A short guide to the

MANAGEMENT OF
THE DIZZY PATIENT
Preface

This practical guide is aimed at professionals who deal with dizzy patients in their everyday practice. The guide will help you in a relatively simple way to deal with dizzy patients. This practical approach is divided into three parts:

I. understanding and explaining vertigo and dizziness,
II. managing the dizzy patient, and
III. the most common diseases causing dizziness.

The first section provides an overall understanding of balance problems. With it you can explain the often-frightening experience to the patients, which is a very important part of the treatment. In-depth anatomical or physiological knowledge of the balance system is not given, as this can be found in excellent books on anatomy, physiology, ENT diseases, and neurology.

The second section gives suggestions on how to examine the patient and take their medical history. You should read through this chapter first and then follow the schematic, highlighted summaries.

The third section presents the most common diseases causing dizziness. They are described simply, focusing on symptoms, necessary examinations, and treatments. The background information can be read at leisure, whereas the paragraphs on the history, symptoms, and examinations can be used as a guide when you have a particular diagnosis in mind.

It is important to stress that this list of diseases and dizzy symptoms is not by any means complete. It is also important to emphasize that the contents primarily reflect our personal thoughts at the time of writing. Hopefully, with advancing knowledge, we will find that there are several shortcomings in this text. Until then we hope that our colleagues, students, and their patients will benefit from this practical guidance.

May 2010, Måns Magnusson, Michael Strupp
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1. Understanding and explaining dizziness

To maintain a desired position and movement in relation to the perceived horizontal/vertical we rely on sensory information from the balance organs, the eyes, proprioception and pressure sensors in the skin. This information reaches the central nervous system (CNS) through a multitude of different nervous pathways (Fig1). In the CNS it is collected and assessed, stored, and used to provide working orders – i.e. a motor command – for the muscles of the body and for eye movements. The aim of these orders is to balance the body, for example, in an upright position and keep the eyes on the target fixated to avoid apparent movements of the visual surrounding (so-called oscillopsia). The small movements generated, trigger reactions from the sensory system and back to the CNS, thereby producing new orders, so-called “feedbacks”. As long as the sensory input from these organ systems match with the previously stored information, balance is maintained. If the sensory systems detect and report different movements, there may be a sensation of vertigo, dizziness and a disturbance of balance and nausea may develop.

Figure 1: Schematic Illustration of human postural control

How do we maintain upright stance while moving on two legs?

What is the significance of these sensory systems?
The five balance organs in each inner ear register position and movements of the head in space. The three semicircular canals in each inner ear detect angular acceleration in the plane of the canal, i.e., turning head movements. However, the canals cannot detect linear acceleration. This is registered by the two otolith organs, i.e., the utricle and saccule. As the most common linear acceleration is gravity, the otolith organs also define the position of the head relative to gravity.

We have two sets of balance organs: one on each side of the skull; they are mirror images of each other. Turning the head to one side causes increased neural activity of the semicircular canals on that side, whereas the activity of the other side is simultaneously reduced. The difference in influx from the two inner ears is registered already in the vestibular nuclei in the brainstem. It results in an activation and inhibition of eye motor nuclei, causing the eyes to turn in the opposite direction of the head but at the same speed. This is the basis for the vestibular ocular reflex (VOR), which allows us to keep the eyes fixed on an object while we move our head up or down or to the side. Without this reflex we would not be able to see clearly while walking and we would experience oscillopsia; the VOR thus compensates for head movements that occur during walking.

The difference in the influx of neural impulses between the two sides determines not only the direction and speed of the induced eye movements but also the vestibular spinal reflexes. Even without any movement, there is a baseline neural activity from the inner ears. If there is sudden damage to one of the inner ears, causing a loss or reduction in neural activity on this side, a difference in neural activity arises in the vestibular nuclei and brainstem. Such asymmetry is equivalent to turning the head and falling. It is interpreted as a continuous turning movement, generating nystagmus and a falling to the lesioned side. Simultaneously there is a feeling of nausea and sickness.

Information mainly from the otolith organs regulates the tonus of the most important muscles that maintain upright stance while standing and walking. A disturbance or a side difference in the influx can lead to a fall or swaying to one side or the other.

The inner ear function can be described to the patient as a balance between stimulation and inhibition. If you lose the function on one side, the continuous neural activity from the other healthy side will be the same as one experienced when turning, and you get a spinning sensation towards the healthy
side. As the information from the balance organs is continuously compared to visual as well as proprioceptive information, the CNS will, in time, at least partially compensate for the loss. Another way of explaining this to the patient is to describe a scale with a balance organ in each of the pans (Fig. 2). If you remove the weight of one balance organ from one pan, the scale will tip to the other side. This also happens with unilateral inner ear damage. The compensation by the CNS is a re-adjustment of the scale to equilibrium, even though the weight has been removed from one pan. But this is only true for so-called static conditions. If the function of the labyrinth does not recover, the patients will always have difficulties during walking or head movements because the dynamic deficit of the VOR cannot be compensated for by any other system.

Vision provides us with the perception of our position in space and the movements of the surroundings in relation to us. The latter can be caused by our own movements or by movements of the surroundings. Sometimes it can be difficult to decide whether the surroundings are moving or if we ourselves are moving, as experienced when sitting in one train looking at another when suddenly one starts to move. Vision allows us to orient ourselves.
towards the horizon and to decide what is up and what is down. We expect a supporting surface to be perpendicular to our upright body position.

Vision also allows us to identify obstacles in our path or threats to our balance in advance and thereby gives us ‘feed-forward’ or ‘anticipatory’ information in order to avoid these obstacles or challenges. This mechanism reduces our dependence on other sensory systems and the risk of situations in which we would lose our balance. This is especially important for the elderly and for frail subjects.

Finally, vision allows us to cancel vestibular reflexes by means of visual fixation, a mechanism that is also called visual suppression of the vestibulo-ocular reflex.

_The term proprioception refers to input from the sensors of the musculoskeletal system. They provide information to the brain about the state of contraction, the length, and the load on all the muscles or tendons in the body. They also register any tension in the joint capsules, thereby giving information on the angulations of the joints. When all this information is conveyed to the CNS, it estimates the body's position in space, i.e., where the different body segments are in relation to each other and how they are affected by weight and gravity._

_The proprioceptive information from the neck and shoulders is particularly important, for it defines the position of the head in relation to the trunk and the rest of the body. Such information is needed in order to utilize the information from the vestibular organs and eyes for adjusting body posture and balance, as these organs are situated in the base of the skull._

_Several different types of pressure receptors in the human skin are able to detect a small indentation of the surface as well as continuous changes in pressure distribution. The receptors in the sole of the foot register those parts of the sole that bear weight as well as any slow or sudden change in the pressure on the underlying surface. These pressure changes reflect body movements. In the elderly, such information may be disturbed due to peripheral neuropathies and foot deformities. Therefore, the use of supportive and light-weight footwear should be recommended._
Integration of balance information and the motor control

As described above, the information on position and movements from the different sensory systems converges and is integrated in the CNS. It is matched with what we intend to do, what position we are in, and what movement we intend to produce. This in turn leads to working orders for the body’s muscles (motor commands), which will cause corrective body movements when put in action. This change in the sensory information leads to new integration and new corrections. In this way there is continuous control of body position and balance, a so-called active feedback system.

We also use our vision and memories to detect any threat to our balance in advance and to prepare us for what we need to do. This is called a feed-forward (or anticipatory) activity. Most of these feed-forward activities are gradually learned by the CNS until they become automatic and no longer require intervention of the conscious mind.

Balance disturbances can be caused by disturbances in the sensory information provided by one or several of the previously mentioned three sensory systems. Reduced balance control can also be due to poor integration within the CNS, in particular the cerebellum and the brainstem, or to insufficient corrections due to disturbances of the muscle control. A patient with balance disturbance has often had a fall or reports falls, veering off line, or a sensation of general unsteadiness. This can often be observed during the examination. The inability to walk, stand still, or to move smoothly can be measured.

When patients describe unsteadiness, it may be linked to a simultaneous change in their balance. However, this disturbance may not always be observable.

Vertigo and Dizziness are sensations. We realize that the information from the different sensory systems do not match either each other or our expectation. This is called a sensory mismatch. If the brain loses information from one of the balance organs, e.g., after vestibular neuritis in one ear and the other ear continues to work normally, the vestibular centers in the brainstem interpret this as if we are continuously spinning towards the normal side. The vestibular ocular reflex will cause the eyes to move in the opposite direction (slow phase). This so-called spontaneous nystagmus can be observed.
The patient experiences rotational vertigo due to the mismatch of sensory input from the inner ears and the eyes. A sensory mismatch can also arise when the proprioceptive information does not match the vestibular information or when the information from the different sensors within the inner ear, such as the semicircular canal and the otolith organ, does not match. The latter is often the case in motion sickness.

Dizziness can, to a certain extent, be considered the conscious representation or a milder form of balance impairment. Even though we may realize that something is not entirely right, we may not always be able to objectify these feelings. This may be the case in non-organic dizziness. Typical examples are the dizziness arising in phobic postural vertigo and from anxiety/panic attacks. There is no doubt that the sensation of balance is also related to our cognition even in other ways. It has been reported that nearly half of patients who have not had adequate rehabilitation after vestibular neuritis may have persistent anxiety. Regardless of the underlying cause of the dizziness, the patients always experience more dizziness if they are simultaneously tired, stressed, or have emotional problems (Fig. 3).
2. Managing the dizzy patient

The patient may be examined during, directly after an acute attack, at the beginning of the complaint, or at a later time when acute symptoms are no longer present. When a patient is examined during the acute phase, it is important to first exclude life-threatening conditions. In the non-acute phase there is more time to plan a strategy for examination and hopefully to establish a diagnosis.

The following management plan is therefore suggested:

<table>
<thead>
<tr>
<th>ACUTE DIZZINESS</th>
<th>ACUTE TREATMENT</th>
<th>NON-ACUTE DIZZINESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute status, vital signs</td>
<td></td>
<td>Thorough history taking</td>
</tr>
<tr>
<td>Acute history</td>
<td></td>
<td>Full physical examination</td>
</tr>
<tr>
<td>Additional history</td>
<td></td>
<td>Information to the patient</td>
</tr>
<tr>
<td>Additional examination</td>
<td></td>
<td>Planning further tests or treatments</td>
</tr>
</tbody>
</table>

**ACUTE STATUS**

Examination of the patients with a first attack or ongoing acute vertigo

<table>
<thead>
<tr>
<th>Examine</th>
<th>Think of</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Consciousness</td>
<td>Acute CNS damage, infarction (brainstem, cerebellum), intracranial infratentorial hemorrhage, meningitis, intoxication, circulatory collapse</td>
</tr>
<tr>
<td>2. Blood pressure, pulse, examination of the heart and lungs</td>
<td>Cardiac arrhythmia, circulatory failure</td>
</tr>
<tr>
<td>3. Stiffness of the neck</td>
<td>Meningitis? Subarachnoid bleeding?</td>
</tr>
<tr>
<td>4. Examination of cranial nerves, mobility and coordination/sensibility in the extremities/looking for ataxia</td>
<td>CNS lesions Brainstem/cerebellar damage</td>
</tr>
<tr>
<td>5. Nystagmus/eye movements Head impulse test Positioning maneuvers (if tolerated)*</td>
<td>Normal head-impulse test and nystagmus strongly suggest CNS (cerebellar-brainstem lesion); (Extra: is also true for signs of central ocularmotor dysfunction such as gaze-holding deficit, saccadic smooth pursuit, impaired saccades)*</td>
</tr>
<tr>
<td>6. Otoscopy</td>
<td>Otitis, cholesteatoma</td>
</tr>
</tbody>
</table>

*HINT: Hallpike-Impulse test-(gaze) Nystagmus – Torsional eye movement when cover-uncover test is done, was recently suggested by Newman-Toker and others to be a good discriminator between central nervous and peripheral vestibular disorders in acute onset of vertigo
ACUTE HISTORY

- When and how did the problem start?
- How long did it last?
- What type of vertigo? Rotational or postural or disorientation or lightheadedness?
- Did it occur spontaneously or was it induced, e.g., by changes in head or body position?
- Pain (head, chest, ears)?
- Hearing? Sudden change/previously reduced hearing, side difference?
- Additional problems or symptoms? Double vision? Problems with speaking or swallowing? Weakness of the arms or legs? Headache? Nausea?
- State of consciousness? (Ask family or other people present)

Comments

Consciousness
Reduced consciousness suggests neurological or circulatory disease or intoxication. Patients with loss of inner ear function can be tired or their ability to express themselves may be limited due to nausea; otherwise, however, they are fully alert.

Blood pressure/pulse/heart/pulmonary status
Irregular heartbeat, a drop in blood pressure, or increased blood pressure indicate a circulatory cause; an ECG should be done immediately (the patient should be seen by a specialist in internal medicine). Patients with vestibular damage, nausea, and sickness could have a parasympathetic overload with a possible decrease in pulse and blood pressure. This, however, should not be prominent. If it is, then a circulatory disease should first be excluded.

