Role of Adaptation Exercises in Clinical Practice

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Abstract

Adaptation, habituation and compensation are the mechanisms involved in rehabilitation of vertigo patients. In Vestibular Rehabilitation Therapy (VRT), patients are advised to perform a series of maneuvers involving head, eye and body movements which stimulate the in-built adaptive mechanisms. Cawthorne and Cooksey were the first to describe adaptation exercises, which are further modified. Norre has designed VRT test battery of specific exercises. Drug treatment used along with VRT should not interfere with the compensation mechanism. Anti-vertigo drug Betahistine has been shown to hasten the compensation and hence is suitable for use with VRT.

Introduction

A sense of balance is one of the most primitive faculties in man's development appearing on biological scale before the sense of sight and hearing and possibly before smell and taste. Any disturbance in this function like vertigo often leads to terror and apprehension. Patients use different terms to explain their symptom as 'vertigo', 'giddiness', 'dizziness', 'unsteadiness' etc. The term vertigo is always loosely used by patients, and sometimes also by physicians. Vertigo is defined as an illusion of movement either of the person or of the objects around the person. True vertigo is an unpleasant sensation of imbalance, spinning and disorientation. During an acute attack many patients are understandably terrified and frightened. Recurrent vertigo can be devastating to the patient's well being. Vertigo is always a symptom and never a disease. Vertigo is very

common, occurring in about 5% of the patients consulting a general practitioner and 10% of the patients examined by an Otorhinolaryngologist (Oosterveld, 1991). The reason for this high prevalence is that more than 80 different disorders are reported to have vertigo as a possible symptom. Managing a vertigo patient still remains a great challenge for clinicians due to the complexity of vestibular system, pathology of vertigo and its diagnosis.

Pathogenesis of vertigo

There are three sensory systems subserving the spatial orientation and balance - the vestibular system from vestibule and semicircular canals of the inner ear, the ocular system from the eyes and the somatosensory or proprioceptive system conveying information from periphery such as skin, joints, muscles. The information from

these three systems is integrated by central nervous system (CNS) and appropriate motor activity is generated to maintain balance and posture. When there is physiologic stimulation or pathologic dysfunction in any of the three systems, vestibular symptoms like vertigo may appear (Daroff, 1994). Generally these systems overlap sufficiently to compensate for each other's deficiencies and hence exist an inborn mechanism for stability and compensation.

Rehabilitation

Adaptation, habituation and compensation are mechanisms involved in this reparation process (Claussen, 1994)

Adaptation means a phenomenon, which helps a patient with a persisting peripheral dysfunctional state to regain normal or near normal space orientation and balance. It is not the result of one particular system but is the distributed property of the CNS. Many mechanisms contribute to this. Besides, the basic phenomenon at the level of vestibular nuclei, other systems or structures like visual systems, spinal cord, cerebellum etc. play a role.

Habituation is the repeated exposure of the system to a "mismatched" sensory input. This repetitive stimulation by the "error" situation is a fundamental mechanism in all-adaptive processes. This error will induce such changes in CNS so that undesirable effects are annihilated for a long time thus leading to compensation. Young (Norre, 1990) has defined habituation as the development of the conditioned compensatory reactions opposing inappropriate responses with visuo-vestibular conflicts and exposure to unusual motion environments.

Basic characteristics of habituation are acquisition manifested by progressive decline of Vestibulo-Ocular Reflex (VOR) response during the period of stimulation, retention manifested by the persistence of modified VOR response after a period of rest, certain specificity for habituating stimulus and finally transfer

manifested by presence of modification in responses evoked either by patterns of vestibular stimulation different from the ones used to provoke habituation or by other sensory stimulation (Norre, 1990).

Habituation and compensation are very closely related neurophysiological mechanisms. Repetition of the stimulus is the fundamental/ indispensable condition for eliciting habituation and thus to compensation.

Compensation is the goal - directed process induced by some recognized errors in the system and is directed to its elimination (Fig 1). Thus compensation will develop in a faster way when error is present.

Compensation can be explained based on two mechanisms:

1. a specific vestibular compensation wherein the unaffected/intact labyrinth will send modified responses to adjust the error situation created by affected labyrinth.
2. a non-specific mechanism wherein the visual and proprioceptive input will substitute for inappropriate vestibular input.

Static and dynamic compensation has to be distinguished. Compensation of postural deficit (tonic compensation) which includes disappearance of spontaneous nystagmus and body deviation is remarkably good. On the other hand, the recovery of dynamic reflexes, recovery of balance in movements and in rotation show deficit persisting even many months after lesion.

This concept leads to explanation of two different types of compensation - acute and chronic.

