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Summary: Dizziness is a relatively common problem that can arise from a variety of causes. In many cases, optometrists can participate in the diagnosis and management patients with complaints of dizziness.

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THE DIZZY PATIENT: A REVIEW OF ETIOLOGY, DIFFERENTIAL DIAGNOSIS AND MANAGEMENT

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in Clinical Optometric Management

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KEY WORDS

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INTRODUCTION

"Doctor, sometimes I feel really dizzy" is a complaint sometimes heard during optometric examinations. A 1985 nationwide survey found that for patients over 25 years of age, dizziness accounted for 1% of all chief complaints presented to primary care providers.[1] In terms of management, 1.5% of patients with this complaint were admitted to a hospital, 4.4% were referred to a specialist, and 89% were prescribed medication.[1]

Dizziness becomes a greater problem as patients get older. In the elderly, almost one out of five report dizziness that impairs their daily activities or is severe enough to warrant medical attention.[1,2] In patients over the age of 75, dizziness is the most common complaint made to primary care physicians.[3] Given the frequency of the problem, it is not surprising that optometrists often encounter complaints of dizziness.

Typically, optometric management of dizziness involves an evaluation of the patient's refractive, binocular, accommodative, and visual health status. If this examination is not productive, the patient is usually referred to a family care physician. The physician will probably treat the dizziness with a symptom suppressing medication or might send the patient on to a specialist in an attempt to discover the cause of the problem. However, as the role of the optometrist expands more into that of a primary care provider, it is probable that optometrists will be expected to define the etiology of the patient's problem. Then, the optometrist can manage the problem if it involves a faulty information being provided to the brain by the visual system, or a consultation with a specialist can be requested.

What is Dizziness?

Patients typically define almost any state involving loss of normal mental processing as "dizziness." For diagnostic purposes, however, it is very important to obtain complete and accurate information regarding exactly what the patient experiences when they are dizzy. Symptoms will be described somewhat differently by each patient, but generally they can be classified into one of four categories: vertigo, near-syncope, disequilibrium, or ill-defined lightheadedness.[4] (Sometimes vertigo is considered to be a separate diagnostic classification, and the latter three problems are grouped together under the heading of "pseudo-vertigo," but it is useful to consider them independently.)

Careful classification of the patient's symptoms is important because it helps to determine a diagnosis and management plan. For this reason, each of the classifications will be reviewed separately and then their associated etiologies will be discussed. (See Appendix 1 for a review of the physiological mechanisms that affect balance and postural stability.)

CLASSIFICATION OF DIZZINESS

Vertigo

Patients who experience an illusion of false motion between themselves and the outside world are classified as suffering from vertigo.[5-9] Patient complaints consistent with this diagnosis include "the room is spinning," "things are whirling," " I am reeling," "everything is swaying," "things are pitching," or "it looks like things are rocking."[10]

Patients who have vertigo share two attributes in common: they perceive illusory motion and they typically do not experience a loss of consciousness. The motion perceived by patients experiencing vertigo can either involve a perception that the world is moving while the body remains still, or that the body

is moving while the world remains still. In the first case, the patient is said to be experiencing objective vertigo, and, in the second case, subjective vertigo.[6] Although different neural mechanisms might be involved in these two conditions, the type of vertigo the patient experiences is not believed to have specific diagnostic significance. In both cases, the vertigo probably results from a discrepancy between the brain's expectation of sensory inputs and the conflicting cues it receives from the vestibular, visual, and/or somatosensory systems.[7,11,12]

Vertigo, except for the very mildest forms, is usually accompanied by varying degrees of nausea, vomiting, pallor, and perspiration.[13] These signs and symptoms indicate excessive activity in the autonomic nervous system. The patient's gait can also become unsteady and walking might be difficult during an acute attack of vertigo. The sensations of false movement can be so intense that the patient could be forced to lie down, usually on one side, with the eyes closed.[13] Even the slightest motion can aggravate the disorientation, nausea, and vomiting, so patients with acute attacks of vertigo tend to remain very still.

Near-Syncope

Syncope (or fainting) is defined as a transient loss of consciousness usually caused by inadequate blood flow to the brain.[5,14,15] Near-syncope (sometimes called pre-syncope) involves the patient's perception that she or he is about to faint.[10] This condition differs from true syncope in that loss of conscious does not actually occur, but there is no qualitative difference between syncope and near-syncope with respect to the differential diagnosis.[16]

Commonly, patients who experience near-syncope refer to their symptoms as dizziness, but careful questioning can separate this sensation from the spinning or moving sensations associated with vertigo. Near-syncope

usually occurs as acute attacks, but occasionally patients describe chronic problems.[17] The severity of the patient's symptoms depends on the magnitude of the blood flow reduction experienced by the brain.[5,16]

Disequilibrium

Disequilibrium involves the sensation of losing one's balance without a feeling of illusory movement or impending loss of consciousness.[5,10] Unsteadiness, particularly during walking, is a hallmark of disequilibrium; patients do not typically report problems when sitting or lying down.[4]

Disequilibrium usually occurs because of a disruption in the integration between the patient's sensory inputs and their motor outputs. In normal persons, the brain coordinates the visual, vestibular, cerebellar, and somatosensory processes to maintain a sense of equilibrium and balance. A dysfunction within any of these systems can create disequilibrium.[11,16,18]

Aging is significantly associated with the onset of disequilibrium because the ability of the nervous system to process sensory inputs and control the postural reflexes declines with advancing age.[11,19,20] The effects result from a widespread degeneration of the musculoskeletal, neuromuscular and sensory systems.[21] Disequilibrium-related gait disturbances in the elderly are particularly accentuated by unfamiliar surroundings and/or dim light, so many older persons avoid these situations. Although disequilibrium is not uncommon in the elderly, this symptom in young patients can suggest neurological disease.

Ill-Defined Lightheadedness

Normal persons feel lightheaded from time to time. The sensation can involve components of vertigo, pre-syncope, and/or disequilibrium, but the key feature of ill-defined lightheadedness is that the sensations are quite mild. For

example turning the head sharply to the side or standing up quickly can produce a transient sensation of lightheadedness.[22]

Most people ignore these common, everyday sensations, but, for some patients, they can become quite bothersome. This is particularly true for patients who become sensitized to them because of psychological problems associated with anxiety, or because of other disturbances that have previously caused acute attacks of dizziness. These patients often place themselves in a "hyper-vigilant" state during which they constantly monitor their bodies for any signs of impending dizziness. This can cause them to exaggerate their reactions to normal changes in body function and can produce great psychological distress.[23]

Lightheadedness can be very vague and difficult to diagnose accurately because patients often have trouble describing their sensations. Stress reactions, anxiety, depression, hypochondriasis, and panic disorders can all have lightheadedness as a primary complaint.[11]

However, care must be taken not to dismiss ill-defined lightheadedness as "all in the patient's head," and therefore of little concern. It can be an early symptom of a serious physiological or psychological problem, and it can be associated with a significant reduction in the patient's quality of life.

ETIOLOGIES OF DIZZINESS

Accurate classification of the patients symptoms into one of the four categories described above often provides valuable clues to the etiology of the underlying problem. Figure 1 shows how such a classification system can be used to aid in determining the cause of a patient's dizziness.

Insert Figure 1 About Here

However, it is quite possible for a patient to experience problems that fall into several different categories. For example, a patient with acute episodes of vertigo might produce enough vagal activity to develop near-syncope or syncope. For this reason, careful clinical evaluation is necessary to discover the actual cause of the patient's dizziness. It must also be kept in mind that patients can present with multiple, co-existing problems, and this greatly complicates the diagnostic process (e.g., a patient with a vestibular problem creating vertigo could easily become worried enough to create a significant psychological problem).

Although there are many causes of dizziness, five major categories will be reviewed: sensory conflicts, psychological problems, blood flow disorders, pharmaceutical effects, and systemic disease (including aging). Of these causes, sensory conflicts will be discussed in greatest detail because they are most closely related to problems involving the visual system.

Sensory Conflicts

Under normal circumstances, the brain compares inputs from all of its sensory systems to determine posture, position in space, and body movement. When the information from all of the systems agrees, there is no problem. But, when disagreement is noted, problems begin. Even in normal patients, these sensory disagreements can occur when riding in the back seat of a car or in an airplane.[24] This is because the vestibular system detects motion, but the visual system sees only the inside of the vehicle that remains fixed in space with respect to the observer. Another example is viewing a wide screen movie in

which the audience vicariously experiences flying a jet fighter. The visual system reports motion, but the vestibular apparatus does not.

Vertigo is often produced when the brain is unable to resolve conflicting sensory messages. These conflicts can arise because the brain is receiving faulty information from the receptor organs (e.g., the eyes or vestibular system), or because of faulty processing within the brain itself.

