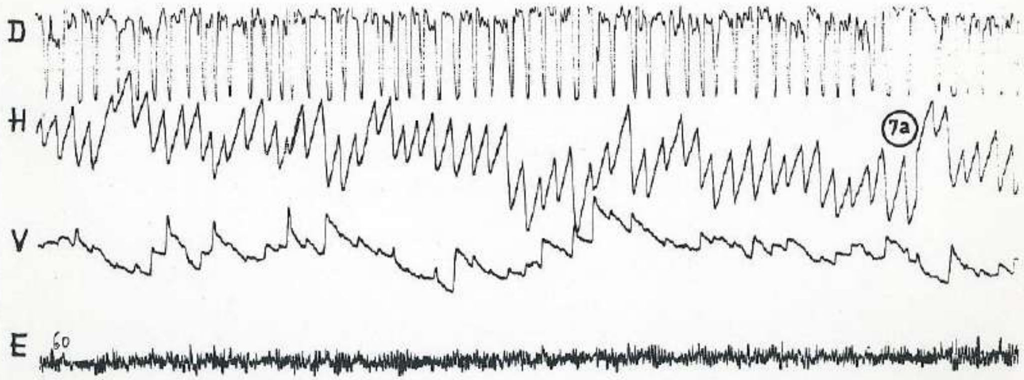

Fig. A56. no. 3, rc, 60"—90", +a

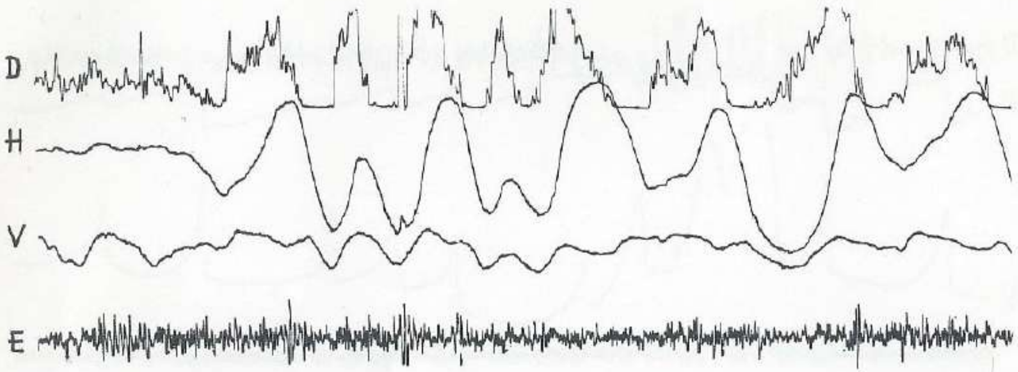

Fig. A57. no. 1, lh, d : 210"—240", +a

STELLINGEN

I

Het bepalen van phenylketonurie bij pasgeborenen zou moeten verplicht worden gesteld.

Hospitals **40**, 1966.

II

Bij onverklaarde buikpijnklachten na de maaltijden dient een aortografie uitgevoerd te worden.

Arch. Surg **88**, 1021-1044, 1964.

III

Na iedere meningitis cerebro-spinalis, en zeker bij kinderen, moet een uitgebreid audio-vestibulair onderzoek volgen.

IV

Bij het uitvoeren van een gastroscopie door middel van een fibroscoop is het gewenst dat een ervaren oesophagoscopist aanwezig is.

V

Frekwente bepalingen van het serum-L.D.H. in het beloop van een acuut myocard infarct blijven vooralsnog zinvol, aangezien er een negatieve correlatie bestaat tussen de hoogte van deze waarde en de overlevingskans op lange termijn.

Acta med. scand. **182**, 597, 1967.

VI

In een moderne psychiatrische inrichting moet men zoveel mogelijk vermijden de patiënt van de buitenwereld af te zonderen.

VII

Het gebruik van de naam "centigrade" voor graden Celsius in het Engelse en Zuideuropese taalgebied is onjuist.

Nat. Bur. of Standards, Washington DC.

VIII

Bij de vaststelling van normen voor de audiometrie dient men zich te baseren op de wijze van audiometreren zoals die gebruikelijk is in de klinieken voor Keel-, Neus- en Oorheelkunde.

IX

Men zou strengere maatstaven moeten aanleggen voor de publikatie van artikelen in medische tijdschriften en het aantal tijdschriften zou men moeten beperken.

X

Zo men in België op research gebied niet achter wil raken, zal er van staatswege meer financiële steun moeten komen.

XI

Het gebrek aan interesse van de Nederlanders voor het taalprobleem in België is betreurenswaardig.

ERRATA

Page	Line		
17	23	:	neurinitis to read neuritis
19	32	:	Cavella to read Cavaller
20	11	:	Pflatz to read Pfaltz
27		:	omit 5th line and insert: It is evident from the literature that most of the authors consider dys-
38	27	:	arhythmetical to read arithmetical
40	16	:	and to read an
46		:	mark: Fig. 4
47		:	omit: line 5 and 6
53		:	insert between line 41 and 42: lowing cold irrigation and a downward-vertical component fol-
65	5	:	our to read the
71	34	:	only to read mostly
	35	:	cold to read hot
	36	:	cold to read hot
	38	:	hot to read cold
75	31	:	tht to read the
	39	:	capital to read cipital
79		:	change para. 8 to read as follows: A strong negative correlation exists with t° and m°; with the number of pauses it is not very clear. With arousal the correlation between both becomes less obvious.
79		:	insert at the end of the page following „alternatives": $H_{1,2}$: „the pairwise observed random variables have unequal means" (two-sided use of the test; the two-sided tail probability p_2 must be used). $H_{1,r}$: „the mean of the first random variable is larger than that of the second" (right hand use of the test; the right hand tail probability p_r must be used). $H_{1,l}$: „the mean of the first random variable is smaller than that of the second" (left hand use of the test; the left hand tail probability p_l must be used). In this case the level of significance (i.e. the maximum permissable probability that the hypothesis H_0 is wrongly rejected) is fixed at 0.05. If the tail probability p_2 (p_r, p_l) < 0.05, then the H_0 is rejected in favor of H_1. When this is not the case then on the basis of the observations H_0 cannot be rejected.
80	8	:	by to read during
Appendix		:	add to the abreviations: rh = right hot Fig. A 12.: lw to read lh; Fig. A 14.: rw to read rh; Fig. A 20.: lw to read lh.

Literature references : RIESCO-MACLURE to read RIESCO-MACCLURE

Amsterdam, 23 april 1970. Clément/Dysrhythmia during caloric nystagmus.