Acute compensation

In order to minimize the side to side discrepancies between firing rates in second-order neurons originating in the vestibular nuclei, acute compensation occurs initially by the influence of cerebellum and then some neurochemical changes at the level of vestibular

nuclei (Telian, 1996). This leads to acute compensation and provides relief from acute symptoms within 24-72 hours.

Even after the intense vertigo has been controlled, patient continues to suffer from motion-provoked vertigo until chronic compensation is achieved.

Chronic compensation

Chronic compensation is essential to cure motion-provoked vertigo. The system has to reestablish the symmetric tonic firing rates in vestibular nuclei and accurate responses to head movements.

The maintenance of compensatory state is an active, continuous and dynamic process in which nervous system must create a normal vestibular input. Hence vestibular compensation develops in active subjects only (Table I). Multisensory input from visual and proprioceptive systems tonically and dynamically substitutes permanently for missing or unbalanced labyrinthine input.

Activities that are commonly endorsed by patients in terms of being affected by dizziness (Beyts, 1987)

- ♦ Climbing ladder
- ♦ Walking in the dark
- ♦ Turning head suddenly
- ♦ Going out alone
- ♦ Bending to pick up things
- ♦ Using escalators
- ♦ Getting up from chair or bed
- ♦ Walking upstairs
- ♦ Playing sports
- ♦ Boarding a public transport

Decompensation

Central vestibular compensation though reliable, is sometimes fragile and an energy-dependent process. After the compensation is

apparently complete, occasional relapses of symptoms may occur due to decompensation (Fig. 1). This is triggered by period of inactivity, change in medication, extreme fatigue, intercurrent illness etc (Telian, 1996).

Thus it is very essential that the patient does not avoid the movements which provoke vertigo. But classical advice still given in few cases is "avoidance" therapy. The patient is advised 'not to move' and 'to avoid the movements eliciting vertigo' and most patients manifest this, in fact because of fear of vertigo. This avoidance therapy impedes compensation or leads to decompensation.

Rehabilitation is the opposite of "avoidance" therapy. The basic principle behind this is to stimulate the in-built adaptive mechanism in a vertigo patient by habituating him repeatedly to these vertigo attacks and thus leading to compensation. It has become an integral part of the total approach to the dizzy patient.

Vestibular Rehabilitation Therapy (VRT)

Vestibular Rehabilitation Therapy is gaining wide acceptance and increasing importance in the management of vertigo.

VRT comprises of a series of, maneuvers designed to stimulate the vestibular system. These movements which in the initial stages provoke vertigo, are combined with exercises involving eye movements and postural changes which encourage vestibular compensation.

Thus an individualized VRT is based on the following principles.

1. Vestibular habituation exercises adapted on an individual basis.
2. Stabilization of vision and co-ordination of eye movements
3. Posture control exercises
4. A program to improve general patient condition or general conditioning activities.

Patients are asked to perform the repeated head, eye, body movements that helps the brain

to calibrate the relationship between visual, proprioceptive and vestibular signals.

VRT is a safe and effective treatment modality for management of vertigo. It is non-invasive and carries a very low risk of adverse effects (apart from inducing vertigo) and relies on the acceleration of the physiological process.

The patient participates actively in the treatment process. Hence self-esteem is improved. Patient education is a very important aspect in VRT (Table II) and patient comes to realize the difference between movement provoked and spontaneous attacks of vertigo.

Adaptation exercises were first reported for patients with post concussion syndrome and other cases of dizziness by Cawthorne and Cooksey (Cooksey, 1945; Beyts, 1987; Robertson, 1997). Cawthorne discovered that a careful explanation of the physiology of the situation, coupled with the graduated series of exercises designed to encourage head and eye movements, was the optimum method of hastening recovery. The exercises by Cawthorne and Cooksey have over the years proved their worth in a range of vestibular disorders, in addition to those treated by Cawthorne and Cooksey.

A computer analogy was used to explain the role of the brain in integrating information from a number of different senses (Hecker, 1974). It was explained to patients that visual information was combined with proprioceptive feedback and vestibular information and that this was reprogrammed by the brain that acted rather like a central computer combining information from different senses. This information can be reassuring to the patient if it is realized that other undamaged sensory systems could help him to keep upright, despite a vestibular malfunction. They found that once this computer rationale for exercises is explained to motivate the patients, the success rate of self-administration is high.

Once the patient education is complete, pa-

tients are asked to perform a set of Cawthorne and Cooksey exercises (Table III). These exercises are carried out in consultation with the therapists and physicians.

Recovery of psychological status

Vertigo, dizziness or imbalance is often associated with emotional distress and a fear to move in public places.