Stiffness of the neck
This should raise suspicion of meningitis, encephalitis, or subarachnoid bleeding (the patient should be sent immediately for a CT scan/lumbar puncture or to consult the infectious disease clinic or departments of neurology/neurosurgery) if possible.
Examination of cranial nerves, mobility and coordination/sensibility in the extremities/looking for ataxia

Besides the ocularmotor system (see below), other cranial nerves have to be examined sooner or later, in particular the visual nerve (defects of the visual field), trigeminal, facial, glossopharyngeal, vagus and hypoglossus. Although the n IX and n X latter may have to be restricted to asking the patient about swallowing or listening for hoarseness in the acute situation, were the patient may not tolerate the examination. Further, the extremities should be examined, looking for weakness or problems with coordination and ataxia (finger-to-nose test) as well as impaired sensation. An acute onset of central neurological deficits most often indicates a vascular origin. In these cases a CT scan has to be performed to exclude intracranial bleeding and thereafter the patient requires neurological treatment.

Nystagmus

A horizontal rotatory nystagmus suggests damage either to the inner ear or to infratentorial structures. If the patient has a horizontal torsional nystagmus which can be suppressed by visual fixation and is therefore more prominent under Frenzel’s glasses, it is probably a spontaneous nystagmus due to a peripheral vestibular lesion. If it is not suppressed by visual fixation, it is a fixation nystagmus caused by a central, most often brainstem lesion. Pure vertical or torsional nystagmus also suggests a central origin.

In addition, positioning maneuvers could be performed to look for benign paroxysmal positioning nystagmus (BPPV) or central positioning or positional nystagmus if tolerated.

The tympanic membranes

Vertigo plus otitis media equals labyrinthitis. The patient should be managed in consultation with an ENT specialist as an emergency case.

Pain

Dizziness/vertigo and pain are an unhappy combination. Although headache may be unspecific, it may indicate vestibular migraine or in rare cases intracranial causes, such as intracranial bleeding. Chest pain should suggest the possibility of cardiac emergencies. Pain from the ear without otitis media should suggest a possibly evolving zoster oticus (Ramsay-Hunt syndrome).
After the acute evaluation of the patient, examinations are usually complemented by an ordinary ECG, another history is taken, and usually also another physical examination is performed, if possible. A patient with acute vertigo often needs to be hospitalized, at least for observation, especially if the patient cannot manage alone. A good rule of thumb is to hospitalize the patient if there is a nystagmus or central ocularmotor dysfunction or other neurological signs or symptoms. Severe vertigo without nystagmus indicates that it is not caused by a peripheral vestibular lesion.

Taking a thorough history is the most important tool for assessing the non-acute dizzy patient. This together with a full otoneurological examination may be a time-consuming task. However, there is nothing gained by taking shortcuts, since the examination will have to be repeated on later occasions. If you do not have time to do a complete examination during the first consultation, the patient should be asked to come back a second time when more time has been allocated.

**ADDITIONAL HISTORY**

**Hereditary:** Migraine, eye diseases, cerebellar or ocularmotor disorders, hearing impairment, vascular diseases.

**Social history:** Alcohol consumption (particularly in males), drugs, work, family, stress?

**Previous medical history:** Cardiovascular/pulmonary, blood pressure, gastrointestinal or urogenital diseases, CNS diseases or trauma. Ear diseases. Psychiatric diseases, treatment with ototoxic drugs or antibiotics? Hospitalization?

**Medication:** Is the patient currently being treated for any disease? Particularly of interest are psychopharmaca and medication to control blood pressure, cardiac arrhythmias or vertigo. Many drugs cause dizziness as a side effect.

**Hypersensitivity:** always ask about any known reactions.
### Present situation

<table>
<thead>
<tr>
<th>Current Vertigo:</th>
<th>Are you dizzy all the time or only occasionally, i.e., in attacks? If you suffer from attacks, how long do they last?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Are you dizzy right now and, if so, describe the sensation.</td>
</tr>
<tr>
<td></td>
<td>Are you dizzy only when standing and walking?</td>
</tr>
<tr>
<td></td>
<td>Do you suffer from vertigo when you turn around bed or get up in the morning or lie down in bed?</td>
</tr>
<tr>
<td></td>
<td>Have you had a fall or tripped; did you sustain any injuries? If yes, this would indicate a significant organic balance disturbance.</td>
</tr>
</tbody>
</table>

### Characteristics of the dizziness: (Try to make the patient describe the sensation without using the words vertigo or dizziness.)

- **Rotation/spinning sensation.**
  The patient has a sensation that the room is spinning around him/her or that the patient is spinning. In which direction is the spinning – anticlockwise or clockwise?

- **Unsteadiness.**
  The patient describes a sensation of swaying or of rocking while walking on a boat deck, a ‘nautical’ sensation.

- **Uncharacteristic.**
  Neither of the above. Light-headed or disoriented?

- **Pre-syncpe, a feeling of passing out**

Ask the patient to describe the first and last attacks of vertigo. This allows the patient to focus better and consequently the description of an attack is usually clearer.

Triggers that make dizziness/vertigo worse: head/body movements usually indicate a vestibular cause. Positional changes like turning in bed or lying down could indicate BPPV. Vertigo in certain environments or situations may indicate psychogenic causes or sensory overload. Vertigo induced by coughing, sneezing or pressing is typical for perilymph fistula.
Duration: try to find out the minimum and the maximum of the duration of the attacks

<table>
<thead>
<tr>
<th>Duration</th>
<th>Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 – 2 seconds:</td>
<td>Unspecific, Vestibular paroxysmia, phobic postural vertigo, paroxysmal brainstem attacks, transient ischemic attack, Psychogenic</td>
</tr>
<tr>
<td>15 – 30 seconds:</td>
<td>If triggered by positional change, BPPV Vestibular paroxysmia, paroxysmal brainstem attacks, transient ischemic attack. If induced by changes of pressure, perilymph fistula</td>
</tr>
<tr>
<td>1 – several minutes:</td>
<td>Vestibular migraine</td>
</tr>
<tr>
<td>1 – 8 hours:</td>
<td>Simultaneous hearing loss: Menière’s disease. Vestibular migraine</td>
</tr>
<tr>
<td>2 – 3 hours and up to 1 – 2 days</td>
<td>Vestibular migraine or transient ischemic attack (&lt;24h)</td>
</tr>
<tr>
<td>24 – 48 hours/weeks:</td>
<td>Vestibular neuritis</td>
</tr>
</tbody>
</table>

Frequency

Number of episodes per day, week, month or year. Clusters or increases in relation to certain time periods/vacation could indicate migraine or Menière’s disease.

HEARING LOSS/TINNITUS

- Unilateral, bilateral loss, or tinnitus?
- Fluctuating loss or tinnitus?
- Simultaneous with vertigo?

Neurological symptoms

Symptoms’ duration and presence in relation to vertigo
- Double vision, blurred vision, oscillopsia
- Reduced vision, defects of visual field
- Perioral paresthesia not correlated to hyperventilation
- Facial weakness
Problems with speech, swallowing, tongue movements
Extremities: paresis, impaired sensation, paresthesia, coordination problems, limb ataxia

PAIN

in the head, chest, neck, ear?
Tenderness in the neck?
Pain with acute onset of dizziness should be taken seriously.

Comments to the Above:

ACUTE VERTIGO

If the patient describes an intermittent dizziness that occurs in attacks like Menière’s, BPPV, or vestibular migraine, this type of vertigo is, in general relatively simple to differentiate from the continuous vertigo usually triggered by cervical or multiple system involvement in the older population.

If the patient is dizzy during the examination, then his/her description of the vertigo/dizziness is linked to the possible presence of other losses of functions or caused by phobic postural vertigo. To be dizzy while walking and standing usually is typical of bilateral vestibulopathy. To be dizzy when standing up could be a sign of orthostatism. To be dizzy when standing only would be typical for orthostatic tremor. An important differential diagnosis of these symptoms is postural phobic vertigo. If the patient has fallen or tripped, it is usually a sign of a real disturbance of balance. Falls should not be disregarded as they themselves may be a threat to the patient’s future health.
The character of the dizziness

**THE SENSATION:**

Vertigo – rotational dizziness

A subjective sensation of movement when there is none or a sensation of a rotational movement in which either the room or the patient is spinning. The cause is often – but not always – in the inner ear or in the pathways involved in the vestibular ocular reflex. If the patient can be observed to simultaneously have nystagmus, the cause is most likely vestibular or a CNS disease. Single or recurrent attacks can suggest that the disease is variable as in vestibular paroxysmia, Menière’s disease or vestibular migraine. Sustained rotatory vertigo over several days to up to a few weeks is usually only seen in cases of unilateral vestibular lesion. The most common cause is vestibular neuritis. Vertigo due to positional changes is usually BPPV, but it can sometimes also be a sign of CNS disease (central positioning vertigo or vestibular migraine).

Unsteadiness

Unsteadiness is an uncharacteristic symptom. It could be a sign of vestibular or CNS disease. It is often reported by older patients with multiple organ deficiency, vascular disturbances, polyneuropathy, psychogenic or postural phobic/visual vertigo or space phobia. Bilateral vestibulopathy is an important differential diagnosis in these patients which is unfortunately often overlooked.

Pre-syncopes

This is a sensation of being about to faint; patients describe it as feeling like a blackout but without falling. This is usually related to cardiovascular problems such as cardiac arrhythmias, orthostatism, or a too vigorous anti-hypertensive treatment.

Further Characteristics

The first/last attack

Asking the patient to focus on one particular dizzy spell can often yield a better description of the attack and the patient’s sensations during it. It is also important to try to define the time of the first attack. This will provide information on whether or not there was some initial triggering event for an underlying deficit/damage and also on how long the problems have been present.
What triggers/aggravates dizziness?

Dizziness is commonly made worse by changes in head or body positions, if the patient has a vestibular disease. Dizziness with positional changes, as when looking upwards or turning around in bed, is typical for BPPV. Dizziness when bending the head backwards is also related to BPPV and is usually not due to vascular diseases. Dizziness triggered by certain places or situations like supermarkets, escalators, and elevators indicates either a sensory mismatch or psychogenic causes.

Duration of the symptoms

- Attacks lasting only seconds can be caused by a number of diseases, e.g., a phobic postural vertigo, vestibular paroxysmia or vascular CNS disease.
- Vertigo triggered by positional changes or head movements lasting up to half a minute indicates a BPPV.
- Attacks lasting minutes can be seen in vestibular migraine or Menière’s disease.
- 10 – 30 minute-long vertigo attacks in conjunction with headaches could be an aura in vestibular migraine.
- Vertigo attacks lasting 1 – 8 hours, together with fluctuating hearing and tinnitus, point to Menière’s disease.
- Vertigo attacks lasting 1 – 2 days may indicate vestibular migraine and in some cases a vascular event.
- Vertigo with a sudden onset but gradual reduction over several days indicates vestibular neuritis or some other acute vestibular damage.

The frequency of attacks

The frequency indicates not only how much the patient is suffering but is also of diagnostic importance. Patients with vestibular paroxysmia often suffer from several attacks per day. On the contrary, it is very rare to have more than one attack of Menière’s disease or vestibular migraine a day.

Reduced hearing/Tinnitus

Vertigo, together with reduction in hearing and tinnitus, is very suggestive of inner ear or nerve pathology. Unilateral symptoms are more specific than bilateral. If the patient describes a continuous, pulsatile, unilateral tinnitus, it is necessary to perform an auscultation of the mastoid and that side of the
Comments on the physical examination

Head in order to rule out an ‘objective tinnitus’, as occurs in certain vascular pathologies. A gradual progressive hearing loss with unilateral tinnitus and discreet unsteadiness always suggests a vestibular schwannoma or other pontine angle tumours.

Simultaneous neurological symptoms

The presence of simultaneous neurological symptoms points to CNS disease as the cause of the symptoms. Symptoms like double vision, speech problems, hoarseness, and swallowing difficulties usually indicate brainstem involvement. The symptoms occur abruptly in stroke, and they may initially recede and then gradually progress as in patients with multiple sclerosis, whereas a space-occupying pathology usually leads to a progressive increase in the symptoms. In the presence of such symptoms, the patients need a full neurological examination with either a CT or, preferably, an MR scan.

Emotional changes

Vertigo is a cardinal symptom of anxiety disorders, but it may also be present in depression. It is therefore important to get comparative information, if possible, also from the patient’s family.

General impression

Observe the patient’s alertness, mimicry, stiffness, reduced emotional state, or signs of cardiac non-compensatory state.