A side-effect of this sensory conflict is an excessive response by the autonomic nervous system This causes the sweating, pallor, nausea and vomiting experienced by patients with vertigo.[13,25] Dimenhydrinate, better known as Dramamine®, and a number of other prescription and non-prescription antihistamines can control some of these symptoms, presumably by suppressing vestibular output and reducing the amount of conflicting information the brain must process.[26-28]

When vertigo is not associated with actual movement, peripheral sense organ problems account for about 85% of the cases, whereas central processing disorders account for only 15%.[29] (Central disorders refer to problems in the brainstem or its projections to parts of the cerebral cortex, especially the temporal lobe. Peripheral disorders refer to problems occurring at the end organs or in the peripheral nerves.[16])

Peripheral Vestibular System Problems

The nature of the patient's symptoms can help in determining what is causing the problem. For example, data in Table 1 indicate how characteristics of the patient's vertigo can be used to help in determining whether the problem is central or peripheral.[11]

Insert Table 1 About Here

Vestibular Apparatus

The vestibular apparatus is located in the inner ear and consists of a network of tubes called the semicircular canals and sacs called the vestibule. This network is filled with a small amount of fluid called endolymph and is surrounded by a second fluid called perilymph.[30] As the head moves, the inertia of the endolymph in the vestibular apparatus causes deformation of hair cells, and this results in the production of neural signals. These signals travel along the eighth nerve (the vestibulocochlear nerve) into the brain. The eighth nerve carries both auditory and vestibular axons, so it is common for patients with vestibular problems to also experience hearing anomalies. Also, because of the several reflexes that link eye movements to outputs from the vestibular system (e.g., the vestibular-ocular reflex), it is typical for patients with vestibular problems to also report visual difficulties including nystagmus and shimmering of objects.[25,31]

Perilymph Fistula

Several problems can cause the vestibular system to transmit faulty information. For example, trauma can cause mechanical damage to the vestibular apparatus or a more subtle problem called a perilymph fistula.[32,33] In the case of a fistula, perilymph leaks from the inner ear into the middle ear through a small hole in either the round or oval window membranes.[32] The result of this leakage can be a major alteration in the function of the vestibular apparatus with vertigo occurring as the brain attempts to process defective messages from the vestibular system. After an acute phase, the fistula can seal

itself, but often it reopens intermittently as a result of exertion or changes in atmospheric pressure (e.g., flying in an airplane or a Valsalva maneuver such as a cough or sneeze).[34] Treatment for this condition usually involves bed rest to encourage sealing of the fistula, or, in severe cases, an attempt can be made to patch the leak surgically.[30]

Endolymphatic Hydrops

Another problem that can cause the vestibular system to transmit faulty information is endolymphatic hydrops. In this condition, the pressure and/or electrolyte balance of the endolymphatic fluid inside the vestibular apparatus becomes unstable.[8] As a result, faulty signals are sent from the vestibular apparatus to the brain and this produces vertigo.

Pressure of the endolymph is maintained, in part, as a result of osmotic gradients between the endolymph, perilymph, and blood, so treatment is usually directed at stabilizing these relationships. Diuretics can be used for this purpose and hydrops patients are cautioned against upsetting their osmotic balance by consuming large quantities of salt, sugar, or alcohol. Some patients have found that they can self-medicate with these substances because their consumption temporarily reduces the endolymphatic pressure; unfortunately, the subsequent pressure rebound can greatly exacerbate their hydrops symptoms.

Hydrops patients often follow a chronic course with periods of greater and lesser symptom production (i.e., their symptoms can vary between mild disequilibrium and significant vertigo).[25] If symptoms become intolerable, a shunt can be surgically implanted to allow regulation of endolymph fluid pressure, or, if the symptoms become incapacitating, the vestibular apparatus and/or the eighth nerve can be partially or totally destroyed.

Ménière's Disease

Ménière's disease is a condition possibly related to hydrops. Both are thought to be related to changes in fluid pressure in the inner ear.[35] However, one of the major differences between Ménière's and hydrops is that Ménière's patients tend to experience severe, acute episodes of vertigo whereas hydrops patients have problems that are often more chronic in nature.[11,13,16,25,29,34] Although the exact cause of these conditions is unknown, some have speculated that an allergy might be involved.[25] Therapy for Ménière's is roughly the same as for hydrops including dietary restrictions, medication, and possible use of destructive procedures in severe cases.[36,37]

Infections

Viral or bacterial infections can also disrupt the function of the vestibular apparatus and cause the transmission of confusing information to the brain.[10,16] Normally the inner ear is well protected against infections because it is sealed by its membranes against contact with the air, but bacterial middle ear infections (e.g. otitis media) can occasionally spread to the inner ear (possibly via a fistula) and produce labyrinthitis. It is also possible to have viral infections of the inner ear secondary to influenza and other systemic diseases.[8] Typically, inflammation of the inner ear will cause both vestibular and hearing problems with the vestibular problems manifesting as vertigo and nausea. Treatment of these problems relies on an accurate diagnosis and the use of appropriate antibiotics if the infection is bacterial.

Benign Paroxysmal Positional Vertigo

Although there are numerous other causes of vertigo related to vestibular function, one of the more common causes is benign paroxysmal positional

vertigo (BPPV). This condition is also sometimes referred to as benign paroxysmal positional nystagmus (BPPN) because its occurrence is often accompanied by nystagmus. As its name implies, BPPV is a benign condition that involves an acute sensation of vertigo that occurs each time the head is moved in a certain direction.[8-10] The particular head movement that elicits the vertigo is different for different patients, but many BPPV patients have discovered for themselves what movement produces their symptoms. As with many of the vestibular problems, the exact cause of BPPV is difficult to specify and may vary from patient to patient. Treatment can range from teaching the patient to avoid the movements that elicit the vertigo, to reassurance, and trial courses with anti-vertigo medications.

Detection of Peripheral Vestibular System Problems

Case History

Probably the most important method used to detect peripheral vestibular problems is a detailed case history including an exact statement of what the patient senses when they are "dizzy," any history of trauma or illness, what conditions elicit the symptoms, and what produces relief. It is also important to consider other physical anomalies that patient might have. A history of trauma can often be helpful in the diagnosis of fistula, specific head movements that elicit vertigo can help to diagnose BPPV, and other physical characteristics can help to diagnose syndromes that include vestibular disorders. An example of such a syndrome is the Waardenburg Syndrome. Patients with this syndrome have unusually wide-set eyes, cafe-au-lait spots, a white forelock, and inner ear problems that cause dizziness.[8,38]

It is imperative that the clinician allow the patient to verbalize their symptoms. Direct questions such as "do you feel as though you will fall?" should

be avoided; patients will many times inaccurately respond yes. "What do you mean by dizzy?" is a better, open-ended question that will yield more beneficial information.[16]

Caloric Nystagmus

Often the reflexive interactions between the visual and vestibular systems can be used to aid in the diagnosis of peripheral vestibular problems. One of the most commonly used tests is caloric nystagmus in which the vestibular apparatus is warmed or cooled by the injection of fluid into the external auditory canal.[6,8] If the vestibular apparatus is functioning normally, a reflexive nystagmus is produced. The direction of this nystagmus is dependent on the ear being stimulated and on the fluid temperature. (When the fluid is cool, the nystagmus fast phase is in a direction opposite to the ear being stimulated, and when the fluid is warm, the fast phase is toward the ear. This relationship is associated with the acronym "COWS" - cold opposite, warm same.)[6,8]

Audiology

Because the peripheral vestibular and auditory systems share a common location, nerve, and fluids, vestibular problems are often associated with auditory difficulties. These hearing problems can range from an absolute or relative loss of ability to detect sound, to the false sensation of ringing or whistling sounds (i.e., tinnitus). These hearing problems can be monaural or binaural and can provide strong clues regarding the etiology of a patient's dizziness symptoms.[39]

Special Testing Procedures

Neurotologists and neurologists have developed a wide range of special testing procedures designed to diagnose vestibular problems. For example, electrocochleography is a technique in which a series of air pressure pulses are presented to the ear and the resultant electrical responses from the cochlea are recorded.[40] Analysis of these responses can help in making a diagnosis of endolymphatic hydrops. Another example is electronystagmography. This procedure can be used to record the nystagmoid eye movements associated with a balance system anomaly.[41]

Clinicians can also employ positional and rotational testing to determine vestibular dysfunction. Some of these procedures are the Nylen-Bárány maneuver, harmonic acceleration test, Romberg test and posturography.[8,11,15,16,30,41]

Other tests, such as measurement of brain stem potentials evoked by sound pulses, can also be helpful in detecting the auditory information processing problems that suggest vestibular or eighth nerve disease.[32,39]

Central Vestibular System Problems

Even if accurate information about movement, position in space, and acceleration is generated by the vestibular apparatus itself, this information can be garbled in transmission along the eighth nerve or it can be misinterpreted within the brain itself. For example, a patient's symptoms of dizziness can be caused by one of the more common brain tumors: an acoustic neuroma.[8] This tumor grows along the course of the eighth nerve as it passes into the brain cavity, and can compromise information transmission in both the auditory and vestibular portions of the nerve. In addition, as the tumor grows into the cranium, it can compress the cerebellum and cause problems with movement

and balance. Patients with these tumors report problems with hearing and tinnitus more often than they report true vertigo.[8]

Within the brain itself, trauma, growths, interruptions in blood supply, and biochemical imbalances can compromise the processing of sensory information. The result of this compromise can be a conflict when the vestibular information is compared to the information produced by other sensory systems. These conflicts can result in "neural confusion" and the generation of perceptions, such as vertigo, that do not conform to physical reality (i.e., the patient senses motion when none is occurring). The conflicts can also result in much more subtle "psychological" problems including an inability to concentrate, depression, and feelings of fatigue that occur as the brain is forced to devote resources to the problem of straightening out conflicting sensory messages. In fact, the "psychological" problems associated with these information processing conflicts can be so severe that they can cause patients to contemplate self destructive acts.[42]

Detection of Central Vestibular System Problems

When central problems are caused by tumors or trauma, they are often detectable by using an imaging technique such as computerized tomography (CT scan) or magnetic resonance imaging (MRI).[8] The more subtle information processing problems are harder to detect because the failures are not detectable with imaging techniques. Instead, they must often be inferred from the patient's history, by observation of the patient performing routine tasks, and the results of more formal neuropsychological testing.

