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The treatment of acute vertigo

Abstract Vertigo and dizziness are very common symptoms in the general population. The aim of this paper is to describe the physical and pharmacological treatment of symptoms characterized by sudden onset of rotatory vertigo. Acute vertigo can be subdivided into two main groups: (1) spontaneous vertigo and (2) provoked vertigo, usually by postural changes, generally called paroxysmal positional vertigo (PPV). Sudden onset of acute vertigo is usually due to acute spontaneous unilateral vestibular failure. It can be also fluctuant as, e.g., in recurrent attacks of Ménière's disease. Pharmacotherapy of acute spontaneous vertigo includes Levo-sulpiride i.v. , 50 mg in 250 physiologic solution, once or twice a day, methoclopramide i.m., 10 mg once or twice a day, or tiethilperazine rectally, once or twice a day, to reduce neurovegetative symptoms; diazepam i.m., 10 mg once or twice a day, to decrease internucelar inhibition, sulfate magnesium i.v., two ampoules in 500 cc physiological solution, twice a day, or piracetam i.v., one ampoule in 500 cc physiological solution, twice a day, to decrease vestibular damage. At the onset of the acute symptoms, patients must lie on their healthy side with the head and trunk raised 20°. The room must be quiet but not darkened. If the patient is able to swallow without vomiting, it is important to reduce nystagmus and stabilize the visual field with gabapentine, per os, 300 mg twice or three times a day. The first step of the physical therapy of acute vertigo is vestibular electrical stimulation, that is to say, a superficial paravertebral electrical stimulation of neck muscles, aimed to reduce antigravitary failure and to increase proprioceptive cervical sensory substitution. PPV is a common complaint and represents one of the most common entities in peripheral vestibular pathology. While the clinical picture is well known and widely described, the etiopathogenesis of PPV is still a matter of debate. Despite the different interpretation of PPV etiopathogenesis, the maneuvers described by Semont, Epley, or Lempert and their modifications are undoubtedly effective. For this reason the first therapeutic approach in acute provoked vertigo must be by means of one of these kinds of treatments.

Key words Vertigo • Acute vestibular failure • Pharmacotherapy • Physical therapy • Vestibular electrical stimulation

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Introduction

Vertigo and dizziness are very common symptoms in the general population. They have a prevalence ranging from 5% to 10%, according to different age groups. They are particularly common in individuals over 40 years of age and represent the first reason for a medical visit in patients over 65 years. The aim of this paper is to describe the physical and pharmacological treatment of symptoms characterized by the sudden onset of rotatory vertigo.

Acute vertigo can be subdivided into two main groups: (1) spontaneous vertigo and (2) provoked vertigo, usually

by postural changes, which is generally called paroxysmal postional vertigo (PPV).

Acute spontaneous vertigo

Sudden onset of acute vertigo is due to acute spontaneous unilateral vestibular failure, which means sudden asymmetrical vestibular functioning, i.e., the sensory input of one side is acutely diminished with respect to that of the other side. Asymmetrical peripheral functioning can be also detected without the patient complaining of vertigo or instability. Such an asymmetry can be stable, being a sequela of a former lesion. It can be also fluctuant as, for example, in patients with recurrent attacks of Méenière's disease. In a more restricted clinical context, unilateral vestibular hypofunction is considered a nonfluctuant unilateral vestibular loss, where compensation plays a fundamental role. The most striking form is the syndrome with sudden loss. In this syndrome the same signs and symptoms can be observed as seen after labyrinthectomy. The course is characterized by four stages [1–3]:

Stage 1: Irritation. In some cases the onset of the syndrome may be characterized by a rather vague dizziness. At the moment the spontaneous nystagmus beats towards the affected side and normal caloric reaction can be observed.

Stage 2: Sudden loss of function or paralysis of the system. Typical rotatory vertigo is now experienced and spontaneous nystagmus beats towards the normal side. The caloric reaction is absent or reduced at the affected side. Patients generally present at the emergency room of the hospital during this stage, and hospitalization is common.

Stage 3: Central compensation. There is a progressive decrease in vertigo as well as in spontaneous nystagmus. This occurs independently of the persistence of unilateral weakness or areflexia at the affected side revealed by caloric stimulations. Provocative maneuvers (especially head shaking) reveal vertigo and nystagmus.