Blood pressure, heart, and lung examinations

Examine to ascertain circulatory causes of the patient’s symptoms. If vascular causes are suspected, in non-acute dizziness/vertigo repeated measures of blood pressure and pulse may be needed to define for orthostatic dysregulation. The neurologist or radiologist may perform Doppler-/Duplexsonography to detect extra- or intracranial stenosis.

Ears

The tympanic membrane should always be examined in a patient with vertigo. The ears need to be assessed for acute otitis media, chronic otitis media with or without cholesteatoma, which could be a cause of labyrinthitis. If there is any question about the state of the ears, the patient should always be referred to an ENT surgeon. In an emergency situation, one should at the very least ask about the patient’s hearing.
**Hearing**

To determine whether a reported hearing loss is due to sensorineural or conduction loss the ‘hum’ test can be used. This test works like the Weber tuning fork test. The patient is asked to make a continuous midfrequency tone, e.g., to ‘hum’. If the patient hears the tone stronger in the ear with decreased hearing, there is a conduction loss and hence, a middle ear problem. If it is perceived only on the normal side, a sensorineural loss is possible.

### COMPLEMENTARY EXAMINATIONS OF A VERTIGINOUS PATIENT

<table>
<thead>
<tr>
<th>Examine</th>
<th>Think of</th>
</tr>
</thead>
<tbody>
<tr>
<td>General examination</td>
<td>‘Vital signs’ and mental state</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Arrhythmia in compensated heart failure, etc.</td>
</tr>
<tr>
<td>Heart/lungs</td>
<td>Don’t forget to perform an ECG, measure blood pressure and pulse and look for orthostatic dysregulation.</td>
</tr>
<tr>
<td>Eardrums</td>
<td>Otitis? Zoster? Perforations?</td>
</tr>
<tr>
<td>Oral cavity</td>
<td>Paresis of IXth, Xth, and XIIth nerves?</td>
</tr>
<tr>
<td>Larynx</td>
<td>Mobility of the vocal cord – voice quality</td>
</tr>
<tr>
<td>Neck</td>
<td>Metastases in the neck or in the parotid gland?</td>
</tr>
<tr>
<td>Cervical muscles</td>
<td>The sternocleidomastoid trapezius should always be examined, as well as the insertions of the muscles on the neck, the masseter muscles, and probably the pterygoid muscle, which has to be palpated via the oral cavity. Ask about reduced head movements, soreness, and points of pain.</td>
</tr>
<tr>
<td>Temporomandibular joint</td>
<td>Crepitations or soreness?</td>
</tr>
<tr>
<td>General neurology</td>
<td>Cranial nerves (see below), coordination, muscle strength, sensibility examination of the extremities, together with reflexes. Cerebellar test with dysdiadochokinesis, finger-to-nose, limb and gait ataxia, standing and walking with the eyes open and closed.</td>
</tr>
<tr>
<td>Cranial nerves</td>
<td>Sensibility on the face and on the cornea (V); movement of the face (VII); soft palate (IX), vocal cords (X), sternocleido and trapezius muscles (shoulder lift) (XI); tongue (XII)</td>
</tr>
<tr>
<td>Balance test while standing</td>
<td>Romberg’s test with the head in normal position, Unterberger’s test while walking on the spot for 30 seconds with eyes closed</td>
</tr>
<tr>
<td>Ocular examination (smooth pursuit)</td>
<td>Uncoordinated eye movements are signs of paresis of the eye muscles involving cranial nerves III, IV and VI. Description of spontaneous nystagmus, gaze-evoked nystagmus. Also look for saccadic eye movements during the smooth pursuit. Examination of the eyes with Frenzel’s glasses to see if there is any spontaneous or directional nystagmus when looking laterally and vertically.</td>
</tr>
<tr>
<td>Dix-Hallpike test</td>
<td>Always perform the Dix-Hallpike test. BPPV can be present in atypical ways in patients who also have other causes of dizziness. Observe nystagmus when patient is supine.</td>
</tr>
<tr>
<td>“Head-shaking test”</td>
<td>Note if there is any nystagmus when the shaking stops. Even 3 or more beats are pathological and suggest a vestibular side difference. Nystagmus usually beats towards the healthy side. The test does not differentiate CNS and peripheral vestibular lesions.</td>
</tr>
<tr>
<td>Head-impulse test</td>
<td>Also called head-thrust or Halmagyi-Curthoys test. A pathologic outcome strongly suggests a peripheral vestibular disorder.</td>
</tr>
<tr>
<td>Vision</td>
<td>Visual field examination according to Donders</td>
</tr>
<tr>
<td>Hearing</td>
<td>A full pure tone audiogram or tuning fork test with Weber and Rinnes tests. The ‘hum’ test may be easily used.</td>
</tr>
<tr>
<td>Laboratory test</td>
<td>Hb, CRP, TSH, B12, creatinine CDT and liver function tests according to the history. In many patients, an ECG is useful.</td>
</tr>
</tbody>
</table>
Oral cavity and throat
Glossopharyngeal or hypoglossus paresis should suggest brainstem lesions and a referral to a neurologist. Oral hygiene and the state of the teeth can sometimes give information about the patient’s social situation and the possibility of drug abuse.

Larynx
Vocal cord paresis also suggests neurological disease or malignancy that primarily spreads from the lungs and thyroid gland. If you are not comfortable with examining the vocal cords, at least listen to the patient’s voice and ask about any changes.

Neck
Suspicious lumps and nodules should be subjected to needle biopsies for cytology and followed-up with CT/MRI of the head to exclude intracranial metastases. Musculoskeletal tensions in the head and neck region may lead to dizziness or may arise secondarily, for example, vestibular side differences. Such a suspicion is supported if the patient describes a significant increase in tenderness of the muscles on one side of the neck compared to the other. So-called tender points in the patient’s description of a spreading pain. Sometimes these points are so tender that the patient will jump when palpated; this is referred to as ‘jump signs’. A referral to the physiotherapist to confirm the suspicion or to initiate treatment is recommended in these cases.

Neurological examination
Any paresis is indicative of a CNS lesion. In older patients, particularly those with diabetes, it is recommended to use a tuning fork resonating at 128 – 256 Hz when struck against the basal joint of the big toe, the medial malleolus, and midway on the anterior edge of the tibia. Reduced vibration sensation suggests polyneuropathy. If the patient reports hearing the tuning fork when it is placed on the ankle, leg or knee, suspect a so-called superior canal dehiscence.

Cranial nerves
Paresis of the cranial nerves (other than the VIIIth) together with dizziness indicates diseases of the brainstem, base of the skull, or the temporal bone.
A horizontal-rotatory spontaneous nystagmus observed without Frenzel’s glasses and suppressed by visual fixation suggests an acute unilateral peripheral vestibular lesion, which usually leads to rotatory vertigo and tendency to fall toward the affected side. If the nystagmus cannot be suppressed by visual fixation (so-called fixation nystagmus), this indicates a CNS lesion of the brainstem or cerebellum, or is, in some rare cases, congenital nystagmus. Gaze-evoked nystagmus indicates a so-called deficit of the neural integrator, caused by cerebellar or brainstem lesions but also by alcohol and drugs, such as anticonvulsants. A pure vertical or torsional nystagmus is caused by central brainstem or cerebellar lesions. The patient should be referred to a neurologist immediately.

The visual fixation suppression of the vestibulano-ocular reflex can be tested by asking the patient to extend his/her arm and continue to gaze on the thumb while rotating around on an office or examination chair. If the visual fixation suppression is impaired this indicates a central, most often cerebellar lesion but can also be caused by intoxication.
Examination of eye movements with Frenzel’s glasses. (Pictures D – G)

Frenzel’s glasses are used to detect a spontaneous nystagmus. An increased nystagmus with Frenzel’s glasses suggests a peripheral vestibular damage. In an ‘out of office situation’ when Frenzel’s may not be available, Dr Lars Ödkvist once suggested to use a ‘poor-man’s Frenzel’s’. For this, a sheet of paper is placed over the eyes of the patient, and the examiner looks from the side for any spontaneous nystagmus. You may also close the eyes and observe or touch the eyelids. Another way is to cover one eye and prevent fixation of the other eye by the examination lamp; this allows the examination of a spontaneous nystagmus.

A positional test with the Dix-Hallpike maneuver should be performed in every patient with vertigo to diagnose benign paroxysmal positional vertigo (BPPV), the most frequent cause of vertigo. This is characterized by a torsional-vertical nystagmus to the undermost ear with a crescendo-decrescendo time-course. The ear in question is usually on the side to which the patient turns to induce vertigo.

Positional nystagmus is observed when the patient is laid flat on his back with head turned to either side. Not only the presence but also the direction of the nystagmus is noted. A nystagmus in one of these positions, which does not fatigue, can be a sign of CNS damage, but it can also occur after consumption of alcohol (alcohol-induced positional nystagmus) or in the less common type of BPPV caused by cupulolithiasis, when the otoliths adhere to the cupula. A positional nystagmus in children should always raise suspicion of intracranial pathology.
In horizontal canal BPPV you will find a linear horizontal nystagmus. A nystagmus beating towards the floor is called geotropic nystagmus, caused by a canalolithiasis of a horizontal canal; a nystagmus beating towards the ceiling is called apogeotropic, caused by a cupulolithiasis of a horizontal canal.

Head-shaking test. The examiner holds the patient’s head between his hands and shakes it gently from side to side with a frequency of 2 Hz for 20 seconds. Patients generally become dizzy during this procedure. In a peripheral vestibular lesion you will find a horizontal-rotatory nystagmus beating toward the non-affected side. In central, in particular, cerebellar lesion you may find a so-called cross-coupling: horizontal head-shaking induces a vertical nystagmus (in some cases this may be seen in vestibular patients as well if the headshake is performed in supine but not sitting position. The head-shaking test should always be performed (Pictures H – I). It needs some practise, but it can be helpful for differentiating vestibular and CNS pathology, in particular posterior fossa problems. The test will be described later.

There are a number of other tests that can be performed, but those suggested above are the most useful in the majority of occasions and should be considered in each case.

**NOTE: A patient with vertigo not examined with the head-impulse test, positioning maneuvers or with and without visual fixation (Frenzel’s glasses/video goggles) has not been properly examined!**
Observation with video goggles of positional, spontaneous, and gaze nystagmus.

This is a very sensitive diagnostic tool as the eye movements are seen on the monitors.

Dix – Hallpike maneuver to the right

E

Turn the patient's head 45° to the side to be provoked, and slightly toward the back. Quickly lay the patient down. Maintain the patient's head in this position for at least 30 sec to check for nystagmus. If nystagmus appears after a short delay, increases in intensity, and then fades, this suggests BPPV.

Positional nystagmus, Head-shaking test

F

Flat on the back  Head Right - Left

Side to side movements for 10 – 20 seconds, then stop to check for the presence of nystagmus.
The video-impulse test can be performed for all semicircular canals. By letting the patients fixate an object on the side and at the same angle as the chosen canal plane, and performing the head impulse in the same plane, a small corrective saccade can be observed, if the VOR is decreased. Test the canal toward which the head impulse is performed.

Normal head-impulse test

Ask the patient to fixate the examiner's nose; then hold the patient's head between your hands and move it suddenly with a high acceleration 10–15° to the side. If the gaze is maintained, there is a normal VOR on the side of the provocation. Repeat several times. This tests the horizontal semicircular canals.
Stepping tests and Romberg’s test

A tendency to fall to either side suggests a postural disturbance. There can be several reasons for this. If the patient tends to fall forward and backward, this would be typical for a downbeat- or upbeat nystagmus, but in most patients an organic underlying pathology is less certain. During the stepping test the patient turns towards the damaged ear when there is inner ear pathology. NB! If you observe a spontaneous nystagmus and the patient falls in the direction of the fast phase of this nystagmus, i.e., toward the non-affected side, you should always suspect a cerebellar lesion.