Peripheral Visual System Problems

Just as unreliable or erroneous information from the vestibular system can cause sensory conflicts, unreliable visual information can also create conflicts. Along with dizziness, many patients with visual system problems complain about photophobia, motion sickness, inability to function in situations involving peripheral motion (e.g., moving down the aisle in a grocery store or library), reading difficulties, and anxiety.[43] Common causes of faulty visual information include abnormal eye movements, lack of image clarity, size differences, binocular conflicts, and field reductions.

Eye Movements

When examining a patient complaining of dizziness, special care should be taken to check for nystagmus or failures of the normal visual/vestibular reflexes. In cases of gross vestibular reflex failure, patients can lose the ability to hold the visual world stable as they move their head and will complain that the world bounces up and down as they walk (oscillopsia).[25,44] Some patients also complain about blur, but, on closer questioning, it will be found that they are really experiencing shimmer or colored fringes on letters possibly because of small nystagmoid eye movements.

Image Clarity

Vision care specialists typically provide lenses that yield the maximum acuity for their patients. Most of the time this approach is successful, but occasionally patients will have trouble adapting to new lenses, especially if the retinal images created are considerably different than the images produced by the patient's old lenses. This is because the brain has adjusted its expectation to the images produced by the old lenses and the new images create conflicts

with the expectation. Another source of conflict might involve conflicts that would occur when the information carried by the new images is compared to the information coming from the vestibular system. Most patients adjust to their new lenses in a few days or weeks, but some will experience the vertigo, nausea, and discomfort associated with sensory conflict during this adaptation period. A few patients, especially those with vestibular or hyper-vigilance related psychological problems, will be unable to adapt to their new lenses and will be very willing to sacrifice acuity for a reduction in conflict symptoms.

Binocular Image Conflicts

Binocular image conflicts can occur because the images produced on the retinas are of unequal sizes, or because the eyes fail to align properly. Often an image size difference (aniseikonia) is created when the lenses are of significantly different powers are used to compensate for anisometropia.[45] This difference in lens powers causes the images produced on the retina to be of different sizes which makes it difficult for the brain to fuse the images into a single precept. Other patients have aniseikonia that occurs without anisometropia; this condition can sometimes be quite difficult to detect. Treatment for aniseikonia can involve the prescription of contact lenses, as in non-axial cases, instead of spectacles for anisometropic patients to reduce the difference in magnification effects, or the use of "size lenses" that can change retinal image sizes.

Ocular alignment problems can involve potential misalignments that require constant effort by the patient to overcome, (e.g., high phorias or fixation disparities), or major problems in which the eyes are significantly misaligned, (e.g.,strabismus). These misalignments can be in the horizontal or vertical directions, or they can involve rotation of the eye around the line of sight.

Detection of these problems ranges from simple observation of ocular postures in the case of strabismus, to the need for prolonged occlusion to elicit the problem in the case of latent vertical deviations.[43] It is also possible for the misalignment to occur only intermittently (e.g., during periods of high stress), and perhaps only during specific tasks such as reading or where the eyes are in specific positions of gaze.

Patients with normal binocular alignment and those with gross misalignments are often relatively symptom free, whereas those with deviations that are intermittent, small enough to overcome with mental effort, or situation-specific experience the most symptoms. As an adaptive measure, the visual system can develop the ability to suppress a considerable portion of the information from a deviating eye if the deviation is large and constant.[45] However, if the deviation is not large and constant, the brain must work to "sort-out" the information from the two eyes and compare it to the information arriving from the vestibular system. This process can produce symptoms of dizziness in many patients.

Treatment of ocular misalignment involves restoring alignment optically, surgically, or with vision therapy. Care must be taken, however, when attempting to restore binocular vision to patients who have adapted to their deviations by learning to suppress the image from the deviating eye. Often these patients experience an increase in symptoms before they feel better. In some cases, especially with elderly patients, binocular vision can be intentionally disrupted with an occluding filter over one eye to remove a sensory conflict.

Reduction in Visual Fields

The brain uses information from the peripheral visual fields, along with vestibular and other sensory information, to maintain balance and a sense of position in the environment. Restricted fields can, therefore, cause many patients to experience disequilibrium, especially when the field loss is recent.[46] Skills associated with cognitive mapping, the ability to move about in a complex environment, and eye-hand coordination can also affected by field restrictions.[46]

PSYCHOLOGICAL CAUSES OF DIZZINESS

Dizziness is seen as a presenting complaint in a variety of psychological problems,[42] especially the anxiety neuroses and other conditions in which hyperventilation or cardiac manifestations occur. At one clinic specializing in dizziness, approximately 1 in 5 patients was diagnosed as having a psychogenic etiology for their dizziness, and at another clinic, the ratio was almost one in three.[4] Although it is easy to dismiss psychogenic dizziness as "all in the patient's head" and thus of little concern, it is quite possible that the initial cause of the psychological problem was physiological (e.g., vertigo caused by a sensory conflict in a high stress situation). For this reason, all dizzy patients should be evaluated for possible physiological problems before making a diagnosis of psychogenic dizziness.

Anxiety Neuroses

About 5% of the population experience significant episodes of unrealistic anxiety (i.e., anxiety that is not appropriate for the situation).[6] This anxiety can be chronic and involve a constant state of heightened internal and external environmental awareness, or it can be acute and occur in the form of panic attacks.[42]

Patients with chronic anxiety often report a constant fear that something bad is going to happen, trembling, frequent sweating, butterflies in the stomach, vague chest pains, palpitations, and other symptoms that suggest the body's flight or fight mechanism is being activated.[23] The dizziness that accompanies these feelings can originate from a variety of sources including vasoconstriction and hyperventilation.

Panic attacks are episodes of acute anxiety that occur for no known reason. They can last from a few seconds to many minutes and are frequently so intense as to be disabling. Sensations include chest pain, near-syncope, dizziness, possible vertigo, intense need to escape from the location where the attack is occurring (i.e., to hide), sweating, and extreme fear.[23] Often patients with panic attacks feel that they are having a heart attack and/or are going to die. These attacks can occur several times per day, or there can be days, weeks, or months between them. This uncertainty intensifies the patient's fear of the attacks, and can result in agoraphobia in which the patient is afraid to leave home. As with chronic anxiety, the dizziness seen during panic attacks probably has a respiratory and/or vascular etiology.[47]

The symptoms associated with chronic or acute anxiety are the same as those seen in conditions having more readily defined "physiological" causes (e.g., sensory conflict or hypoglycemia), and it has even been suggested that some anxiety episodes are caused by a heightened responsiveness to the sensations produced by sensory conflicts. In addition, many conditions such as vestibular or visual disturbances have anxiety as a symptom, so great care must be exercised when a diagnosis of dizziness caused by an anxiety neurosis is made just because no other explanation could be found.[42]

Hyperventilation Syndrome

To function properly, the body maintains relatively tight control over its internal environment. Temperature, pH, osmolarity, oxygen/carbon dioxide balance and other factors are typically well regulated through the use of various receptors and feedback loops. In the case of oxygen/carbon dioxide balance, however, this regulation can be upset when the patient's pattern of breathing is altered and hyperventilation occurs.[47] Symptoms can include air hunger, difficulty breathing, numbness or tingling around the mouth and in the extremities, dizziness, and possibly near syncope or syncope. Treatment ranges from breathing into a paper bag to restore the body's oxygen/carbon dioxide balance,[25] to the use of medications to prevent the anxiety that can trigger the hyperventilation. If actual syncope occurs, the body typically restores the proper balance without further intervention.

It is often impossible to determine whether hyperventilation creates anxiety, anxiety creates hyperventilation, or possibly a third factor such as sensory conflict creates both conditions. By the time the patient is seen, even if a treatable sensory conflict originally caused the problem, the anxiety (and dizziness) could have taken on a life of its own and can persist after the sensory conflict has been resolved.[42]

REDUCTION OF BLOOD FLOW TO THE BRAIN

Along with a proper chemical balance, the brain must also have an adequate supply of blood to function properly. Maintenance of the blood supply requires the heart to pump blood in proper quantities, at the proper pressure, through patent blood vessels. Compromising either the heart or the vessels can result in a variety of problems, one of which is often dizziness. Occasionally it has been suggested that hypertension itself can be a direct cause of dizziness,

but it more likely that one of the indirect consequences of hypertension such as cardiac or vascular abnormalities actually cause the dizziness.[6]

Cardiac Problems

Because the heart is a mechanical device with pump chambers, valves, and an electrical control system, failure of any component can result in reduced blood flow to the brain. Some failures, such as myocardial infarctions, are catastrophic, but others, such as arrhythmias, valves that do not close completely, or muscle weakness can result in either chronic or episodic reduction in blood flow. When these problems occur, the patient might be aware of chest sensations (e.g., in the case of some arrhythmias), of they might simply experience dizziness related to near-syncope or syncope.[5,7,11] If the cardiac problem is chronic, it can often be detected by the use of traditional electrocardiography, or echo-electrocardiography.[48] If the problem is episodic, accurate diagnosis could require the patient to wear a portable cardiac rhythm recorder (e.g., Holter monitor) until an episode occurs.[11] Some episodic problems occur as a result of excessive vagal activity that can produce transient bradycardia (i.e., slow heart rate). This excess vagal activity can result from anxiety and lead to dizziness, near-syncope, or syncope.[11]

Vascular Problems

Vascular problems limiting blood flow to the brain can also be chronic or acute. Chronic problems usually involve deposits that have built up on the walls of the vessels (e.g., atherosclerosis). Acute problems can involve mechanical obstructions such as clots or plaques that have become lodged at narrow spots in the vessels.[49] Other acute problems can be neural in origin such as

excessive vagal activity that can cause constriction of the vessels supplying blood to the brain.