Stage 4: Recovery. When the function of the affected side recuperates, this may lead to a spontaneous nystagmus, reversed in direction, i.e., beating towards the affected side.

Therapy should be directed to: (1) decreasing the neurovegetative symptoms, (2) decreasing antigravitary failure of the affected side, (3) decreasing oscillopsia due to nystagmus, (4) activating sensory substitution phenomena, (5) decreasing internuclear inhibition that decreases progression of functional compensation, (6) re-activating coordination, and (7) decreasing spatial disorientation (vertigo).

In the early phase of acute vertigo, symptoms are characterized by nausea and vomiting. Rotatory vertigo is present in every position of the head and the body, slightly less when lying on the opposite site of the lesion, and includes symptoms of oscillopsia due to nystagmus and unsteadiness, typically with standing and gait deviations toward the affected side. The first mechanism of recovery is internuclear inhibition that can lead to a bilateral reduction of the vestibular responses. In the acute phase, drug treatment is necessary. The aims of pharmacotherapy are:

1. To decrease neurovegetative symptoms and general patient distress: Levo-sulpiride i.v., 50 mg in 250 physiologic solution, once or twice a day; methoclopramide i.m., 10 mg once or twice a day; or tiethilperazine rectally, once or twice a day.
2. To decrease internuclear inhibition: diazepam i.m., 10 mg once or twice a day.
3. To decrease vestibular damage: magnesium sulfate i.v., two ampoules in 500 cc physiological solution, twice a day or piracetam i.v., one ampoule in 500 cc physiological solution, twice a day. At the onset of the acute symptoms, patients should lie on the healthy side, with the head and trunk raised 20°. The room should be quiet but not darkened.
4. If the patient is able to swallow without vomiting, it is important to reduce nystagmus and stabilize the visual field: gabapentine, per os, 300 mg twice or three times a day.

The first step of the physical therapy of acute vertigo is vestibular electrical stimulation (VES) [4–7], i.e., a superficial paravertebral electrical stimulation of the neck muscles. VES is aimed to reduce antigravitary failure and to increase proprioceptive cervical sensory substitution. Electrical stimulation (ES) is a noninvasive technique that provides nerve and/or muscle stimulation by means of surface electrodes through hand-held electrostimulators called TENS stimulators (transcutaneous electrical nerve stimulations). We know that vibration can provide specific exogenous stimulation of the propriocetors of the paravertebral muscles. Our previous experimental experience showed that vibration of the paravertebral muscles can be substituted by electrical stimulation. In fact, it has been widely shown [8–12] that the vestibular nuclei are really polysensorial relais and that they are not only correlated with the labyrinth and that it is not possible, under natural circumstances, to activate the vestibular receptors without implicating other force-sensitive receptor systems that convey information relevant to spatial orientation [13, 14].

On the basis of this extensive, but physiologically justified, point of view of the vestibular system, we named the superficial paravertebral electrical stimulation *vestibular electro-stimulation* (VES). According to the neurophysiological evidence, electrodes must be placed on the paravertebral muscles opposite to the affected side (the side of the direction of the spontaneous nystagmus) and on the trapezius of the affected side. Two stimulations have to be performed per day for at least 1 h, each. During the first half hour patients remain in bed, possibly in the most com-

fortable position lying on the healthy side, in the light, and should try to keep their eyes open and fixate a target on the opposite position of the visual field (on the side of the affected labyrinth). In this phase it is better if visual prisms are also used. Generally low prisms are used, e.g., 3°–5°, with the basis in the opposite side of the lesion [15, 16]. After a half hour of VES the patient, under the effect of drugs, must reach an upright position and then, accompanied by the therapist, begin walking. Gait must be practiced during VES, wearing visual prisms, and the patient taught to fixate a target in front of him [17]. After taking some steps, the patient must then turn around again. The sense of rotation must be in the sense of the healthy labyrinth. Instructions for re-fixating a target must be furnished.

After the first day of treatment, simple in-bed exercises must be performed, twice a day, for at least 20–30 min in each session [18–20]:

1. The patient fixates a target on the ceiling and slowly moves his head toward the right and the left.
2. The patient looks for two equidistant targets on the ceiling and then he fixates them alternately, first moving only the eyes and then moving only the head.
3. The patient rotates his head in the direction of the affected labyrinth and fixates a target on the lateral wall. Then he straightens his head, maintaining the visual fixation. He counts to 10 and then he rotates his head again.