The ’quick fix’: 7 QUESTIONS TO A PATIENT SEEKING ADVICE FOR DIZZINESS:

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. How long have you been dizzy?</td>
<td>Acute onset? Relapsing? Attacks?</td>
</tr>
<tr>
<td>2. Are you dizzy right now?</td>
<td>Findings will reflect function losses</td>
</tr>
<tr>
<td>3. Do you get dizzy when turning over in bed?</td>
<td>Suspect BPPV</td>
</tr>
<tr>
<td>4. Do you have or have you had migraine?</td>
<td>Suspect vestibular migraine</td>
</tr>
<tr>
<td>5. Are you only dizzy when walking or standing?</td>
<td>Suspect postural phobic vertigo or rarely bilateral vestibulopathy</td>
</tr>
<tr>
<td>6. Has your hearing deteriorated?</td>
<td>Suspect Menière’s disease cholesteatoma, vestibular schwannomas</td>
</tr>
<tr>
<td>7. Have you any pain in the ears, chest, or head?</td>
<td>You should take such signs as a serious warning and be very careful</td>
</tr>
</tbody>
</table>

If the VOR is significantly reduced, the eye movement follows the direction of the impulse. Then the eye refixates the target as in the picture on the right. A normal test does not rule out a slight reduction in the VOR. If the patient has acute vertigo with spontaneous nystagmus and a normal impulse test, this can indicate CNS pathology. Such patients should be immediately referred to the hospital for imaging and further assessment.
### SPONTANEOUS NYSTAGMUS

<table>
<thead>
<tr>
<th></th>
<th>Peripheral</th>
<th>‘Central’</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horizontal-rotatory</td>
<td>If spontaneous nystagmus</td>
<td>If fixation nystagmus</td>
<td>I.e central if fixation do not decrease the nystagmus intensity</td>
</tr>
<tr>
<td>Purely vertical</td>
<td>No</td>
<td>Yes</td>
<td>In most cases: a brainstem lesion</td>
</tr>
<tr>
<td>Purely torsional</td>
<td>No</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Horizontal and Torsional</td>
<td>Yes</td>
<td>Perhaps</td>
<td>Most probably peripheral</td>
</tr>
</tbody>
</table>

### GAZE NYSTAGMUS

<table>
<thead>
<tr>
<th></th>
<th>Peripheral</th>
<th>‘Central’</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gaze in extreme positions</td>
<td>–</td>
<td>–</td>
<td>Physiological</td>
</tr>
<tr>
<td>Gaze shifting within 30°</td>
<td>No</td>
<td>Yes</td>
<td>Due to brainstem or cerebellar lesions, drugs, alcohol</td>
</tr>
<tr>
<td>Vertical when gazing upwards / downwards</td>
<td>No</td>
<td>Yes</td>
<td>Indicates midbrain lesion (Nucleus interstitialis Cajal)</td>
</tr>
<tr>
<td>Increasing toward fast phase, decreasing toward the slow phase</td>
<td>Yes, typical if spontaneous</td>
<td>Maybe</td>
<td>This means: increased spontaneous nystagmus when looking into the direction of the quick-phase (according ot Alexander’s law)</td>
</tr>
</tbody>
</table>

### POSITONAL NYSTAGMUS

<table>
<thead>
<tr>
<th></th>
<th>Peripheral</th>
<th>‘Central’</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>With latency, peaking, and fading (especially in Dix-Hallpike)</td>
<td>Yes</td>
<td>Very rare</td>
<td>BPPV, if torsional-vertical and induced in the plane of the affected canal</td>
</tr>
<tr>
<td>On both sides</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Geotropic – lateral</td>
<td>Yes</td>
<td>Sometimes</td>
<td>Suspect lateral canal BPPV – canalolithiasis</td>
</tr>
<tr>
<td>Apogeotropic–lateral</td>
<td>Yes</td>
<td>Sometimes</td>
<td>Suspect lateral canal BPPV – cupulolithiasis</td>
</tr>
<tr>
<td>On one side only</td>
<td>Maybe</td>
<td>Yes</td>
<td>Can be both, common in schwannomas</td>
</tr>
</tbody>
</table>

**NOTE:** Positional nystagmus in children should always be suspected to be of central origin.
Figure 4: Vestibular Information
Head turnings in all planes.
Vestibulo-ocular reflex,
eye direction
Direction of gravity
Muscular tension in theback and legs.
A specific diagnosis is the first prerequisite for the therapy of vertigo, dizziness, and nystagmus. The various forms of vertigo are then treated with pharmacological therapy, physical therapy, psychotherapeutic measures or, rarely, surgery. If a patient receives pharmacotherapy, not only the correct diagnosis but also the drug administered, its dosage, and the duration of the treatment should be noted. This also means that there should be regular follow-up examinations, in particular to check if the dosage and the duration of the treatment are adequate.

PHARMACOTHERAPY OF VERTIGO, DIZZINESS, AND NYSTAGMUS

There are basically seven groups of drugs that can be used for pharmacotherapy: antiemetics; anti-inflammatory, anti-ménières, and anti-migraineous medications; anti-depressants, anti-convulsants, and aminopyridines (the “7 A’s”). Further, before treatment begins, the patient should also be told that the prognosis is generally good for two reasons: (a) many forms of vertigo have a favorable natural course (e.g., the peripheral vestibular function improves or central vestibular compensation of the vestibular tone imbalance takes place spontaneously) and (b) most forms can be successfully treated. In the following the current pharmacotherapy of the most frequent forms of vertigo and dizziness will be briefly summarized.

PERIPHERAL VESTIBULAR DISORDERS

Acute vestibular neuritis. A prospective randomized placebo-controlled study (on 141 patients) showed that methylprednisolone (beginning with 100 mg per days, tapering of dosage every third day by 20 mg) alone significantly improves the recovery of peripheral vestibular function in patients with vestibular neuritis, whereas valacyclovir does not. The combination of methylprednisolone and valacyclovir was not superior to steroid monotherapy.

Menière’s disease (MD). An open, non-masked trial, in which patients with MD received either a low dosage of betahistine-dihydrochloride (16 or 24 mg tid) or a higher dosage of 48 mg tid for at least 12 months was recently performed. The outcome measure was the number of attacks per month during a 3-month period. A total of 112 patients were included in the analysis: 50 received betahistine-dihydrochloride in a low dosage (16 mg tid,
Follow-up examination every 3 months showed that the number of attacks per month decreased in both groups over time. For instance, after 12 months the mean (median) number of attacks dropped from 7.6 (4.5) to 4.4 (2.0) \( (p<0.0001) \) in the low-dosage group, and from 8.8 (5.5) to 1.0 (0.0) \( (p<0.0001) \) in the high-dosage group. The number of attacks after 12 months was significantly lower in the high-dosage group than in the low-dosage group \( (p_{12M}=0.0002) \). The treatment was well tolerated in both groups. Despite the considerable limitations of an open, non-masked trial, particularly for MD, a higher dosage of beta-histine-dihydrochloride and a long-term treatment seem to be more effective than a low dosage and short-term treatment.

**Vestibular paroxysmia.** Vestibular paroxysmia is characterized by recurrent brief attacks of vertigo due to a neurovascular cross-compression of the vestibular nerve. In an open trial it was demonstrated that carbamazepine in a dosage of 400 to 600 mg per day significantly reduces the number of attacks of vestibular paroxysmia.

**CENTRAL VESTIBULAR, OCULAR AND CEREBELLAR DISORDERS AND NYSTAGMUS**

Recent trials showed that the potassium channel blockers 3,4-diaminopyridine and 4-aminopyridine (in well-tolerated dosages of 5 to 10 mg tid) are effective for the treatment of downbeat nystagmus and upbeat nystagmus. It was also demonstrated that the potassium channel blocker 4-aminopyridine (in well-tolerated dosages of 5 to 10 mg tid) is effective for the treatment of episodic ataxia type 2 (EA 2) (a hereditary disorder caused by mutations of the P/Q-calcium channel gene CACNA1a). These findings are the basis for an ongoing randomized controlled cross-over trial on the treatment of EA 2 with 4-aminopyridine. The effects of these potassium channel blockers can be attributed to an increase in the excitability of cerebellar Purkinje cells. Other pharmaceuticals increasingly being used are beta receptor blockers for vestibular migraine (for instance, 100 mg metoprolol per day) or selective serotonin re-uptake inhibitors for phobic postural vertigo. The latter, however, must still be proven in ongoing clinical trials.
The most common causes of vertigo and dizziness

The lifetime prevalence of vertigo and dizziness lies around 30% and the annual incidence increases with aging. Not only do many patients have vertigo and dizziness, but there is a multitude of disorders and conditions in which dizziness can occur. In a recently published overview of patients who consult their GP for the symptom of dizziness the cause of the problem was vestibular in 35 – 55%, psychiatric in 10 – 25%, and cerebrovascular in 5%. A diagnosis was made on the basis of a medical history and clinical examinations in 75% of the cases. Approximately 10% of the patients in the overview mentioned above never received a clear diagnosis.

As a rule of thumb, half of the patients with dizziness have a vestibular cause, ¼ have in one way or another a psychogenic/psychiatric cause, and ¼ have a mixed origin including neurological and circulatory disorders. There is a general agreement that the most common cause of acute vertigo and dizziness is BPPV. The other most common disorders are phobic postural phobic vertigo (related to: somatoform vertigo, chronic subjective dizziness, visual vertigo), central vestibular syndromes (ischemia, multiple sclerosis, neurodegenerative disorders, vestibular migraine, Menière's disease, vestibular neuritis). Multiple sensory deficits are common in the elderly, but if vertigo or dizziness is the leading symptom this may be caused by a vestibular dysfunction which has to be found out and the underlying cause identified.

BACKGROUND AND ETIOLOGY

Vestibular neuritis is characterized by a sudden unilateral loss of vestibular function (Fig. 4). Controversy surrounds the etiology of this condition. Today most evidence points to a reactivation of a latent herpes simplex virus type 1, which is thought to cause an edema of the nerve in its bony canal and in turn interrupt nerve conduction from the labyrinth. The superior branch of the vestibular nerve is believed to be most often affected. This nerve carries nerve fibers from the lateral and superior semicircular canals and the parts of the utricle. Symptoms arise from a sudden loss of the labyrinthine afference. The sparing of the inferior vestibular nerve and thereby the posterior canal explains why posterior canal BPPV can arise from a labyrinth that was thought to have lost its normal function due to vestibular neuritis.

The diagnosis of vestibular neuritis is a diagnosis of exclusion. Rarely similar symptoms are thought to arise in conjunction with vascular damage of the
brainstem or cerebellum, so-called vestibular pseudoneuritis. Infarction of the medial PICA (posterior inferior cerebellar artery) territory may result in only vertigo and dizziness. In such cases the head-impulse test is often normal in contrast to vestibular neuritis where it is pathological. As the labyrinthine artery to the inner ear originates most often from the AICA (anterior inferior cerebellar artery). Vascular damage in this area may have circulatory effects on the inner ear, including the cochlea. However, the latter patients generally develop some neurological symptoms as well. A minute infarction in the brainstem that is restricted to the root entry zone of the vestibular nerve may cause a sudden loss of vestibular function, mimicking a vestibular loss with pathological impulse test and with few, if any, discernible neurological findings. But such lesions are often benign in the short run.

**Epidemiology**

Vestibular neuritis is a relatively common disease with a lifetime prevalence of about 3.5 per 100,000. It is the third most common cause of peripheral vestibular vertigo and affects men and women equally. It occurs most frequently in patients in their younger years to middle age, but it can occur at any age.

**SYMPTOMS**

**ACUTE**

1. Acute onset of rotational vertigo leading to nausea and vomiting.
2. No reduction in hearing and no (new) tinnitus.
3. No other neurological symptoms.
4. Nystagmus is horizontal, with a torsional component. Nystagmus is more pronounced with gaze in the direction of the quick phase and is reduced with gaze in the opposite direction. The head-impulse test is pathological on the affected side. The head-shaking test increases nystagmus, however it may not be possible to perform in the acute stage as it strongly increases the nausea.
5. Balance problems in maintaining upright position. The patient is often unable to stand up without support and will usually fall in the direction of the slow phase, i.e. toward the affected ear.
6. When seen in the emergency room, these patients prefer to lie on their healthy side with eyes closed.
PROGRESSION

An acute onset of severe vertigo. Then gradually a reduction in symptoms. The first 24 – 48 hours are the worst, after which the patient can start moving more independently. The dizziness usually disappears gradually over the next 3 – 4 weeks. In approximately half of the cases the patient regains some vestibular function within the following 3 months; they are not usually troubled anymore with imbalance. In the other half vertigo is gradually reduced due to central compensation for the loss of function. It is important to instruct these patients to continue being active and to perform vestibular exercises and movements that trigger the dizzy spell (see later). However, since recovery cannot be predicted, all patients should be involved in early training and rehabilitation programs.

DIFFERENTIAL DIAGNOSIS

Cerebellar and brainstem infarctions

- May mimic vestibular neuritis,
- Patients with cerebellar lesions generally have a normal head-impulse test in spite of spontaneous nystagmus.
- Patients can have a tendency to fall in the direction of the fast phase of the nystagmus.
- Pathological dysdiadochokinesis,
- Gaze-shifting nystagmus,
- Loss of visual suppression can occur.

When these patients are admitted to the hospital, they are usually much better the next day in contrast to patients with vestibular neuritis, who are generally still dizzy. However, if the vestibular neuritis patients are successfully treated with steroids, they recover surprisingly fast as well.

MS

An MS plaque in the junction between the vestibular nerve and the brainstem can cause similar symptoms.
Management
A patient with vestibular neuritis should usually be hospitalized. They should also be referred to an ENT department/Medical Audiology or Neurology department. If a brainstem or cerebellar lesion is suspected, an MRI scan with diffusion weighted imaging, Doppler sonography and cardiovascular diagnostics should be done. An early CT scan can exclude a bleeding but may not detect an infarction.