PHARMACOLOGICAL CAUSES OF DIZZINESS

Perhaps the first question that should be asked after a patient has described a problem with dizziness is: "What medications are you taking?." A brief review of any standard reference on pharmacology will show that many drugs ranging from aspirin to cold medications (and even to the drugs actually used to suppress dizziness), have dizziness as a possible side-effect.

A search through the computer accessible version of the Physician's Desk Reference revealed that of the more than 4000 drugs listed, over 25% had dizziness listed as a possible side-effect.[50] This should not be too surprising when the various etiologies of dizziness are considered. For example, medications such as the diuretics change the body's osmolarity and this can affect the inner ear. Hydrochlorothiazides and alpha$_1$ blockers such as prazosin can cause dizziness as can calcium channel blockers such as verapamil.[27] These effects can be potentiated when multiple drugs are taken.

Long term or high dosage use of some medications such as the aminoglycosides can actually damage the inner ear.[8] In the case of streptomycin, the damage involves the vestibular hair cells beginning in the crista of the semicircular canals and extending to the otoconia in the utricle and saccule.[29,51]

Other medications that reduce blood pressure can create postural hypotension and dizziness. Still others create anxiety which results in dizziness (e.g., some decongestants and diet pills). Sometimes, sorting out the dizziness caused by the patient's disease from the dizziness caused by the medications used to treat the disease can be very frustrating.

SPECIFIC DISEASES AND AGE RELATED FACTORS ASSOCIATED WITH DIZZINESS

Many systemic and focal diseases can produce sensations of dizziness.

Specific Diseases

Dizziness is a common problem accompanying relatively benign diseases such as colds and influenza. In these cases, it can result from occlusion of the eustachian tubes, inflammation of the middle or inner ear, dehydration and resulting osmolarity changes, elevated temperature, or even the medications taken for symptom relief.

Other diseases producing dizziness are more significant. Perhaps the most common neurological diseases is multiple sclerosis in which the myelin sheaths of the nerves are affected.[13] In this disease, one of the early targets can be the optic nerves. The resulting optic neuritis can distort the information being sent to the brain and this can result in sensory conflict. Similar problems can affect the eighth nerve, and the parts of the brain responsible for balance and muscular control; it is common for patients with multiple sclerosis to report disequilibrium and dizziness.

Age-related Problems

The syndrome of multiple sensory deficits is probably the most common cause of disequilibrium in the elderly.[4] The syndrome can be caused by a combination of visual impairment, vestibular anomalies, peripheral neuropathies, or orthopedic problems, including cervical spondylosis, osteoarthritis and muscle weakness.[11] Parkinsonism, cerebellar disease and frontal lobe apraxia are other important causes of disequilibrium.[11]

Aging can also make patients more susceptible to the adverse reactions of medications. Antianxiety agents, such as the benzodiazepines (e.g.,

diazepam, chlordiazepoxide, and lorazepam) have been reported to cause dizziness, and this side-effect is especially prevalent in the elderly.[51] It is also common for the elderly to be taking diuretics that can affect the inner ear, or hypertension medications that can make them susceptible to orthostatic hypotension.

MANAGEMENT OF DIZZINESS

Dizziness is typically a symptom of an underlying problem, so management requires accurate diagnosis and appropriate treatment of the underlying problem.

Visual/Vestibular Conflict Therapy

If a sensory conflict is suspected, thorough examinations of the visual and vestibular systems are required. If ophthalmic problems are found they can usually be corrected by the use of lenses or vision therapy. In the case of vestibular problems, the treatment might require surgery to close a perilymphatic fistula[52] or remove a tumor, antibiotics to treat an infection, dietary restrictions to maintain a constant state of blood osmolarity, or the use of diuretics. In extreme cases, surgical or medical destruction of the vestibular apparatus or the vestibular portion of the eighth nerve might be required to provide relief for the patient.[53]

In other cases, simple procedures have been shown to dramatically reduce dizziness. For example, it has been suggested that BPPV is caused by dislodging the tiny calcium deposits that are normally attached to the hair cells in the inner ear. To treat this condition, the patient lies supine on a table with his or her head hanging off the side. The clinician then rotates the patient's head to the side until the dislocated calcium deposits move away from the part of the

inner ear where they are causing problems.[8] The patient then maintains an upright posture for 48 hours to allow the calcium deposits to settle into a less troublesome location. Using this treatment, Baloh has been reported to achieve a 90% first treatment cure rate.[54]

Habituation Exercises

If the problem causing the dizziness cannot be found or treated, sometimes it is possible to reduce the patient's awareness of the dizziness by habituation to the situation or stimuli that cause dizziness. This habituation is similar to what an ice skater or an acrobat goes through as they learn not become dizzy when spinning.[54]

Dynamic Adaptive Vision Therapy developed by Gillilan to treat motion sickness is an example of habituation training.[55] The therapy involves the use of devices including a lettered ball suspended from the ceiling. In some exercises, the patient is asked to watch the ball as it moves or to shift their gaze between various objects until symptoms are produced. The patient is then allowed to relax until the symptoms dissipate. This is followed by exposure to the provocative stimuli again. Gradually, over a four to eight week period, the patient's tolerance for the stimuli increases as habituation or adaptation occurs. In successful cases, the symptoms totally disappear by the end of therapy and the patient remains symptom free for years.[55]

Physical Therapy

Many patients with vertigo or disequilibrium have trouble standing or walking; in these cases, physical therapy might be very helpful. The therapy could be designed to help the patient adjust to the balance difficulties, teach new

methods of coping with the problems, or even attempt to reduce the level of the problem by use of habituation exercises.[30,56]

A commercial posturography test unit known as the Equitest has can be used diagnostically and to monitor the patient's progress.[57] The patient stands on a platform and undergoes a series of movements with feedback from one of the body's sensory systems (visual or proprioceptive) modified to give deceptive information. Results of posturography can be used to pinpoint which senses are responsible for the balance problem. Maintenance of equilibrium is then quantified and can aid in evaluation of the rehabilitation plan.[39,57,58]

Psychotherapy

Most subjects experiencing dizziness will also experience concomitant anxiety, especially until a physiological cause for the dizziness is found. If the underlying disorder can be treated, the anxiety is usually of little consequence. However, if no physiological cause is found, or if the underlying cause is not treatable, psychotherapy can help the patient adjust to the disability produced by the dizziness. The anxiety associated with the dizziness must not be allowed to develop into a major psychological problem such as agoraphobia in which the patient is afraid to leave home.

Psychotherapy is also useful for teaching anxiety reduction techniques such as diaphragmatic breathing. This is especially valuable in cases involving dizziness associated with hyperventilation.[42]

Medical or Surgical Enhancement of Blood Flow

If the patient suffers from episodes of near-syncope or syncope, evaluation of the cardiovascular system might indicate the need medication or surgery to enhance blood flow. For example, a malfunctioning heart valve may

need repair or a endarteriectomy might be necessary to restore blood flow blocked by atheromatous plaques.[48]

Medications Used to Suppress Symptoms

If the cause of the patient's dizziness cannot be found or treated, medications might be used to suppress the symptoms.[30] These medications fall into several categories: antihistamines, anticholinergics, diuretics, and anti-anxiety agents.

Antihistamines

Antihistamines are the mainstay of both over the counter and prescription anti-dizziness medications. Although there is still some question regarding their mode of action, the most commonly accepted theory is that they work by suppressing the output from the vestibular system.[27]

Common drugs used to manage dizziness are listed in Table 2. [27,59,60] Although these drugs are mainly antihistamines, they also have anticholinergic properties so they can cause drowsiness (and possibly anxiety reduction) as adverse reactions.

Insert Table 2 About Here

Anticholinergic Agents

Scopolamine (Transderm Scop®) is the only medication in this category commonly used in the management of dizziness; it is also effective against the nausea and vomiting associated with motion sickness.[16,27,30] The delivery vehicle is a skin patch placed behind the ear a few hours prior to the offending activity. One patch will release scopolamine for 3 days.

Scopolamine's mechanism of action is not completely understood, but it might act within the central nervous system[60] and/or on the vestibular system itself.

Scopolamine is a potent anticholinergic with significant adverse reactions; they include drowsiness, dry mouth, mydriasis, and accommodative dysfunction.[27]

Antidepressant/Antianxiety Agents

Dizziness can produce anxiety and depression, but anxiety and depression can also produce dizziness. This circular relationship suggests that most patients experiencing dizziness would benefit from the use of an antianxiety or antidepressant medication. If the underlying disorder is psychogenic in nature, this is probably true. But, if the dizziness is caused by sensory conflict or disequilibrium, these medications can sometimes exacerbate the problem. This is because the tranquilizing effects of these medications make it harder for the brain to resolve conflicting sensory messages and keep the body balanced. Never-the-less, anti-anxiety agents are frequently prescribed because the symptoms of dizziness are so upsetting to the patient.