When it is possible to maintain a sitting position, the following exercises must be performed during VES and wearing visual prisms:

1. The patient extends his arms and lifts his thumbs. He puts his arms about 50 cm from his eyes, along a horizontal plane, and then fixates alternately his thumbs. He begins slowly and then increases progressively the velocity of alternate fixation.
2. The patient extends his arms and lifts his thumbs. He puts his arms about 50 cm from his eyes, along a vertical plane, and then fixates alternately his thumbs. He begins slowly and then increases progressively the velocity of alternate fixation.
3. The patient extends his right arm and lifts his thumb. He slowly moves his arm to-and-fro, first in a horizontal direction and then in a vertical direction. He follows his thumb with the eyes only, first slowly and then increasing progressively the velocity of thumb displacement.
4. As above but also moving the head while trying to keep the eyes still at the same time.
5. The patient first moves his head slowly and then faster in all directions, fixating a target straight in front of him.
6. The patient looks for three targets placed, respectively, in front of him, at his left, and at his right. He fixates the frontal target, then he rapidly moves his head, fixating the target on the right. Now he turns his head to the left, maintaining the fixation of the target on the right. He fixates the frontal target. Now he rapidly moves his head to the left and fixates the target on the left. Then he turns his head to the right, maintaining the fixation of the target on the left

Paroxysmal positional vertigo

Paroxysmal positional vertigo (PPV) is a common complaint and represents one of the most common entities in peripheral vestibular pathology. The principal complaint is the occurrence of sudden attacks of vertigo precipitated by certain head positions. The attacks can be induced by rolling over in bed, to one side or to both sides, by sudden movement of the head, by extending the neck, by looking upwards, for example, when reaching for an object from the top shelf in the kitchen, by bending the head backwards when washing the hair, in lying down beneath a car, or throwing the head backwards to paint a ceiling. The patient sometimes recognizes that the onset of the vertigo is associated with this critical position and will say he does his best to avoid it.

The initial occurrence is usually experienced on awakening in the morning, which is considered characteristic, or during the night. Many patients are frightened by the intense vertigo and try to shut out the sensation by closing their eyes. Nausea, often accompanied by anxiety, even vomiting, may follow the attack in few cases. In rare cases the nausea persists for hours. Vertigo is of short duration (<1 min) but, because the intensity of the vertiginous sensation, a longer duration can be assigned to the attack.

Generally speaking, two main kinds of PPV can be recognized:

1. The most frequent is elicited by the so-called Dix-Hallpike maneuver that induces a typical horizontal-rotatory geotropic nystagmus while the patient is lying during simultaneous rotation and hyperextension of the head. Nystagmus appears some seconds after the execution of the maneuver (latency) and it is decreased by the following repetition of the same maneuver (fatiguability due to habituation phenomena).
2. Vertigo and pure horizontal nystagmus are provoked by rolling the head from side to side while the patient is recumbent. In this case (so-called MacClure maneuver), two kinds of nystagmus can be observed, geotropic or ageotropic nystagmus.

While the clinical picture is well known and widely described, the etiopathogenesis of PPV is still a matter of debate. The most accepted theory is the lithiasis theory, originally described by Schucknecht (1974) and termed cupolithiasis. According to this theory, then generally accepted, substances having a specific gravity greater than the endolymph, and thus subject to movement with

changes in the direction of gravitational forces, come into contact with the cupola of the posterior semicircular canal (in the case of vertigo induced by the Dix-Hallpike maneuver) or of the lateral semicircular canal (in the case of vertigo elicited by the Mac Clure maneuver). The change in position of the labyrinth during movement of the head provokes the displacement of the cupola by direct influence of these heavy substances on it. Recently cupolithiasis has been the subject of discussion and the theory of free-floating particles into the canal (so-called canalolithiasis) postulated.

On the basis of these theories, maneuvers aimed to "liberate" the cupola or the canal from floating particles have been proposed. The two main ones are the Semont [14] and the Epley [21, 22] maneuvers. They are different in movement and in rapidity but they are both effective in at least 80%–90% of patients affected with so-called lithiasis of the posterior semicircular canal, the most frequent kind, after one or two single treatments. For the less frequent kind, horizontal semicircular canal lithiasis, Lempert proposed an effective maneuver [23].