TREATMENT

Acute phase
It has been demonstrated (Strupp et al. 2004, NEJM) that steroids in the acute phase may not only ameliorate symptoms but also reduce the vestibular loss.

The following regimen was used in that study:

| Acute, i.e., within the first three days after symptom onset | 100 mg methylprednisolone, taper dosage every third day by 20 mg or Betamethasone (see below) |

In the Ent DEPT IN Lund the following approach is used:

Betamethasone
As soon as possible 8 mg betamethasone (Betapred) or the equivalent is injected intravenously, because the patients may not be able to retain medicine given orally. After this the patient is started on 50 mg of prednisolone once daily for 5 days followed by a 5-day tapering-off period (40-30-20-10-5 mg).

NOTE: Betamethasone may be exchanged for equivalent doses of methylhydrocortisone or dexamethasone.

Day 1 – 2.
General supportive care. Prevent the patient from falling and sustaining damage. Medication for motion sickness can be tried, but anti-emetics have little effect. Suppositories or injections are usually very helpful. Don’t forget intravenous fluids if necessary.
**Sub-acute phase**

Day 2 – 10.
Start the rehabilitation with provocations or vestibular rehab exercises as soon as possible (see appendix, page 76). It is often helpful to involve the physiotherapist in this phase of the disease. It is also important to avoid sedatives and medication for motion sickness, which would reduce the effect of the habituation therapy.

**Compensation phase**

From Day 10 and onwards
The patient is supposed to do more and more advanced exercises, which involve triggering sensory conflict in the balance system. These exercises are to coordinate the input from vision, proprioception, and vestibular function.

**Work and sick leave**

If the steroid treatment does not reverse the vestibular loss immediately, 100% sick leave is recommended for 3 – 4 weeks; thereafter it is reduced to 50%, depending on the type of work. The older the patient, the longer the period of rehabilitation needed. Patients who start to work too early often have a continuous sensation of unsteadiness to such an extent that they are not capable of doing their job properly, resulting in further sick leave. It is therefore thought that 50% sick leave the first week after returning to work is important.

For special categories of work there may be a need for longer sick leave (machine operators, pilots, chauffeurs, chimney sweepers, construction workers, etc.). A long period of sick leave combined with inactivity are, however, contraindicated and can, in actual fact, reduce the rehabilitation and prolong the problems.

“Resting will never cure vertigo”.

Inform the patient that some patients may experience increased fatigue and tiredness for a prolonged period after the acute phase (up to 6 – 9 months).

**Associated Diseases**

It is not uncommon that BPPV develops after vestibular neuritis: about 15% will develop post vestibular neuritis BPPV.

A number of patients develop so-called phobic postural vertigo. This is particularly common in patients who have been inactive in the immediate period after the acute phase.
**BACKGROUND**

This is the most common type of vertigo. BPPV is most frequently triggered by pathology in the posterior semicircular canal. The diagnosis is straightforward with the Dix-Hallpike and lateral canal tests (Fig. E). It is important to remember that the patient suffers significantly and that the disease is treatable.

**ETIOLOGY**

Detritus, made up of otoliths or degenerative products of these otoliths, is collected in the posterior semicircular canal (Fig. 5). When the patient is lying or turns so that the affected side is down, or bends the neck backwards and upwards, this mass of detritus sinks slowly under the influence of gravity. Material then collects in the undermost position of the canal, dragging the endolymph in the same direction and leading to a deflection of the hair cells of the cupula. When the otolithic mass settles, the symptoms subside. When the head is moved in the opposite direction, the particles also shift in the opposite direction to induce a nystagmus in the reverse direction. The latency of the development of nystagmus may be attributed to the time taken for the particles to be displaced and also to adhere to the membranous wall of the labyrinth. Adherence may also explain why a brisk head movement is needed to induce the vertigo. The hair cells of the cupula return to their resting position and the dizziness subsides when the detritus settles. This produces dizziness with a delayed onset of a few seconds and with a sudden intensity. Generally within 30 seconds it gradually subsides.

Agglomerated masses can also settle in the horizontal and superior semicircular canals. For the lateral canal the nystagmus will be lateral and beat towards the floor independently of which ear is undermost. Movements that trigger the vertigo are somewhat different. The duration and fatigability of the symptoms are, however, similar. In the case of anterior canal cupulolithiasis when tested with a Dix-Hallpike position the nystagmus beats toward the lower eyelid on the opposite side of the affected canal. In a canalolithiasis of the horizontal canal there is a horizontal geotropic nystagmus, in the more rare form of a cupulolithiasis of the horizontal canal the nystagmus beat ageotropic.
Another variation of this pathology is that the otoliths adhere to the cupula, i.e., *cupulolithiasis of the semicircular canal*. The symptoms will then be slightly different and the nystagmus continuous. This could be a differential diagnosis for positional vertigo/nystagmus triggered by diseases in the posterior fossa.

The most frequent causes are head trauma, vestibular neuritis, Menière’s disease and vestibular migraine but the vast majority of cases are cryptogenic. Long bed rest, or dental work have also been reported to evoke BPPV.

**EPIDEMIOLOGY**

Benign positional vertigo is the most common cause of dizziness with a lifetime prevalence of at least 10%. It occurs at all ages and equally in males and females. In fact, it seems to be even more common among the elderly. In children it may occur after a head trauma. It is rarely seen in children.

**SYMPTOMS**

Typical BPPV is characterized by short attacks of vertigo. Head movements such as looking up, lying down, or turning over in bed precipitate the attacks. The patients may also experience a slight unsteadiness when moving around or walking.
Findings – Canalolithiasis

**Posterior canal BPPV:**
The diagnosis is confirmed by the Dix-Hallpike positional test. It shows a characteristic torsional nystagmus to the undermost ear or a nystagmus beating towards the upper eyelid when the head is reclined and turned to the affected side. The difference in direction of the nystagmus depends only on the direction of gaze relative to the semicircular canal. Sitting up results in most cases in a nystagmus in the opposite direction. The vertigo and the nystagmus usually last less than 30 seconds and never more than 60 seconds.

Repetitive maneuvers reduce both the vertigo and the nystagmus, which usually disappears after 2 – 3 maneuvers. A normal neurological status, normal hearing, and no spontaneous or gaze-evoked nystagmus are observed in the sitting position with or without Frenzel's glasses.

**Horizontal canal BPPV:**
The patient lies supine with the head lifted about 30°. When the head is turned to the side, the nystagmus beats toward the floor; this is a geotropic nystagmus that occurs with a latency, peaks, and fades as in posterior canal BPPV. Turning the head to the contralateral side also evokes a geotropic nystagmus. The side with the BPPV is the one with the most vivid nystagmus.

**Anterior canal BPPV:**
A Dix-Hallpike maneuver towards the healthy side evokes an inverted response compared to a maneuver to the posterior canal. That is, the nystagmus beats towards the lower lid. This type is the least common.

Findings – Cupulolithiasis

**Lateral canal cupulolithiasis:**
The patient lies supine with head lifted about 30°. When the head is turned to the side, there is an apogeotropic nystagmus, i.e., it beats toward the ceiling. There is no latency and no fatiguing. The nystagmus will be somewhat stronger when the head is turned toward the healthy ear. This is the most common form of cupulolithiasis.

**Anterior canal cupulolithiasis:**
A Dix-Hallpike maneuver towards the healthy side evokes the opposite response compared to that toward the posterior canal. The nystagmus generally beats towards the lower lid. It occurs without latency and without fatigue.
**Posterior canal cupulolithiasis:**
The Dix-Hallpike maneuver evokes a nystagmus like that in canalolithiasis but without latency and without fatiguing. This type is uncommon.

<table>
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<td>Posterior cupulolithiasis</td>
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<tr>
<td>Lateral cupulolithiasis</td>
<td>Turning head to side at 30° elevation</td>
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<tr>
<td>Anterior cupulolithiasis</td>
<td>Dix-Hallpike</td>
<td>As in canalolithiasis but weaker and without termination</td>
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**PROGRESSION**

BPPV may have a relatively sudden onset. A significant number of patients will be spontaneously better within 6 – 12 weeks (45 – 70%). Recurrences of the symptoms occur in about 50% of the patients: If the maneuvers are performed correctly no chronic BPPV will develop. If not treated, however some patients may continue having symptoms.

**TREATMENT**

Posterior canal BPPV:
Produce movements that aim to loosen the otolith /debris in the affected semicircular canal.
Figures 6:
1. Habituation movements according to modified Brandt-Daroff

2. Semont’s maneuver, which is for all practical purposes a Dix-Hallpike maneuver performed on the affected side followed by quickly moving the patient to the opposite side.

3. Epley’s or Parnes, and other maneuvers (‘particle repositioning’ or ‘liberatory’ maneuver), again like a Dix-Hallpike maneuver towards the suspected side, thereafter a slow rotation to the opposite side (see pictures J and K).
The three suggested methods are approximately equivalent in effectiveness if they are performed correctly. About 90% success can be expected after one manipulation. Maneuvers should be performed as soon as the diagnosis is made to cure the patient. Treatments with section of the vestibular nerve, labyrinth destruction, or obliteration of the posterior semicircular canal are no longer considered appropriate treatments for this disease.

1. The patient is kept still for about 1 min after The Dix-Hallpike maneuver.

2. The patient’s head is moved in the other direction.

3. The patient then rolls over onto the side, while the head is fixed in its position.
4. Turn the head further until the nose points to the floor.

5. The patient is then helped to sit up, while the head is supported in a fixed position. If the patient has a sensation of vertigo or nystagmus reoccurs, the procedure is repeated.
Left ear lateral canalolithiasis (Gufoni/Appiani), Lay patient down on healthy ear; turn head fast, so that nose is down; Leave patient in this position for at least 2 minutes. (5 – 30 min is OK)

Left ear lateral canalolithiasis (Gufoni/Appiani), cont’d. Raise patient; return head rather slowly to normal position.
Lateral canal BPPV:
Test with head slightly raised – by 30° – with the horizontal canal in the earth vertical axis (fig F). Then the Barbecue Maneuver in which the patient is rotated by 360° can be used. As an alternative, place the patient on a couch or a bed and perform the Apiani/Gufoni maneuver (see Fig. on page 47). The latter maneuver is in many case swifter, more comfortable for the patient, and takes less time for the physician to perform.

Cupulolithiasis
The treatment needs to be in two steps:
- a) mobilize the otoliths to move from the cupula;
- b) treat like a canalolithiasis.

The first step may be achieved by strong head shaking (as demonstrated by Magnusson et al. 2002) or by prolonged postioning. This is generally done on the healthy side as for canalolithiasis. Repeated training programs may help the patient. Sometimes the training induces such pronounced nausea that it may be a good idea to supply the patient with a vestibular depressant before the exercises.
BACKGROUND AND ETIOLOGY

The etiology of Menière’s disease is not fully understood. There is a general agreement that it is associated with an increase in the volume and pressure in the endolymphatic space. It is assumed that this ‘hydrops’ leads to reduced function of the vestibular apparatus and damage to the outer hair cells of the cochlea, which causes hearing impairment and tinnitus.

The sudden attacks of vertigo are presumed to be related either to a rupture or increased permeability of the membranes containing the endolymphatic fluid or to a sudden fall in endolymphatic pressure caused by an evacuation of fluids through the endolymphatic duct. How this hydrops starts or whether it is the cause or the result of Menière’s disease is still not known.

Menière’s disease is characterized by the Menière’s triad: reduced hearing, vertigo, and tinnitus. The diagnosis is based on the presence of this triad.

DIFFERENTIAL DIAGNOSIS

- Lesion in the posterior fossa, central positional vertigo. These patients have continuous vertigo and nystagmus when lying on one side.
- “Pseudo BPPV” can be seen in patients with tumors like meningiomas of the posterior Fossa or MS; similar findings as described above.
- Alcohol-induced nystagmus also provokes a position-related nystagmus and vertigo. However, it usually has no torsional component.

NB: a positionally provoked nystagmus in children should suggest a lesion in the posterior fossa and should be considered as such, until ruled out.

HISTORY

The patients often describe an initial sensation of fullness or pressure in the affected ear. They sometimes see their physician assuming they have obstructing ear wax (cerumen).

At the same time patients have reduced hearing, distortion (diplosis) and oversensitivity to sound, and tinnitus. Thereafter the patient may experience a sudden spell of rotatory vertigo, which often leads to nausea and vomiting.
During an attack, the patient is usually unable to move and often seeks advice the day after or later. Early in the disease, hearing is usually restored after the attacks, but the patient will gradually develop permanent unilateral hearing loss with a variable degree of tinnitus.

**SYMPTOMS**

If the patient is seen during an attack, spontaneous nystagmus is observed. Early in the attack the quick phase is towards the affected side; later it changes direction towards the good side. For this reason the direction of spontaneous nystagmus in acute Menière’s disease may be confusing. Nevertheless, a reduction of hearing in one ear followed later by acute vertigo and nystagmus beating toward the same ear strongly suggests Menière’s disease.