Antianxiety agents can help patients to cope with their dizziness and some (especially Xanax®) can block panic attacks and the resultant near-syncope and hyperventilation. Most of these drugs appear to act on the limbic system, the thalamus, and/or the hypothalamus to produce their calming effects.[27,60]

For reasons that are not yet clearly understood, several antidepressant agents can lessen the severity of ill-defined lightheadedness found in some mood disorders. The main drugs in this category are the tricyclics, heterocyclics, monoamine oxidase inhibitors, and phenothiazines.[25,59] All

appear to exert their effects on the monoamine neurotransmitter/receptor system.[60]

Some of the commonly prescribed antidepressant and antianxiety agents that might be used in the management of dizziness are listed in Table 3.

Insert Table 3 About Here

Diuretic Medications

These agents have shown some promise in treating Ménière's syndrome and endolymphatic hydrops. They appear to function by correcting or stabilizing the osmotic balance in the inner ear fluids. The most commonly used diuretics are the thiazides and their combination formulations.[8,25,59] Acetalazolamide (Diamox®) has also been used for this purpose.

Calcium Antagonists

Some of these agents have been shown to have a vestibular sedative effect. The main drugs used are betahistine and flunarizine. The latter has provided better treatment results for both subjective symptoms and objective signs associated with dizziness.[61,62] These agents are under investigational use in Europe but are not yet available in the United States.

SUMMARY

Although dizziness is a relatively common patient complaint, it is often difficult to understand exactly what sensations the patient is experiencing. Careful history taking will usually help to define the sensations more fully and classify them as vertigo, near-syncope, disequilibrium, or ill-defined

lightheadedness. The correct classification is important because, as shown on Figure 1, this can suggest a diagnosis and management plan.

Some types of dizziness are relatively easy to manage (e.g., problems caused by improper spectacle lens powers), but most involve the failure of a peripheral sensory system, or the failure of the brain to process information correctly. In either case, diagnosis and management often requires a team approach including one or more specialists (e.g., optometrists, neurotologists, neurologists, etc.). Then, if the problem causing the dizziness cannot be eliminated, physical therapists and/or psychologists can help the patient adapt to the resulting disability.

Dizziness is a very uncomfortable and disconcerting problem for many patients, especially when its cause cannot be found. Dizziness can cause a significant decrease in the patient's quality of life, therefore complaints of dizziness must be taken seriously. Dizzy patients can experience profound depression associated with their dizziness and professionals who work with these patients should be alert for warning signs of self-destructive behavior.

Acknowledgment

Some of the material in the vestibular section of this paper has been drawn from information provided by the Vestibular Disorders Association (VEDA). Patients with vestibular problems, or health care providers who want more information on this topic, can contact VEDA at PO Box 4467, Portland OR 97208-4467; (503) 229-7705.

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Appendix 1

Physiology of Balance

It is helpful to review what mechanisms play a role in balance and position awareness with respect to the surrounding space. The eyes, labyrinths, muscles and joints offer continuous afferent impulses to inform us of bodily position.[13] As these inputs are received, our bodies make adjustments to maintain equilibrium. Most of these adaptive movements occur on a reflex level, therefore we are unaware of them.[8,13]

Vision provides sensory input which allows us to judge the distance of objects from the body.[8] Body orientation data is gathered from the visual system so as to place oneself properly in the environment.[63] Directional positioning, with respect to the vertical and horizontal planes, is sensed by the eyes; this occurs after the visual system has learned up, down and sideways from years of comparing visual cues with vestibular data.[28] Vision is a far reaching and rapidly processed sense. Therefore, it contributes strongly to bodily position awareness.[63] It has been postulated that proprioceptors from the extra ocular muscles also provide input but that appears unlikely.[30]

The labyrinths are composed of three semicircular canals (located at approximately right angles to one another) and the maculae of the saccule and utricle. The semicircular canals respond mainly to movement and angular acceleration, while the maculae are primarily concerned with static head position and linear acceleration.[8,13] With either subsystem, the stimulus to generate an afferent impulse is movement of the hair cells.[64,65] This process occurs when movement of the internal canal fluid bends small hair shafts within the canal creating an electrical output to nuclei in the brainstem. The electrical potential changes generated on one side are equal and opposite to the other side. The

hair shafts, rooted in cup-like hair cells, are composed of the proteins which form muscles, myosin or actin.[8,13] These hair shafts sense movement and transmit its intensity to the nerve synapses in the hair cells.

Two distinct fluid compartments exist within the inner ear: the endolymph and the perilymph.[65] This membranous separation allows a chemically created electrical potential to develop; the endolymph is high in potassium and low in sodium, whereas the perilymph is high in sodium and low in potassium.[65] A very high metabolic rate is maintained in the inner ear to ensure that the electrical potential across the endolymph-perilymph barrier can respond instantaneously to a stimulus.[30]

Impulses are received from the joint and muscle proprioceptors. This sensory information is vital to all reflex, positional and fine volitional movements. The proprioceptor input from the neck muscles and joints are important in relating the head position to the rest of the body.[13]

Coordination of the above sensory information occurs in the cerebellum and certain ganglionic centers and nuclei in the brainstem. From these areas, motor outputs are sent to the muscles to make postural adjustments and maintain equilibrium.[13] When these neural mechanisms are disrupted, dizzy symptoms ensue. These symptoms can also arise from the conflicting reports from different sensory receptors.[13,24,64,65] An example would be the motion sickness some patients suffer while reading in a moving automobile. Since the head is down and the eyes are focused on a stationary page, visual cues from the outside environment are not received. The eyes report to the brainstem centers that the body is stationary. The vestibular receptors are not blocked from the sensory data, as are the eyes. They properly report the movement of the vehicle to the brainstem.[24]

Appendix 2

Office Neurological Examination

The optometrist can examine a patient for neuropathology in an indirect manner. Since the brain and spinal cord communicate with many receptors through primarily involuntary reflex arcs, dysfunction within these arcs may indicate a brain disorder. A neurological hard sign can help pinpoint the nervous system location for the damage.

Diagnosis of the patient suspected of having a neurological basis for the complaint begins with a cranial nerve assessment. This diagnostic tool is well suited for the optometric clinic. The instruments and supplies needed are minimal and inexpensive. The testing procedures are non-invasive and mastered within a short time.[30,31,41,66]

CN	Name	Testing Procedures
I	Olfactory	Take 3 vials of strong and familiar odors such as coffee, vanilla or oil of peppermint. Have patient close eyes and occlude one nostril. Present the vials under the open nostril in a random order. Repeat for the other nostril. Failure to identify most of the smells may indicate dysfunction of the olfactory receptors in the nasal mucosa, dysfunction of the olfactory nerve or a lesion in the subcallosal or hippocampal gyri.
II	Optic	Perform visual acuity, pupillary function, field testing and ophthalmoscopy.
III	Oculomotor	Test EOM, lid and pupillary function.
IV	Trochlear	EOM function.
V	Trigeminal	Test jaw muscles (temporal and masseter) for strength and symmetry. Place palms on patient's cheek with fingers on patient's forehead and compare mouth opening, chewing and lateral jaw movement. Evaluate the sensory portion by having patient close eyes and touching all regions of the face with a cotton wisp. Have patient relate the location touched. Map any areas of anesthesia and repeat with a slight pin prick.
VI	Abducens	EOM function.

VII	Facial	Observe normal facial movements like smiling, raising eyebrows, whistling and closing eyes. Note any asymmetry or dysfunction.
VIII	Acoustic	The cochlear division involving hearing can be screened with a tuning fork. Hearing loss can be conductive (outer or middle ear) or sensorineural (inner ear, CN VIII or cochlear nuclei). Place the vibrating tuning fork in the middle of the forehead. If sound is softer on one side, it tends to indicate conductive loss. Place vibrating tuning fork one inch from the ear canal and then place on the ipsilateral mastoid bone. Then alternate these positions every few seconds until no sound is heard. Hearing longer through bone indicates sensorineural loss. The vestibular division involving balance and coordination can be tested in a rudimentary fashion by having patients perform simple motor skills. Some are standing on one foot, leaning over, jumping and spinning.
IX	Glosso-pharyngeal	CNs IX and X are tested together since they innervate many of the same structures. To test the motor function of the 2 nerves, have the patient open his mouth and say "ahh". Observe the palate and uvula position for asymmetry. Try to elicit the gag reflex by touching the back of the throat with a cotton swab. Ask the patient to swallow and observe if his larynx moves upward normally. Also, note any difficulty swallowing. The sensory components of the nerves can be tested by having the patient signal when he detects a cotton swab touching the pharynx.
X	Vagus	
XI	Accessory	Motor functions can be tested by placing hands on the patient's shoulders and having him shrug. Note any weakness or asymmetry of the trapezius muscle. Grasp the patient's chin and have him turn his head from side to side. Note any weakness or asymmetry of the sternocleidomastoid muscle.
XII	Hypoglossal	Motor responses can be tested by having the patient protrude his tongue, wiggle it from side to side and then place in each cheek. Note any atrophy, involuntary veering, asymmetry or weakness.

Appendix 3

Presentation for the Pacific University College of Optometry Vision Research Conference on March 13, 1994. Bold type and titles surrounded by asterisks indicate transparencies.

*******DIZZINESS ETIOLOGIES AND MANAGEMENT*******

IN A NATIONWIDE PRIMARY CARE SURVEY, DIZZINESS SYMPTOMS ACCOUNTED FOR 1% OF ALL CHIEF COMPLAINTS BY PATIENTS OVER 25 YEARS OF AGE. DIZZINESS BECOMES A GREATER PROBLEM AS PATIENTS GET OLDER. IT IS THE MOST COMMON COMPLAINT OF PATIENTS AGED 75 AND UP. ALMOST 1 IN 5 OF ELDERLY PATIENTS REPORT DIZZINESS THAT IMPAIRS THEIR DAILY ACTIVITIES OR IS SEVERE ENOUGH TO WARRANT MEDICAL ATTENTION. GIVEN THE FREQUENCY OF THE PROBLEM, IT IS NOT SURPRISING THAT OPTOMETRISTS OFTEN ENCOUNTER COMPLAINTS OF DIZZINESS.