Lithiasis theories are still controversial because they are not able to explain all the features of PPV, especially when the proposed maneuvers are not effective or when signs and symptoms are not as typical as those described above (about 10% of the patients suffering from positional vertigo). Another explanation considers the otholiths as the source of nystagmus, but in the sense of influencing and favoring a nystagmus primarily elicited by the canals. Emphasis is placed upon the otholith-canal interaction. According to Ledouz, the vertical canals and the utricles form a system of sensory organs functioning synergistically. The system of the controlateral side has an antagonistic function. Asymmetry in this coupling can bring about a disturbing situation, i.e., vertigo and nystagmus. Some authors proposed that central mechanisms are involved. Ledoux argued that the modalities of the appearance of PPV can only occur through a modification in the central channels of the equilibrium normally existing between the influxes of both labyrinths. This rupture is checked by an inhibition phenomenon during the attack or during repeated provocation. McCabe explained PPV as the result of focal microscopic loss of otholithic macular epithelium by either a neurovirus or a microstroke.

According to our concepts [15, 16, 24] and some neurophysiological evidence [25–28], positional vertigo can be interpreted as a proprioceptive mismatch between the so-called general proprioception (from muscle, ligaments, and joints) and the so-called special proprioception (from maculae and cristae) according to the spino-cerebello-vestibulo-spinal circuitry; this means that the different proprioceptive information is integrated and elaborated in the cerebellum and then projected on the vestibular nuclei.

Despite the different interpretation of PPV etiopathogenis, the maneuvers described by Semont, Epley, or Lempert and their modifications are undoubtedly effective. For this reason the first therapeutic approach must be by means of one of these kinds of treatments.

Semont maneuver (original description)

The patient lies on the affected side for at least 3 min. Then the head is rotated upward about 105° in order (in the cupolithiasis theory) to allow the displacement of the heavy particles at the basis of the posterior semicircular canal with downward deflexion of the cupola. Then the therapist firmly takes the head of the patient and decisively brings the patient to the opposite side with a simultaneous rotation of the head downward about 195°. Usually, in this position, after a few seconds, a "liberating" nystagmus toward the affected side appears. When nystagmus and vertigo disappear, the patient returns slowly to the sitting position

Epley maneuver

According to the original description of the technique, the patient is premedicated with transdermal scopolamine or diazepam, 5 mg, the night before. The patient is seated on an examining table so that when the patient is brought to the supine position, the head extends beyond the bed. The operator is located directly behind the patient and an assistant at the patient's side. Then he lies downward with a slight hyperextension of the head and a simultaneous 105° rotation towards the affected side. In this position nystagmus and vertigo appear. The patient remains in each position 6–13 s but this may be extended to more than 30 s. Then the head is rotated on the opposite side toward the healthy labyrinth for 90°. At the same time as the head is rotated the trunk and the legs also rotate towards the healthy side. In this position, usually, a "liberating" nystagmus is observed. Then he completes the rotation of the legs and returns to a sitting position, keeping the head turned toward the opposite side with respect to the affected labyrinth.

Personal maneuver for PPV elicited by Dix-Hallpike positioning (Epley modified)

The patient is seated on the bed. Then a Dix-Hallpike positioning is performed in order to elicit symptoms and nystagmus. After the end of the nystagmus, the patient remains in a recumbent position with the head extended and rotated for 30 s. With a slight traction on the head, the therapist rotates gently the head of the patient to the oppo-

site side and the patient rotates simultaneously his trunk and his legs, taking his legs off the bed. This position is maintained for 30 s, and a "liberating " nystagmus is usually observed. Then the patient returns to a sitting position, without turning the head.

Lempert maneuver

This maneuver represents an adaptation of Epley's posterior canal repositioning procedure and it aims to shift heavy particles (in the canalolithiasis theory) ampullofugally toward and beyond the horizontal canal opening into the utricule. The maneuver consists of 270° head rotation around the supine patient's longitudinal axis; the rotation is performed in rapid steps of 90°. The patient is in a recumbent position. Then the head is rotated toward the affected side (MacClure position). In this position nystagmus is elicited. When vertigo and nystagmus have disappeared, the therapist performs a complete rotation of the head and the body of the patient in steps of 90° until complete 270° rotation has been achieved. Head positions are maintained for between 30 and 60 s until all nystagmus has subsided.

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