Head movements enhance nausea and vomiting. The patient reports unilateral reduced hearing. The sensory neural hearing loss is ascertained with a tuning fork (Weber) or the ‘hum’ tests (the Weber test is lateralized to the better hearing ear and the Rinne test is positive; patients report hearing the tone stronger in the better hearing ear when they make a continuous tone, i.e. when humming). The patient has normal neurological and cardiopulmonary status and normal blood pressure. The attacks usually last for a minimum of one hour and rarely more than 8 hours (attacks must last at least 20 minutes). If the attacks are shorter or last longer, the diagnosis must be questioned and vestibular migraine be considered.

If the patient is seen between attacks, the neurological, and ENT status are usually normal. In the early stages of the disease the patient may also have normal hearing in both ears and no tinnitus. Later, after several attacks, there is usually a unilateral sensorineural hearing loss on the affected side. When the patient is examined with Frenzel’s glasses, a head-shaking test may provoke nystagmus, the direction of which may depend on the stage of the disease: irritation (affected side), non-compensated loss (to the healthy side), no nystagmus (compensated loss), nystagmus to the affected side (uncompensated return of function).

Patients with Menière’s disease have periods/clusters of attacks. It is common that the intervals between attacks last a couple of months to years. The course of the disease is very individual and is difficult to predict. Involvement of the
other ear with bilateral Menière's disease has a tendency to increase, the longer the patient has the disease.

All of these patients should be seen by a specialist.

DIFFERENTIAL DIAGNOSIS

Acute vertigo, as occurs in Menière's disease, can also be related to vestibular migraine. Hearing impairment can be present, but if the hearing is normal the likelihood of vestibular migraine is a greater possibility. Cerebello-pontine angle tumors can cause hearing impairment suggestive of Menière's disease, but they do not usually induce vertigo attacks. Autoimmune diseases, acute lues, or acute labyrinthitis are rarely seen.

MANAGEMENT

1) The Acute Vertigo Attack

The patients cannot usually care for themselves during this phase. It is therefore important to reduce the sensation of vertigo and to exclude the possibility of any other serious disease (if it is the first attack, see Acute Vertigo).

Status:

- Level of consciousness
- General neurological examination
- Blood pressure, heart examination
- Check eyes with Frenzel's glasses
- Check the eardrums
- If the diagnosis is known or confirmed, the patient should be treated with anti-emetic or anti-histamine preparations.
- The best way to administer medication is usually by suppositories or injections.

2) If the patient seeks advice between attacks

Determine the hearing loss. If there is unilateral hearing impairment, the patient needs to be assessed by an ENT specialist for a full audiological / otoneurological examination.
Re-read the history.
Check ENT status.
Check eye movements with Frenzel’s glasses, in particular spontaneous or gaze-evoked nystagmus.
Look for nystagmus after head shaking.
Perform head-impulse test.
Check neurological status.
Check hearing, if possible with audiometry including speech discrimination.

3) **Patient with a history of frequently remitting dizzy spells examined between attacks**

Repeat steps under point 2 and refer to an ENT department for further management

### Treatment options

1. Acute treatment. See point 1.

2. The patient has a history but infrequent attacks. Examine the patient and inform of possible diagnosis.

3. If the patient has a recurrent attack, see below.

A Betahistine. Betahistine is an H1 agonist and H3 antagonist. It improves the microcirculation by acting on the precapillary sphincters of the stria vascularis. There is evidence that it reduces the production and increases the absorption of endolymph. In an open trial on 112 patients with Menière’s disease it was shown that a higher dosage of betahistine-dihydrochloride (48 mg tid) and a long-term treatment (12 months) seems to be more effective than a low dosage (16 to 24 mg tid) and short-term treatment. Therefore, nowadays a long-term prophylactic treatment (>12 months) with betahistine in a dosage of at least 48 mg tid, i.e., 144 mg per days is recommended. Recently patients have been treated with dosages of up to 480 mg per day.

These data are the basis for a recently begun prospective, randomized, double-blind dose-finding study comparing placebo with 16 mg did and 48 mg tid betahistine-dihydrochloride. It must, however, be pointed out
that up to now no state-of-the-art studies have been conducted in this field despite the large number of trials.

B. Diuretics for possible endolymphatic hydrops. Although there is no good evidence from meta-analyses that diuretics have a real benefit in Menière's disease, it is still in use. This treatment should usually be given in periods of 3 – 4 months, after which the dosage should be reduced if the patient has no further attacks. Sodium-potassium/serum controls after 14 days and thereafter as indicated. Daily thiazide diuretics and if contraindicated 60 mg furosemide in slow-release preparations may be tried.

C. Medication during the attack. The patients will feel better, if acute treatment is made available at home. Antihistamines, phenothiazines, and related drugs can be given by injection or as suppositories. It is important to be aware of the extrapyramidal side effects of this treatment in children and the elderly.

D. All patients benefit from anxiolytics and muscle relaxants given for 1 – 2 weeks if they have a difficult period.

E. ‘Psycho-supportive therapy’. Information about the disease is given to the patient.

F. Salt restriction. There is no good evidence from meta-analyses that salt restriction has any effect on Menière's disease. Although reduced salt intake to less than 1-1.5 g/day is often suggested. Sometimes the help of a dietician may be needed.

G. Vestibular rehabilitation exercises between the acute attacks will speed up the compensatory phase and permit the patient to differentiate between Menière's attacks and subsequent dizziness due to non-compensated vestibular mismatch.

**Further Treatments**

If the patient has frequent vertiginous attacks, it may be necessary to reduce or destroy the function of the inner ear on the affected side. Secondary treatment for the muscular tension, which leads to unsteadiness between the attacks, is also helpful. The treatment is aimed at the patient's vertigo attacks. There is no proven effective treatment for progressive hearing loss, but tinnitus can be treated in the usual manner with neuropsychological management.
Gentamicin injection

Gentamicin is an ototoxic drug that is injected into the middle ear cleft. This causes a reduced or total loss of function in some of the nerve cells of the inner ear. As the sensory cells in the vestibular organ are more sensitive than those in the hearing organ, the vestibular function can be sufficiently reduced and the symptoms stopped without affecting the hearing in most cases.

There is, however, always a risk of hearing impairment after this treatment, which is technically simple and cheap.

Saccus operation (Shunt operation)

Surgery of the endolymphatic sac. The surgeon tries to reduce the endolymphatic hydrops/increased pressure by different procedures involving the endolymphatic sac. Double-blind studies have shown this procedure to be doubtful, but nevertheless, it is still used with varying success.

Steroids

Intra-tympanic steroids are increasingly gaining popularity. However, there is no consensus about doses, drugs, or treatment repetition. In our experience daily repeated installments of dexamethasone or betamethasone for 4 – 5 days can induce at least a temporary remission of attacks. Other used regimes are 2 installments per day twice a week for 2-3 weeks. Different drugs has been used. Most often dexamethasone is favoured although it recently has been shown that methylhydrocortisone may remain in the endolymph for a longer period. However, due to it’s acidity and low pH, the latter might be painful for the patient.

Vestibular nerve section

After intracranial nerve section, the impulses from the inner ear no longer reach the brainstem and thus the attacks of vertigo are prevented. There is, however, a risk of affecting the cranial nerves of the pontine angle adjacent to the vestibular nerve. Hence, side effects may be hearing impairment or complete loss of hearing and facial palsy, as well as other intra cranial complications.

Labyrinthectomy – Destruction of the inner ear

Surgical labyrinthectomy means that the labyrinth is surgically destroyed. Total deafness on the operated side is the result.

Nowadays we do neither recommend vestibular nerve section nor labyrinthectomy in Menière’s disease in particular since the long-term treatment
There is an ongoing debate about cervicogenic dizziness. Several authors (including one co-author of this booklet, M.S.) doubt that cervicogenic dizziness even exists. Pain and discomfort from the neck and shoulders are very common, as is dizziness. It is therefore not surprising if these symptoms coincidentally occur at the same time. The eyes and the balance organs of the inner ears are anatomically located in the base of the skull. They play an important role in both orientation and the balance reflexes. Their position in relation to the rest of the body has to be defined, in order for them to functionally maintain the body's balance during standing and walking. Information about the head's position in relation to the rest of the body, in other words the movement and position of the neck, is therefore crucial. Doubts have been expressed that symptoms or disturbances in neck proprioception play a part in the triggering of unsteadiness and balance disturbances. In support of these doubts it is argued that, for example, patients with acute torticollis have no specific vertigo or balance disturbance. However, it has been shown, both experimentally and in clinical studies, that a disturbance of neck proprioception can lead to an impairment in the control of balance during standing, which creates a sensation of vertigo.

Even more treatments

A number of other treatments for Menière's disease have been tried, e.g., local or general pressure treatments, acupuncture interventions on the head and neck muscles, and different diets. No documented independent studies support most of the beneficial claims.

Currently, investigations are in progress to determine whether local pressure treatments (Meniette) can reduce the frequency of vertigo attacks. Finally, it is important to stress that any study of Menière's disease is always difficult to conduct due to the intermittent attacks and also the very high frequency of spontaneous remission and recurrences.

Cervical origin of dizziness

with a high dosage of Betahistine (48 mg tid) seems to be very effective and in case of incapacitating situations, transtympanic treatments seems to be sufficient in most cases.

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Treatment of the painful neck with simultaneous vertigo reduces both symptoms. There are close links, both physiologically and anatomically, between the balance organs of the inner ears and groups of neck muscles. The doctor who makes the diagnosis of cervicogenic dizziness must therefore be fully aware that pain and tension in the neck along with the vertigo can be a result of a primary problem from the neck or primary disease of the inner ear as well as the CNS. In other words, tension in the neck can trigger vertigo, but disease of the inner ear or the brain that provokes dizziness can secondarily lead to tension in the neck just as stress and psychological problems can. No reliable diagnostic tests have yet been developed for cervicogenic dizziness. So far the best way to be sure that the dizziness is really of cervicogenic origin is that physiotherapy of the neck brings the patient relief from dizziness.

**MEDICAL HISTORY**

- The patients usually describe the appearance of neck pain/problems before the onset of the dizziness
- There are usually no neurological symptoms. Possible loss of sensitivity or reduced strength in the arms could be related to a cervical disc herniation.
- The dizziness is usually described as nautical unsteadiness.
- This sensation can be very strong and even lead to nausea periodically, although less commonly.
- This kind of dizziness often gets worse after the initial treatment by the physiotherapist.

**FINDINGS**

- Normal ear, nose, and throat and neurological examinations.
- Normal cardiovascular findings
- Examination of the eyes with Frenzel’s glasses does not reveal any spontaneous or directional nystagmus, nor is any nystagmus found on the head-shaking test.
- No hearing impairment
Manual examination of the neck muscles, in particular the sterno-cleido-mastoid muscle. The midline neck muscles, the muscle insertions in the occiput, and trapezius edges are usually sore and tender (“tender points”). The patient may show “jump signs”, which means that he/she reacts with severe pain and usually jerks on palpation of a tender point.

MANAGEMENT

A neck problem in combination with dizziness does not necessarily mean that the dizziness is caused by the neck problem. It is therefore important to exclude the possibility of any serious neurological or inner ear diseases before treatment of the neck problem. A follow-up of these patients is important in order to make sure that they have improved, not only from the neck pain but also from their balance problems.

- Anti-inflammatory and eventual muscle-relaxing preparations for a short period of 2 – 3 weeks with benzodiazepines may be helpful.
- Encourage physical activity, particularly in the younger patient.
- Physiotherapy

Figure 7:
Dizziness in the older patient

**BACKGROUND**

An older patient can suffer from the same vertiginous diseases as a younger patient. In fact, according to a study of ‘senior citizens with an urban lifestyle’ 9% had a positive Dix-Hallpike test, indicating BPPV. Furthermore, several studies have reported that up to a third of senior citizens seeking advice for dizziness have BPPV (Katsarkis et al 1994). In addition, the frequency of depression and anxiety increases among the elderly, and disorientation is one of its symptoms.

The older patient is also prone to dizziness and has mobility problems due to ‘multiple sensory system failure’. This includes the effects of ageing on all of the sensory systems, information management of orientation and balance reflexes in the CNS, muscular strength, and joint movements (Fig. 8). Moreover, the frequency of vascular and metabolic diseases also increases in older patients. All of this leads to a further reduction in information about movement and control of balance while standing and while walking. Finally, many older subjects receive often many drugs and combinations of drugs which can cause dizziness and postural imbalance.

Nevertheless, in most older patients you can identify the two most important vestibular causes of their symptoms, i.e., BPPV or bilateral vestibulopathy by a careful patient history and clinical bedside examination.