TYPICALLY, OPTOMETRIC MANAGEMENT OF DIZZINESS INVOLVES EVALUATION OF THE PATIENT'S REFRACTIVE STATUS, PAYING CLOSE ATTENTION TO BINOCULAR AND ACCOMMODATIVE FINDINGS AND OCULAR HEALTH. IF THIS EXAMINATION PROVES NEGATIVE, WHAT IS THE NEXT STEP?

*******WHAT IS THE NEXT STEP?*******
SEND THE PATIENT HOME WITH AN APPOINTMENT FOR A CHECKUP?
MAKE A REFERRAL.... TO WHOM?
TO FAMILY PRACTICE?
TO CARDIOLOGY?
TO NEUROLOGY?
TO PSYCHOLOGY?
THE DISPOSITION OF THIS TYPE CASE CAN BE VERY CONFUSING.

AS THE ROLE OF THE OPTOMETRIST EXPANDS INTO THAT OF A PRIMARY CARE PROVIDER, IT IS POSSIBLE THAT FOR SOME PATIENTS, ODs WILL BE EXPECTED TO DIAGNOSE THE CAUSE OF THEIR DIZZINESS AND MAKE APPROPRIATE REFERRALS RATHER THAN JUST SENDING THEM TO A PRIMARY CARE CLINIC.

*****NORMAL SENSORY AND MOTOR RELATIONSHIPS*****

WHAT MECHANISMS PLAY A ROLE IN BALANCE AND POSITION AWARENESS?

EYES AND VISUAL SYSTEM
LABYRINTH SYSTEM (SEMICIRCULAR CANALS, SACCULE, UTRICLE)
PROPRIOCEPTIVE SYSTEM (MUSCLES & JOINTS)

COORDINATION OF THE SENSORY INPUTS OCCURS IN THE CEREBELLUM AND BRAINSTEM. EFFERENT (MOTOR) OUTPUTS FROM THESE AREAS PROVIDE POSTURAL ADJUSTMENTS AND THE MAINTENANCE OF EQUILIBRIUM.

LET'S DO A SHORT REVIEW OF THE MECHANISMS THAT PLAY A ROLE IN BALANCE AND POSITION AWARENESS.
THE INNER EARS OR LABYRINTHS CONTAIN 3 SEMICIRCULAR CANALS THAT ARE SENSITIVE TO ANGULAR MOVEMENT, THE UTRICLE THAT IS SENSITIVE TO LINEAR MOVEMENT AND THE SACCULE, WHICH MAY ASSIST THE SEMICIRCULAR CANALS. THEY ARE ALL PAIRED ORGANS LOCATED ON THE RIGHT AND LEFT SIDES.
THE LABYRINTHS, EYES & MUSCLES & JOINTS OFFER CONTINUOUS SENSORY IMPULSES TO THE BRAIN INFORMING IT OF BODILY POSITION AND MOVEMENT.
COORDINATION OF THIS SENSORY INFORMATION OCCURS IN THE CEREBELLUM AND BRAINSTEM. AS THESE INPUTS ARE RECEIVED, EFFERENT OR MOTOR OUTPUTS ARE SENT TO THE MUSCLES AND JOINTS TO MAKE ADJUSTMENTS TO MAINTAIN EQUILIBRIUM. MOST OF THESE ADAPTIVE MOVEMENTS OCCUR ON A REFLEX LEVEL, THEREFORE WE ARE UNAWARE OF THEM.
WHEN 1 OF THESE SENSORY INPUTS SENDS FAULTY DATA TO THE BRAIN, DIZZINESS SYMPTOMS OCCUR.
MEDICALLY SIGNIFICANT DIZZINESS DOES NOT INCLUDE NORMAL PHYSIOLOGIC RESPONSES. MOST PEOPLE FEEL LIGHTHEADED FROM TIME TO TIME. THE SENSATIONS ARE QUITE MILD AND QUICKLY PASS. FOR EXAMPLE, TURNING THE HEAD SHARPLY TO THE SIDE CAN PRODUCE DIZZINESS SYMPTOMS. THIS MERELY TELLS US THE SEMICIRCULAR CANALS ARE FUNCTIONING PROPERLY. WHEN ONE JUMPS OUT OF BED QUICKLY, A SENSATION OF LIGHTHEADEDNESS OCCURS. THIS IS MERELY A NORMAL TIME LAG FOR POSTURAL VASOMOTOR REFLEXES TO CONSTRICT THE PERIPHERAL BLOOD VESSELS. MOST PEOPLE IGNORE THESE COMMON SENSATIONS.

*****4 MEDICAL SYMPTOMS*****

VERTIGO
NEAR-SYNCOPE
DISEQUILIBRIUM
ILL-DEFINED LIGHTHEADEDNESS

NOW, LET'S DEFINE WHAT WE'RE DEALING WITH HERE. "DIZZINESS" IS A TERM WE USE ALL THE TIME, BUT IT IS NOT A VERY USEFUL MEDICAL DEFINITION. PATIENTS TYPICALLY DEFINE ALMOST ANY STATE INVOLVING LOSS OF NORMAL MENTAL PROCESSING AS "DIZZINESS". EVEN THOUGH SYMPTOMS WILL BE DESCRIBED SOMEWHAT DIFFERENTLY BY EACH PATIENT, GENERALLY THEY CAN BE CLASSIFIED INTO 4 MEDICAL SYMPTOMS. THEY ARE.....

*****VERTIGO*****

VERTIGO IS THE PATIENT'S PERCEPTION OF FALSE MOVEMENT. IT MAY BE THAT THE PATIENT FEELS AS IF HE/SHE IS MOVING IN SPACE OR IT COULD BE THAT SPACE IS MOVING AROUND THE PATIENT. THE DISTINCTION BETWEEN THESE 2 VERTIGINOUS SYMPTOMS IS NOT IMPORTANT BECAUSE IT PROVIDES NO USEFUL DIAGNOSTIC INFORMATION.
VERTIGO, EXCEPT FOR THE VERY MILDEST FORMS, IS USUALLY ACCOMPANIED BY VARYING DEGREES OF PALLOR, SWEATING, NAUSEA & VOMITING. THESE SIGNS AND SYMPTOMS INDICATE EXCESSIVE ACTIVITY IN THE AUTONOMIC NERVOUS SYSTEM. THE PATIENT'S GAIT CAN ALSO BECOME UNSTEADY AND WALKING CAN BE DIFFICULT DURING AN ATTACK. THE SENSATIONS OF FALSE MOVEMENT CAN BE SO INTENSE THAT THE PATIENT WILL BE FORCED TO LIE DOWN, USUALLY ON ONE SIDE, WITH THE EYES CLOSED. EVEN THE SLIGHTEST MOTION CAN AGGRAVATE THE DISORIENTATION, NAUSEA & VOMITING, SO PATIENTS WITH ACUTE ATTACKS TEND TO REMAIN VERY STILL. THIS SYMPTOM OF VERTIGO NORMALLY POINTS TO A VESTIBULAR DYSFUNCTION. SOME OF THE CAUSES OF VERTIGO ARE.....

PERIPHERAL VESTIBULAR DISTURBANCES
THESES ARE END ORGAN DISORDERS USUALLY AFFECTING THE VESTIBULAR SYSTEM OR PERIPHERAL NERVE. THEY ARE VERY DISCOMFORTING FOR THE PATIENT BUT TEND TO BE MORE BENIGN. ALL CAUSES CREATE THEIR SYMPTOMS BY SENDING FAULTY DATA TO THE BRAIN.
PHYSIOLOGIC CAUSES (MOTION SICKNESS)
VESTIBULAR NEURONITIS

THIS DISORDER IS CHARACTERIZED BY A SUDDEN AND SEVERE ATTACK OF VERTIGO. IT POSSIBLY HAS A VIRAL ETIOLOGY BUT THAT HAS NOT BEEN PROVED.

BENIGN PAROXYSMAL POSITIONAL VERTIGO

AN ACUTE SENSATION OF VERTIGO OCCURS WHEN A PATIENT PLACES HIS/HER HEAD IN A PARTICULAR POSITION. THE PATIENT MAY ALSO EXHIBIT NYSTAGMUS DURING THE EPISODE.

MÉNIÈRE'S SYNDROME/ENDOLYMPHATIC HYDROPS

THE PRESSURE AND/OR ELECTROLYTE BALANCE OF THE ENDOLYMPHATIC FLUID INSIDE THE VESTIBULAR APPARATUS BECOMES UNSTABLE.

POSTRAUMATIC VERTIGO

THIS MAY RESULT FROM A FRACTURE OF THE BONY LABYRINTH AFFECTING THE MEMBRANES OF THE OVAL & ROUND WINDOWS.

PERILYMPHATIC FISTULA

IN THIS CONDITION, PERILYMPH LEAKS FROM THE INNER EAR INTO THE MIDDLE EAR THROUGH A SMALL HOLE IN ONE OF THE WINDOW MEMBRANES. IT CAN SEAL ITSELF BUT MAY REOPEN AS A RESULT OF EXERTION OR CHANGES IN ATMOSPHERIC PRESSURE. EXAMPLES ARE FLYING IN AN AIRPLANE OR A VALSALVA MANEUVER SUCH AS A COUGH OR SNEEZE.

CENTRAL VESTIBULAR DISTURBANCES

THESE ARE CENTRAL NERVOUS SYSTEM DISORDERS AFFECTING THE BRAINSTEM OR ITS PROJECTIONS INTO THE TEMPORAL LOBE OF THE CEREBRAL CORTEX. THEY TEND TO HAVE OTHER NEUROLOGICAL SYMPTOMS AS WELL. THEY ARE.....

BRAINSTEM ISCHEMIA
MULTIPLE SCLEROSIS
POSTERIOR FOSSA TUMOR
BASILAR MIGRAINE

*****NEAR-SYNCOPE*****

NEAR-SYNCOPE CAN BE DEFINED AS A SENSATION OF IMPENDING FAINT. THE UNDERLYING CAUSE IS INADEQUATE BLOOD FLOW TO THE BRAIN. IT COMMONLY OCCURS IN ACUTE ATTACKS LASTING ONLY A FEW SECONDS, BUT OCCASIONALLY PATIENTS DESCRIBE CHRONIC PROBLEMS. THE NATURE OF THE PATIENT'S SYMPTOMS DEPEND ON THE MAGNITUDE OF THE BLOOD FLOW REDUCTION EXPERIENCED BY THE BRAIN. LOSS OF CONSCIOUSNESS USUALLY DOES NOT OCCUR. THIS SYMPTOM NORMALLY POINTS US TO HEART OR BLOOD VESSEL PROBLEMS. THE DISORDERS CAN BE LOOSELY GROUPED INTO 2 CATEGORIES - "PUMP" OR "PLUMBING" TROUBLES. SOME OF THE MEDICAL CONDITIONS PRECIPITATING NEAR-SYNCOPE ARE.....

CARDIAC "PUMP" DISTURBANCE

ARRHYTHMIAS
DECREASED CARDIAC OUTPUT

VASCULAR "PLUMBING" DISTURBANCE

ORTHOSTATIC HYPOTENSION
VOLUME DEPLETION
PERIPHERAL VASODILATION
PARTIAL OCCLUSION OF CAROTID ARTERIES

*******DISEQUILIBRIUM*******

HERE, THE PATIENT WILL EXPERIENCE A SENSATION OF LOSING THEIR BALANCE WITHOUT AN ILLUSION OF MOVEMENT OR IMPENDING LOSS OF CONSCIOUSNESS. HE/SHE WILL FEEL UNSTABLE, ESPECIALLY WHEN STANDING OR WALKING. PATIENTS DO NOT TYPICALLY REPORT PROBLEMS WHEN SITTING OR LYING DOWN. DISEQUILIBRIUM USUALLY OCCURS BECAUSE OF A DISRUPTION IN THE INTEGRATION BETWEEN THE PATIENT'S SENSORY INPUTS AND MOTOR OUTPUTS.
THIS SYMPTOM IS SEEN MOST OFTEN IN THE ELDERLY POPULATION. THE ABILITY OF THE NERVOUS SYSTEM TO PROCESS SENSORY INPUTS AND CONTROL POSTURAL REFLEXES DECLINES WITH ADVANCING AGE. DISEQUILIBRIUM-RELATED GAIT DISTURBANCES OF THE ELDERLY ARE PARTICULARLY ACCENTUATED BY UNFAMILIAR SURROUNDINGS AND DIM LIGHT, SO MANY OF THEM AVOID THESE SITUATIONS. THIS PROBLEM USUALLY POINTS US TOWARD A NEUROLOGICAL ETIOLOGY. UNDERLYING MEDICAL REASONS FOR DISEQUILIBRIUM ARE.....

NEUROLOGIC DISORDERS

MULTIPLE SENSORY DEFICITS
THE ELDERLY HAVE DIMINISHED PROPRIOCEPTION IN THE LEGS. THESE PATIENTS ARE PARTICULARLY UNSTEADY IN THE DARK WHERE VISUAL CUES CAN'T COMPENSATE FOR A LACK OF SENSORY INPUT. SOME CAUSES ARE PERIPHERAL NEUROPATHIES AND VITAMIN B12 DEFICIENCIES.
CEREBELLAR DYSFUNCTION
THIS MAY STEM FROM ALCOHOLIC DEGENERATION, TUMORS OR INFARCTS.
NON FUNCTIONING LABYRINTHS

EXTRA PYRAMIDAL DISORDERS
PARKINSONISM IS THE MOST LIKELY CULPRIT HERE.
DRUG INTOXICATIONS
ANTICONVULSANTS LIKE PHENOBARBITOL WILL PRODUCE ATAXIA IF THE BLOOD LEVELS ARE TOO HIGH. ALCOHOL PRODUCES UNSTEADINESS BY TEMPORARILY CHANGING THE SPECIFIC GRAVITY OF THE ENDOLYMPH. AMINOGLYCOSIDES LIKE GENTAMICIN OR TOBRAMYCIN CAN CAUSE SYMPTOMS OF DISEQUILIBRIUM. SALICYLATES LIKE ASPIRIN CAN ALSO BE A CULPRIT.

*****ILL-DEFINED LIGHTHEADEDNESS*****

THIS IS A VAGUE SYMPTOM AND PATIENTS OFTEN HAVE DIFFICULTY DESCRIBING THEIR SYMPTOMS. PATIENTS MAY REPORT A SENSATION OF GIDDINESS OR OF FEELING SPACEY. THESE PATIENTS OFTEN PLACE THEMSELVES IN A HYPERVIGILANT STATE DURING WHICH THEY MONITOR THEIR BODIES FOR MEDICAL SYMPTOMS. THIS COMPLAINT MAY BE DEFINED AS ONE OF EXCLUSION AS THE PATIENT JUST DOESN'T FIT INTO THE OTHER CATEGORIES. HERE, WE TEND TO THINK OF.....

PSYCHIATRIC DISORDERS

HYPERVENTILATION SYNDROME
ANXIETY NEUROSIS
AFFECTIVE DISORDERS

*****DIAGNOSIS*****

A CAREFUL HISTORY IS THE MOST IMPORTANT DIAGNOSTIC TOOL. IT IS VERY IMPORTANT TO OBTAIN COMPLETE AND ACCURATE INFORMATION REGARDING EXACTLY WHAT THE PATIENT EXPERIENCES WHEN THEY ARE SYMPTOMATIC. THE CLINICIAN MUST BE VIGILANT DURING THE HISTORY TAKING NOT TO LEAD THE PATIENT. THE PATIENT SHOULD RESPOND WITH THEIR OWN WORDS WITHOUT HELP FROM THE DOCTOR. IF ASKED WHETHER THEY FEEL LIGHTHEADED OR MAY FALL, MOST PATIENTS WILL RESPOND YES."WHAT DO YOU MEAN BY DIZZY?" SHOULD BE ONE OF YOUR FIRST HISTORY QUESTIONS. THEN LET THE PATIENT VERBALIZE FREELY IN THEIR ANSWER. OTHER PERTINENT QUESTIONS ARE:

WHEN DO YOU GET ATTACKS?
ARE YOU DOING ANYTHING SPECIAL PRECEDING THE ATTACK?
HOW LONG DO THEY LAST?

WHAT CAN YOU DO TO LESSEN THE SEVERITY OR STOP THE SYMPTOM?

SOME OF THE KEY PATIENT RESPONSES TO BE ALERT FOR ARE.....

PATIENT RESPONSE	SUSPECT
"I'M TILTING OR ROCKING" "THE ROOM IS SPINNING" "I'M WHIRLING OR SWAYING"	VERTIGO
"I MIGHT FAINT" "I'M LIGHTHEADED"	NEAR-SYNCOPE
"I MIGHT FALL" "MY BALANCE IS OFF"	DISEQUILIBRIUM
"I'M JUST DIZZY" "I FEEL WEIRD"	ILL-DEFINED LIGHTHEADEDNESS

*******CATEGORIZATION OF SYMPTOMS*******
PHYSICAL EXAMINATION

AFTER THE EXTENSIVE HISTORY, WE CATEGORIZE THE SYMPTOMS INTO 1 OF THE 4 MEDICAL DEFINITIONS AND MOVE ON TO THE PHYSICAL EXAM.

PHYSICAL EXAMINATION
MANY OF THESE TESTS WILL BE COMPLETED BY OTHER HEALTH CARE PROVIDERS, BUT WE CAN DO SOME OF THEM. THE MAIN SPECIALTIES INTERACTING WILL BE.....

VISUAL & OCULAR
NEUROLOGICAL
OTOLOGICAL
AUDITORY
CARDIO-VASCULAR
PSYCHOLOGICAL

SOME OF THE SPECIFIC TESTING THAT MAY BE DONE IS.....
FOR EAR DISEASE - OTOSCOPIC EXAM LOOKING FOR WAX OF PERFORATED DRUMS. HEARING TESTS. WEBER & RHINE TESTS WITH TUNING FORKS.