Multiple-system failure can be shown to be a reduction in the function of:

**Vision**
Cataract and retinal degeneration, with reduced detection of movements.

**Balance organs in the inner ear**
The vestibular system in the inner ear, vestibular nerve, and the balance nuclei all show signs of age-related cell loss. This is similar to the findings in patients with presbyacusis. Whereas a side difference in hearing may play a minor role for the patient, a side difference in the vestibular function significantly disturbs balance control. Recent studies suggest that this vestibular asymmetry can be common in otherwise healthy older people. In conclusion, the vestibular information about the position of the head and its movements is reduced with increasing age.
**Proprioception**

Decreased proprioception can be caused by age-related polyneuropathy, B12 insufficiency, diabetes, and circulatory problems. This leads to a reduction in information about the movement of different body segments in relation to each other.

**Sensation of touch**

The mechanoreceptors in the sole of the foot and other pressure-sustaining skin surfaces are also exposed to age-related degeneration, as is the skin in general.

Polyneuropathy also leads to a reduction in the information transmitted to the CNS.
**CNS**
Age-related degeneration, circulatory disturbances, cognitive disturbances, and increased frequency of neurodegenerative diseases occur. This results in a general reduction in the information management of the central nervous system.

**Reduced muscular bulk – ‘Sarcopenia’**
Since information and information management are reduced in the elderly, they are even more reliant on sufficient muscular bulk in relation to their total body mass to help prevent a fall or to control movements. However, with increasing age the muscular bulk is often reduced, in part due to ageing but also due to reduced physical activity.

The degeneration of all functions in the balance maintenance system in the elderly leads in part to less and slower recognition of the movements they produce or are exposed to and in part to reduced strength to prevent a fall.

**HISTORY AND SYMPTOMS**
Patients often have a history of falls or a fear of falling. It has been suggested that patients who have to stop walking when talking are at increased risk of having accidents with falls and fractures in the near future.

The physical examination does not normally detect any symptoms of acute vestibular or neurological disturbance. Only a further examination often finds that the patient has age-related degenerative changes. For example, vestibular asymmetry has been found many times in this population after head shaking with Frenzel’s glasses.

**EXAMINATIONS AND TREATMENT**
For patients with multiple-system function impairment it is important to examine and later optimize the different body functions.

- Circulation, arrhythmia, blood pressure, hypertonia, hypotonia, orthostatic blood pressure, and anemia should be examined and treated.
Polyneuropathy, reduced deep vibration with no reflexes, needs further examinations to determine B12 insufficiency, peripheral circulatory disturbance, diabetes, neuropathy, etc.

CNS disturbances: Signs of vascular or degenerative CNS disturbances need to be explored. Many older people commonly take a significant amount of sedatives, which impair their attention and balance.

Visual function. Assess or refer patients to specialists for possible cataracts or macular-degeneration. Patients with these problems need to have well-lit homes.

Vestibular disturbances. Do a full oto-neurological examination with Frenzel's glasses. A hearing test indicating presbyacusis may suggest that there is also an inner ear degeneration. Patients with signs of decreased vestibular function should generally be put on a retraining program and increase their physical activity.

Proprioception. Test the deep vibration and the peripheral reflexes. Check B12, blood glucose, and peripheral circulation.

Examination of the sole of the foot. Recommend that the patient wear supportive but lightweight shoes for maximum support and sensory transmission.

Psychological state. Depression and anxiety are common in the older population. At signs of depression it is important to intervene or to refer the patient to a specialist. However, note that vertigo is a side effect of some anti-depressive medication.

Medication. The more medicine the patient takes, the higher the risk of drug effects on blood pressure and wakefulness. Painkillers with opiate-like substances may influence the blood pressure as well as pain itself.

Vertigo in the older patient, who has no significant symptoms from any particular organ system, is often caused by a multiple-system reduction. However, even these patients can be successfully treated, and their symptoms reduced and their quality of life improved.
**EXPLORE**

Define function and loss of function in

**THINK OF**

- Vision
- Vestibular system in the inner ear
- Proprioception/pressure sensors in the sole of the foot
- Circulation
- Hyper-, hypotonia/arrhythmia, anemia, heart failure
- Respiratory function
- Muscular bulk, muscular function
- Psyche/cognitive function

**Explain**

Explain the link between multisensory deficit, reduced muscle power, and CNS function to the patient and his/her family.

**Optimise**

Intervene to optimize vital functions according to the above. This often means checking and hopefully reducing prescribed medication.

**Activate**

Motivate the patient to become more active. No patient gets better balance by resting and inactivity. Less activity means a reduced amount of muscles and also reduced control function. Use it or lose it! In fact, dizzy patients who do light exercises, such as taking a walk, feel better than those who do not.
Phobic postural vertigo

Related to visual vertigo and chronic subjective dizziness (‘tension dizziness’, ‘functional dizziness’, or ‘psychogenic vertigo’)

BACKGROUND

Phobic postural vertigo is relatively common among both sexes. The complaint usually includes unsteadiness and a significant sensation of unease, possibly associated with fear, which then leads to fear of moving and a gradual reduction in mobility. Some patients describe a sensation resembling that of a fear of heights, even though they are on the ground.

The symptoms usually appear in the aftermath of other balance problems such as BPPV or vestibular neuritis, particularly if the patient has not been properly informed or involved in a rehabilitation program. As a result both mobility and compensation are less.

The physiological mechanism behind this state is possibly due to the fact that we do not normally perceive our own movements. When we initiate a movement by coordinating the involvement of several central nervous structures, this information also reaches similar sensory centers simultaneously. This means that the return information coming from the different sensory systems (the inner ear, vision, and proprioception) when we move, does not lead to a conscious sensation of movement. At least this does not lead to a sensation similar to that of slipping or falling or moving involuntarily by other means.

In patients with phobic postural vertigo it is believed that the sensory information triggered by the body’s own movements is not filtered out. Instead the information is allowed to reach consciousness, causing the voluntary movements to be experienced like externally induced movements. This leads to dizziness when we move.

It has also been suggested that the patient distrusts proprioceptive and vestibular cues and relies more on, or is sensitized to, visual information – visual vertigo. The symptoms can be seriously incapacitating and often lead to the patient’s fearfulness of leaving his/her own home. Nevertheless, no pathology is found on a thorough examination.
PROGRESS
These symptoms often start after the patient has another disease related to balance such as vestibular neuritis or BPPV. They can also begin after motion sickness, other long-lasting somatic diseases, or psychological strain. The symptoms usually become gradually worse and more pronounced. Spontaneous remission may occur, but the few investigations involving this population show that the symptoms often continue or even become worse.

HISTORY
- The patient often feels unsteady and has a feeling of moving while standing still. There are usually no problems when lying down or sitting still.
- The symptoms are usually related to a significant sensation of unease, eventually producing an anxiety attack, which then leads to a fear to move.
- Avoidance behavior. The patient avoids more and more any kind of movement, particularly standing or walking.
- The patient avoids walking in open spaces or in crowds. Movements in rich visual surroundings make the symptoms worse.
- Sedatives and alcohol may temporarily relieve or ameliorate the symptoms.
- No finding or history of any other serious medical condition.
- The symptoms are thought to be more common in patients who have a compulsive and controlling personality.

SYMPTOMS/PHYSICAL EXAMINATION
Completely normal findings for ear, nose, throat, circulatory, neurological, and eye movement examinations.

EXAMINATIONS
These patients usually demand a thorough examination. They can only be influenced by information and rehabilitation. This entails vestibular testing with calorics and a full neurological examination.
TREATMENT

In the less severe cases home training and reactivation programs may suffice. In more severe cases a course of anti-depressive medication for 9 – 12 months or longer; initially a combination with diazepam or a similar drug, for 2 – 3 weeks, may be necessary. Simultaneously it is important to start a structured rehabilitation program with emphasis on gait and vestibular retraining. The patient needs to be gradually exposed to more and more trigger situations. In addition, it is important to have a psychologist support the patients through relaxation/stress therapy or even cognitive therapy. In severe cases all of these elements have to be done simultaneously to achieve a good end result.

BACKGROUND

Labyrinthitis is an infection of the inner ear. Usually it starts with a middle ear infection that spreads to the inner ear or, in some cases, begins with meningitis and then leads to an infection spreading to the inner ear from the intracranial space. This causes reduced function of the inner ear with reduced hearing and balance function. As this is a relatively acute disease, it provokes vertigo and usually nystagmus. If labyrinthitis is not treated, it damages the inner ear permanently. If the cause of this infection is otitis, then there is a substantial risk of intracranial spread. Otogenic meningitis usually takes a very rapid and life-threatening course.

HISTORY AND SYMPTOMS

- Pain in the ear and increasing unsteadiness leading to rotatory vertigo.
- Acute otitis media with a purulent discharge from the middle ear.
- Nystagmus: Initially only observed with Frenzel's glasses and after head shaking.
- It is important to describe the quick phase of the nystagmus.
  - Quick phase towards the infected ear: irritation nystagmus
  - Quick phase towards the good ear: reduced function/destruction. This indicates that the infection has developed further and caused reduced function of the inner ear and may be developing to a meningitis.
EXAMINATION AND TREATMENT

Acute otitis and vertigo should always suggest the possibility of labyrinthitis. Check for alertness and stiffness of the neck. Contact the nearest ENT department or immediately transfer the patient to the nearest hospital.

BACKGROUND

Most patients fear that the dizziness they experience is a sign of a brain tumor even though a brain tumor is rarely a cause of dizziness. However, space-occupying processes intracranially can lead to vertigo by various mechanisms.

The examining doctors should be guided by other neurological signs that suggest such pathology. Isolated vertigo is a rare sign of intracranial tumors. Nevertheless, it is important to rule out tumors in the posterior fossa and in particular in the cerebellopontine angle. Neuromas from the Vth, VIIth and VIIIth cranial nerves can be found in this region as well as meningiomas from the tentorium or associated with the pontine angle. Even less common are space-occupying processes like metastases or cholesteatoma.

The most common tumor is the acoustic neuroma (schwannoma). This benign tumor develops from the Schwann cells surrounding the vestibular nerve. The tumor does not spread, but its growth may damage or exert pressure on surrounding tissue in the posterior fossa. If radically removed, it does not recur. A special case is neurofibromatosis type II, where there may be bilateral neuromas. These patients may also have neuromas of other cranial nerves or spinal roots, specifically of the sensory nerve and roots. The younger the patient with a vestibular schwannoma, the more likely it is that they have neurofibromatosis type II. Patients with neurofibromatosis type I (von Recklinghausen’s disease) are at no greater risk of developing vestibular neuromas than the average population.

The symptoms that the neuroma can cause depend on its size and speed of growth. Usually the tumors grow slowly, and the vestibular system usually compensates for the slowly progressive loss of vestibular function. Therefore the patient is seldom dizzy. These patients, however, describe periods of unsteadiness that usually disappear within 6 – 9 months. This most likely reflects the progressive loss of function, which is hidden by the compensatory mechanisms.
With increasing size, the tumor will have an effect on hearing. Patients perceive this as difficulties understanding telephone conversations with one ear or as a sudden reduction in hearing. Unilateral tinnitus is frequent.

A larger tumor impedes the brainstem and the cerebellum. At this stage the patient starts having symptoms of cerebellar dysfunction and may also be of effects on the trigeminal ganglion. Symptoms like gaze-evoked nystagmus and ataxia usually appear. Facial problems are not commonly an early sign of a vestibular schwannoma. If the tumor continues to increase in size however, it will eventually cause an increase of the intracranial pressure, which could lead to visual problems and even blindness. Untreated, the increasing intracranial pressure and the direct pressure on the intratentorial structures is fatal. The size of the tumor usually increases very slowly, but there are large individual variations.

**HISTORY AND HISTORY TAKING**

Small tumor:
Episodes of unsteadiness, which can disappear without the patient noticing.
- Unilateral retro-cochlear impairment found on examination.
- Unilateral tinnitus
- Less often there may occur unilateral sudden hearing loss
- Sudden vestibular loss – uncommon.

Larger tumors:
Cerebellar signs and balance problems with ataxia. Signs of increased intracranial pressure, headaches, visual disturbances, nausea, and sickness.

**SYMPTOMS**

In the earlier phases the patients have a normal ENT examination and neurological status. Unilateral sensorineural hearing impairment can be present. Examination of the eyes with Frenzel’s glasses rarely shows significant spontaneous or gaze-evoked nystagmus if small tumors are present.

However, a continuous positional nystagmus and particularly a head-shaking test showing a quick phase towards the normal ear occur relatively early.
Patients with larger tumors have cerebellar signs like ataxia and gaze-evoked nystagmus during fixation. The sensibility of the cornea and later of the skin of the face is gradually reduced. Facial nerve disturbances are rare.

**EXAMINATIONS**

Patients with unilateral hearing impairment or unilateral tinnitus should, of course, have their hearing tested. If pathology is found, further audiological testing is necessary to confirm or exclude retro-cochlear pathology (i.e., disease in the cerebellopontine angle). If available, an MRI scan is preferable.

To exclude the possibility of an acoustic neuroma, it is important to do an MRI scan of the cerebellopontine/internal auditory meati. The examination needs to be specified in order to have the required resolution. This can be done with a s.c. CISS program or with contrast (gadolinium) and with thin slices.

**TREATMENT**

Acoustic neuroma is usually treated in specialized centers. Treatment involves either surgical intervention in the posterior fossa through the inner ear or from the middle fossa or stereotactic irradiation (gamma-knife). Although the aim is to preserve hearing in case of smaller tumors, the patient can expect a further loss of hearing on the affected side.

Recent studies have shown that smaller tumors do not always grow or else they grow so slowly that surgery may be avoided or postponed. Intrameatal tumors and tumors extending less than approximately 10 – 15 mm into the pontine angle may not have to be treated immediately. In these cases ‘wait and scan’ is the recommended policy, i.e., following the tumor with repeated scans every year. This should be done in cooperation with a specialist. In fact, some data suggest that up to 80% of patients with intracanalicular tumours may never need any treatment. However, how this holds true in the long run remains to be seen.

*The most important symptoms of tumors of the brain other than acoustic neuroma: objective ataxial/balance disturbance, gaze-evoked nystagmus, or positional nystagmus, which is non-fatigable.*

Examination of patients suspected to have intracranial tumors/neuromas.
Vascular vertigo

Blood pressure and Dizziness

Dizziness caused by vascular problems can be schematically divided into three groups: blood pressure and vertigo, ischemia/stroke, and vertigo. Earlier migraine would have been included, but this is now controversial.

Vertigo can be found in patients whose blood pressure is too high or too low. The cerebral auto-regulation maintains a constant blood flow through the brain’s blood vessels. Oxygen transport and circulation, however, are seriously impaired if the blood pressure falls below 60 – 80 mmHg. It is possible that damage to the blood vessels and stenosis or arteriosclerosis in the older patients can lead to reduced blood flow and oxygen supply in localized areas.

Blood pressure values can fall due to:

- cardiac arrhythmia (e.g., atrial fibrillation), heart insufficiency, and stenosis of the larger blood vessels;
- disturbed blood vessel tonus as, for example, in Shy-Dräger syndrome or as part of neurological diseases with loss of vascular tension control;
- the classic teenage orthostatism. NB! Do not confuse with BPPV;
- over-treatment of hypertension in the elderly.

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HISTORY AND SYMPTOMS

The patients usually describe their symptoms as an unsteady feeling or maybe as pre-syncope with unsteadiness and a flickering in front of their eyes or a spinning sensation inside the head. With a greater drop in blood pressure the patient has a feeling of blackout and a falling sensation. The symptoms are usually triggered by positional changes either from sitting or lying down to standing up. It is important to differentiate this from BPPV. The patients can sometimes relate their symptoms to a sudden throbbing/pounding of the heart, which could indicate arrhythmias. It is, however, important to keep in mind that heart throbbing/pounding can be a secondary phenomenon to the unpleasant sensations triggered by the dizziness.

However, it is not uncommon that patients in the initial phases of hypertension describe periods in which they have significant balance problems. This can be a significant symptom of malignant hypertension.

TREATMENT AND EXAMINATION

The GP should take an ECG of most patients who have vertigo symptoms, at least those without any clear, specific diagnosis. Otherwise the examination should at least include measurements of the blood pressure.

BACKGROUND

Ischemia, stroke, and vertigo

Embolism or reduced blood supply to areas for balance control can lead to vertigo. The reduced blood flow can also affect the blood supply to the inner ear and the balance organs as well as the CNS. The circulation to the inner ear/labyrinth and areas of the cerebellum and brainstem can also be simultaneously reduced, which again leads to problems with balance control. This can, for example, happen with infarctions provoked by embolisms in the PICA (posterior inferior cerebellar artery) and the AICA (anterior inferior cerebellar artery) and in Wallenberg’s syndrome (tendency to fall, lateropulsion towards the diseased side, gaze-evoked nystagmus, ataxia, vertigo, and Horner’s syndrome). Some of these types of vascular insults can simulate vestibular neuritis, but most often have a normal impulse test.
The symptoms depend on the extent of the stroke or the ischemic area involved. In general, increased rotatory vertigo with apparent balance problems is found in patients with infratentorial damage, whereas supratentorial damage usually causes a sensation of unsteadiness. Another rule of thumb is that a combination of vertigo and other neurological symptoms suggests a CNS origin, whereas vertigo alone can be caused by CNS damage as well as damage in other parts of the balance system. According to our experience a CT scan is of less value in the acute phase, since ischemic lesions may not be observed initially. The golden standard is an MRI with thin slices over the posterior fossa. But also with an MRI, small lesions may not be detectable until after 48h.

HISTORY AND SYMPTOMS

The patient history and symptoms vary considerably. Patients are initially presumed to have had a previous history of suspected vascular disease, arrhythmias, and thromboembolisms. It has been shown that patients with recurrent, light rotatory vertigo over a short period of time are later hospitalized with a stroke in the posterior fossa.

The history and findings of simultaneous neurological deficit symptoms suggest CNS damage. A sudden onset usually indicates a vascular origin. A tendency to fall in the direction of the fast phase of nystagmus also suggests a posterior fossa lesion or vascular origin. Falling in the direction of the slow phase can be caused by both CNS damage and acute vestibular damage.

TREATMENT AND EXAMINATION

Although the neurological literature on stroke should be consulted, there are some ground rules: exclude sources of emboli. Take an ECG, do auscultation/ultrasound of neck blood vessels, and perform a full otoneurological examination. If the source of embolism is excluded, an echocardiography must be performed. Patients with acute disease should be hospitalized. Observation and intervention can follow neuroradiological mapping. Acute recurrent attacks should be treated with prophylaxis, for example, with ASA preparations or their analogues. If chronic unsteadiness is suspected to have a vascular etiology:
BACKGROUND
Vertigo and unsteadiness are found in up to 70% of patients with migraine. These patients also commonly have an uneasy feeling when moving and an increased propensity for motion sickness both between and during the attacks. In about 5% of these patients the vertigo attack is rotatory. Moreover, patients with so-called Menière-equivalent disease or diseases with vertigo as the main or only symptom are thought to have migraine as cause. This category includes benign paroxysmal vertigo of childhood, benign recurrent vertigo, and basilar migraine. It has been suggested that it should be called vestibular migraine.

HISTORY AND SYMPTOMS

Benign paroxysmal vertigo of childhood. These are sudden, minute-long attacks of vertigo. The disease usually affects pre-school children who either gradually grow out of the symptoms or develop a classic migraine. If examined during a good phase, the children appear to be normal.

Recurrent vertigo. These attacks usually last for minutes to hours in the older patient but can occasionally also last up to 24 hours. The patient complains of unsteadiness and occasionally rotatory vertigo. Spontaneous nystagmus and positional nystagmus have occasionally been observed in the acute phase in these patients. There are no other audiological or neurological symptoms. If examined in a symptom-free interval, the patient appears to be normal.

Basilar migraine. The neurological literature on this diagnosis should be consulted. Vertigo and other posterior fossa neurological symptoms are gradually replaced by migraine-like headaches, nausea, and vomiting.

“Vestibular migraine”. This entity is considered a part of the basilar migraine as discussed above. The patient has a monosymptomatic vertiginous attack.

1. Optimize the vascular function – refer to internist.
2. Give anti-thrombosis prophylaxis.
3. Refer patient to vestibular rehabilitation and motion training.
The vertigo is often rotatory, sometimes accompanied by nausea. The attacks can last for minutes to hours and even up to days and then gradually disappear. According to the International Headache Society’s criteria, these attacks alternate with a characteristic migraine attack in the vast majority of patients. The following recommendations are given for establishing a diagnosis of vestibular migraine:

**Definite Vestibular Migraine:**

A Episodic vestibular symptoms – vertigo, prolonged positional dizziness/vertigo, head-movement discomfort

B History of certain migraine headaches

C At least one of the following symptoms during at least two different attacks
   - Migrainous headache
   - Photophobia
   - Phonophobia
   - Auras

D Other causes ruled out by careful examinations

**Probable Vestibular Migraine:**

A Episodic vestibular symptoms of at least moderate severity – vertigo, prolonged positional dizziness/vertigo, head-movement

B At least one of the below:
   - Migrainous headache
   - Triggered by migraine precipitants like special food, stressful situation, hormone changes, etc.
   - Effect of pharmacological treatment of migraine

C Other causes ruled out by careful examination
CRITERIA FOR VESTIBULAR MIGRAINE

EXAMINATION AND TREATMENT

If a patient with a migraine diagnosed according to the above criteria has attacks of rotatory vertigo similar to the ones described, prophylactic treatment can be tried. If a migraine-related disease without any other migraine history is suspected, the patient should be referred to a neurologist or an neuro-otologist.

Prophylactic treatment can, for example, include beta-blockers. Anti-emetics can be tried during the acute phase. Triptans may or may not work. As the symptoms usually cause nausea, nose-spray preparations are recommended.
Literature

Vertigo: Its Multisensory Syndromes

Brandt Th, Dietrich M, Strupp M.

Vertigo And Dizziness: Common Complaints

Bronstein A & Lempert Th, Cambridge University Press

Dizziness

Disorders of the vestibular system

edited by Robert W. Baloh, G. Michael Halmagyi
New York; Oxford University Press, 1996

Clinical Neurotology

LR. Lustig, JK. Niparko, LB. Minor, DS. Zee
Martin Dunitz, London New York
A home-based training program for dizziness

NOTE: You will experience some dizziness when you do these exercises!

1. Sit down, fixate an object 1.5 - 2 m away. Shake your head horizontally from side to side – still fixating the object. Repeat twice within one second for 15 seconds. Count from one-thousand-one, one-thousand-two …to one-thousand fifteen to keep track of the time.

2. Stand up and put a finger on a stable object (chair/table), fixate an object 1.5 – 2 m away. Shake your head horizontally from side to side – still fixating the object. Repeat twice within one second for 15 seconds. Count from one-thousand-one; one-thousand-two …to one-thousand-fifteen to keep track of the time.

3. Stand up without supporting yourself or touching any object, fixate an object 1.5 – 2 m away. Shake your head horizontally from side to side – still fixating the object 1.5 – 2 m away. Shake your head horizontally from side to side – still fixating the object. Repeat twice within one second for 15 seconds.

4. Stand up and close your eyes (first with and then without support). Fixate an object 1.5 – 2 m away. Shake your head horizontally from side to side – still fixating the object. Repeat twice within one second for 15 seconds.

5. Stand up, fixate an object 1.5 – 2 m away. Shake your head vertically, up and down – still fixating the object. Repeat twice within one second for 15 seconds.

6. Walk forwards fixating an object 1.5 – 2 m away. Shake your head horizontally from side to side – still fixating the object. Repeat twice within one second for 15 seconds.

7. Stand on a pillow from the couch in a corner of the room. Remain there for 1 minute, then close your eyes and remain standing like this for another minute. If it is difficult, place a chair in front of you. You can initially place a fingertip on the chair.

8. Stand up again in a corner of the room, this time holding a half-filled glass of water. Remain there for 1 minute, then close your eyes, and keep standing for another minute. If it is difficult, put a chair in front of you. You can initially place a fingertip on the chair.

9. Take a walk outdoors for at least 30 minutes. Try window shopping. While window shopping you will turn your head from side to side while walking.
Training program for benign paroxysmal positional vertigo

BPPV is caused by aggregation of debris or small crystals in a part of the inner ear, i.e., the posterior semicircular canal. The aim of this training program is to relieve you from the vertigo of BPPV by moving these small particles out of the posterior semicircular canal. When correctly performed, the three physical liberatory maneuvers (Semont or Epley liberatory maneuvers, Brandt-Daroff exercises are successful in almost all patients. Nowadays we recommend either the Semont or the Epley maneuver as the therapy of first choice. The Semont maneuver is to be performed in the following way:

1. In the sitting position, the patient turns his/her head horizontally 45° to the unaffected ear.
2. The patient is tilted approximately 105° to the affected ear. The patient maintains this position for 1 min.
3. The patient is turned approximately 195° toward the unaffected ear with the nose down. The patient remains in this position for 1 min.
4. The patient is slowly moved into the sitting position. The patient remains in this position for 1 min.

The three positioning steps are performed with the aid of a therapist as the patient lies on the examination couch and has then to be learned by the patient so that he or she can do it by themselves. It is important that the head of the sitting patient is turned by 45° to the healthy ear, in order to put the responsible posterior canal into a position parallel to the plane of movement during the positioning. Relief is thus achieved in about 50% of the cases with one single maneuver. Liberatory maneuvers should be performed three times in the early morning and three times at noon. They are more effective in the first half of the day, because the clot that has developed overnight can be more easily removed from the canal than can single particles.