FOR CARDIOVASCULAR DISEASE - BP IN 3 POSITIONS, ECG, ATHEROSCLEROSIS ASSESSMENT
FOR NEUROLOGICAL DISEASE - CRANIAL NERVE WORKUP, ESPECIALLY 2 - 8 WITH THE 8TH ACOUSTIC NERVE DIVIDED INTO VESTIBULAR AND AUDITORY DIVISIONS; CALORIC STIMULATION TO INDUCE NYSTAGMUS (COWS); ELECTRONYSTAGMOGRAPHY; CEREBELLUM FUNCTIONING TESTS; NECK EVALUATION INCLUDING THE CERVICAL SPINE; NEURO-IMAGING STUDIES SUCH AS CT OR MRI SCANS; BRAINSTEM AUDITORY RESPONSE; VISUAL EVOKED POTENTIALS, ESPECIALLY WHEN LOOKING FOR MULTIPLE SCLEROSIS.
FOR ANY OF THESE DISEASE CATEGORIES - LAB STUDIES INCLUDING COMPLETE BLOOD COUNTS W/Hg & GLUCOSE; CHEST XRAYS

*******MANAGEMENT*******

PHYSICAL CORRECTIONS
SPECTACLES
REMOVE CERUMEN
AVOIDANCE OF STIMULUS

HABITUATION EXERCISES

HEAD MOVEMENTS IN BPPV
SHOW PATIENTS WHERE THE OFFENDING POSITION IS LOCATED.
VISION THERAPY
ROD GILLILAN IN EUGENE USES A SYSTEM HE DEVELOPED CALLED DYNAMIC ADAPTIVE VISION THERAPY TO TREAT PATIENTS WITH MOTION SICKNESS. IT BASICALLY IS A DESENSITIZATION PROGRAM TO THE OFFENDING STIMULI.

PSYCHOTHERAPY
PSYCHOLOGY AND PSYCHIATRY CAN TREAT THE UNDERLYING DISORDER.
HYPERVENTILATION SYNDROME
ANXIETY ATTACKS
AFFECTIVE DISORDERS

SURGERY - IT IS RARE TO REQUIRE THESE EXTREMES FOR DIZZINESS COMPLAINTS.

CARDIAC - FIX A LEAKY VALVE, CAROTID ENDARTERIECTOMY
NEUROLOGIC - REMOVE AN ACOUSTIC NEUROMA
OTOLOGIC - CLOSE A PERILYMPHATIC FISTULA

*****MEDICATIONS*****

THE MOST PREVALENT METHOD OF TREATING THESE DISORDERS IS WITH MEDICATIONS. DRUGS MAY REDUCE OR ELIMINATE THE SYMPTOMS BUT DON'T CURE THE DISORDER.

ANTIHISTAMINES - DEPRESSES THE HYPER-STIMULATED LABYRINTH FUNCTION. EFFECTIVE AGAINST NAUSEA/VOMITING OF MOTION SICKNESS AND VERTIGO.

DIMENHYDRINATE (DRAMAMINE®)
DIPHENHYDRAMINE (BENADRYL®)
MECLIZINE (ANTIVERT®)
PROMETHAZINE (PHENERGAN®) PHENOTHIAZINE

ANTICHOLINERGICS - DEPRESSES INPUT TO THE VESTIBULAR SYSTEM.

SCOPOLAMINE HBr - THIS IS AVAILABLE AS A PATCH TO WEAR BEHIND THE EAR. A POTENTIAL PROBLEM WITH THESE IS ACCIDENTAL DILATION. SOME OF YOU MAY HAVE SEEN A PATIENT WITH UNILATERAL MYDRIASIS FROM RUBBING THEIR NECK THEN WIPING THEIR CONJ. THEY CAN STAY DILATED FOR 3 DAYS.

ANTI-DEPRESSANT & ANTI-ANXIETY MEDS - TREATS THE AFFECTIVE DISORDER

AMITRIPTYLINE (ELAVIL®)
FLUOXETINE (PROZAC®)
THIORIDAZINE (MELLARIL®)
DIAZEPAM (VALIUM®)

DIURETICS - DECREASE THE FLUID RETENTION IN MÉNIÈRE'S SYNDROME.

HCTZ & TRIAMTERENE (MAXZIDE®)

*****CONCLUSION*****

QUICK CLINICAL PEARLS

SYMPTOM	SUSPECT	REFER
VERTIGO	VESTIBULAR	OTOLOGY
NEAR-SYNCOPE	CARDIO-VASCULAR	CARDIOLOGY
DISEQUILIBRIUM	NEUROLOGIC DZ	NEUROLOGY
ILL-DEFINED LIGHTHEADEDNESS	PSYCHIATRIC	PSYCHIATRY

Table 1

Characteristics of Centrally versus Peripherally Caused Vertigo

	Central	Peripheral
Duration	Long	Brief
Intensity	Moderate	Severe
Nausea/Vomiting	Mild	Moderate to Severe
Neurological Symptoms	Common	Rare
Hearing Loss	Rare	Possible
Affected by Head Position	Not usually	Frequently

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Table 2

Antihistamines Effective for Reduction of Dizziness [25,27,60]

Generic Names	Brand Name	Dosage
Dimenhydrinate	Dramamine®	50 mg TID
Diphenhydramine	Benadryl®	25-50 mg TID
Meclizine	Antivert®	25-50 mg BID
Promethazine	Phenergan®	25 mg QD

Table 3

Commonly Prescribed Anti-Depressant and Anti-Anxiety Agents[25,27,60]

Generic Name	Brand Name	Indication	Dosage
Amitriptyline	Elavil®	Depression	25 mg TID
Fluoxetine	Prozac®	Depression	20 mg QD
Phenelzine	Nardil®	Depression	15 mg TID
Thioridazine	Mellaril®	Depression	25 mg TID
Diazepam	Valium®	Anxiety	2 mg TID
Alprazolam	Xanax®	Anxiety	0.25 mg TID
Buspirone	Buspar®	Anxiety	5 mg TID

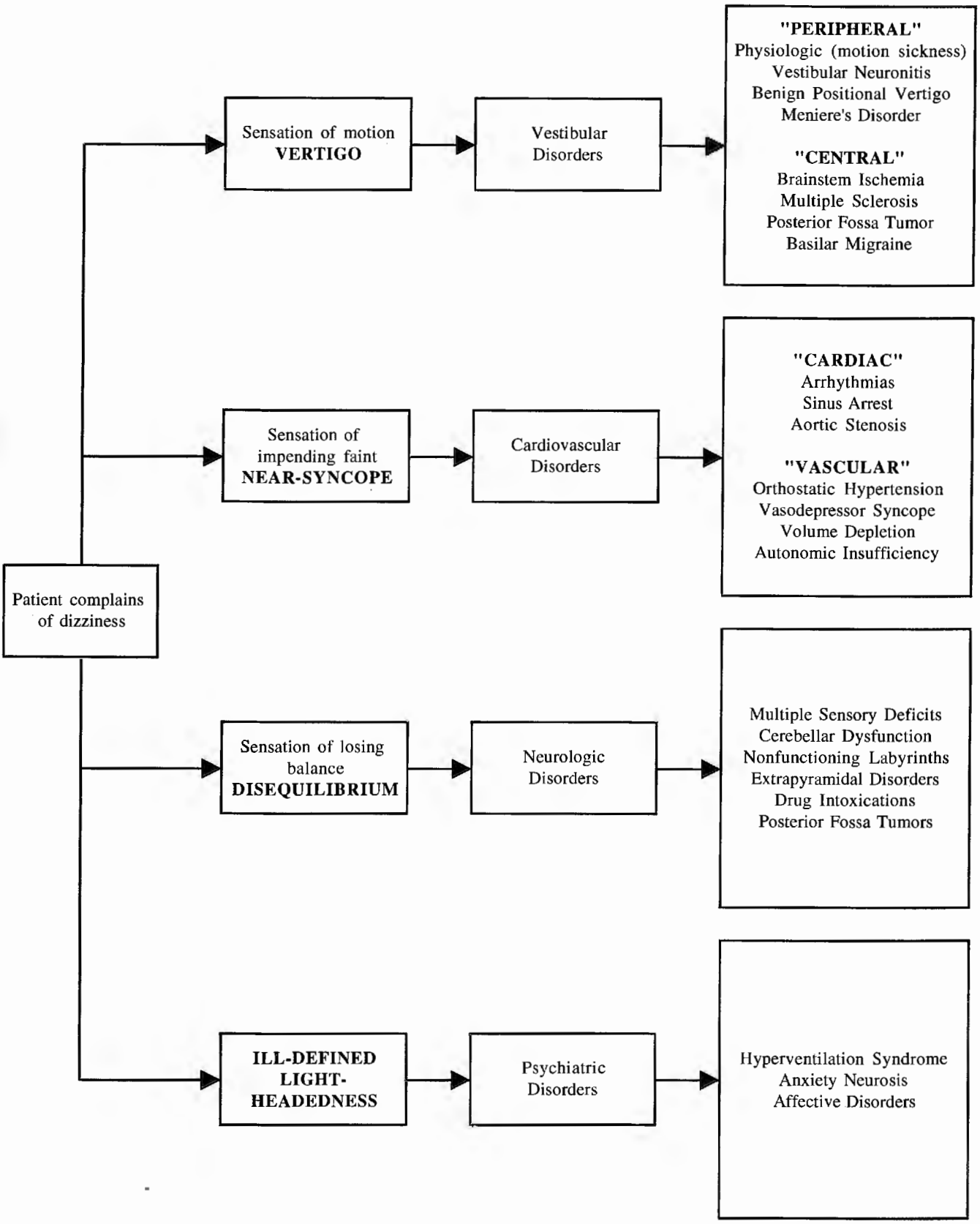

Figure 1. Dizziness Categorization. Modified with permission from Manual of Neurology: Diagnosis and Therapy, 4th edition, by MA Samuels, copyright 1991, published by Little, Brown and Company