In addition to vestibular exercises, management of the psychological status of the patient with vertigo is now recognized as being a significant factor in the recovery process (Luxon, 1997). Patients with chronic vertigo may suffer from psychological stress to their disabling state. Thus while rehabilitating chronic vertigo patients a dual approach involving management of vestibular component as well as psychiatric component is of utmost importance re-emphasizing the importance of patient education.

Role of therapist in VRT

The therapist plays a key role in VRT to educate the patient about the illness. A patient who is well educated about the nature of the vestibular disorder understands the rationale for vestibular rehabilitation. Such a patient understands that these measures are not curative but management techniques. With this attitude, patient takes an active role in the VRT. Optimally, the therapist has to understand something about the diagnostic tests as well as strengths and limitations of conventional medical and surgical treatment modalities. The therapist can modify the standard VRT and design a set of modified exercises so that activities of interest to each patient can be incorporated in individual program.

Role of physician in VRT

In recent years, otologists are realizing the importance to develop a successful program of VRT. The program of VRT is best worked out

by a team of qualified professionals - the otologists, vestibular testing personnel, and the physical therapist.

Table No. I

Do's and Don'ts in encouraging vestibular compensation

Encourage	Avoid
♦ Alertness	♦ Sedation
♦ Active & passive movement	♦ Immobility
♦ Large Support surface	♦ Dark environment
♦ Fine motor task	♦ Solitude standing
♦ Visual stimuli	
♦ General care	

A mutually supportive interaction between the otologist and the physical therapist is essential to satisfy the needs encountered by VRT. Otologists should appreciate and respect the role that therapist plays in such a challenging program.

Specificity of adequate exercises

Till now the approach to VRT was to apply the same set of exercises systematically. But experimental research and clinical experience have pointed interindividual difference in evolution of adaptation and compensation after labyrinthine lesion and hence the need for individually selected exercises. This technique emphasizes on the specific exercises that are

Table II: Instructions for patients before starting VRT

The objective for VRT is to provoke deliberately and systematically, as many spells of vertigo as can be tolerated. This is difficult for the patient to understand and accept, so his education is an essential part of the plan and active participation of the patient becomes an important factor governing the success of VRT. Patients should be explained the cause of vertigo, mechanism of adaptation and these exercises.

Patients can be educated and instructed based on the following lines.

- The balance parts of the two ears complement each other, sending equal impulses to the brain that is essential for balance. If either or both balance centers are damaged, equilibrium is lost and vertigo or giddiness is experienced, may be accompanied by nausea and vomiting. This condition may be very frightening, but it is not life threatening and not serious.
- The purpose of this VRT is to build up a tolerance mechanism in the brain, which will compensate for the disorder. The exercises advised will stimulate the in-built tolerance mechanism. Only the patient can build a tolerance in his brain to overcome the symptoms. Consider it like building muscle strength. A muscle begins to grow in strength only when exercise begins to hurt. The same is true for brain. Increasing its capacity will increase its tolerance. More regularly and repeatedly these exercises are performed, the sooner will vertigo disappear.
- Patients should be warned that their return to normal life may be gradual and will depend largely upon their ability to overcome the fatigue.
The exercises should be performed persistently for at least 5 minutes three times daily and for as long as vertigo persists and as per the medical advice. This may be for 1 to 3 months. A conscious effort should be made to seek out the head positions and movements that cause vertigo which one can tolerate, because the more frequently vertigo is induced the more quickly is the brain compensation mechanism built up. Remember that each spell brings you closer to the last, each spell is 'money in the bank'. Choose a sport activity that is difficult or demanding due to dizziness and spend time on it daily until it no longer produces any symptoms.
- Certain medications help to control vertigo while the brain compensation is being achieved and any such tablets should be taken regularly during the course of exercises with medical advice only. Diligence and perseverance will be required but the earlier and more regularly the balance exercise regime is carried out the faster and more complete will be recovery to normal activity.

(Dix, 1984, McCabe, 1970)

selected individually for each patient. Norre (1988) has designed a test battery (Table IV). This battery contained a series of maneuvers, which could induce positioning vertigo with typical rotatory nystagmus (M+ Ny+) or only vertigo without nystagmus (M+ NY -). Positive maneuvers can be advised as being adequate exercises. By applying these exercises vertigo must be elicited and maneuvers must be executed actively in a rather vivid way.

The development of compensation and adaptation occurs progressively, stimulated by repeated presentation of situation that has to be adapted. It is called error-controlled process. Therefore rehabilitation treatment should be in accordance with the features of compensatory process.

The first prerequisite for success for such a treatment is strict selection. Only a provoked type of vertigo, based on peripheral lesion with stable dysfunctional state that needs compensation and adaptation, constitutes an indication for VRT. Secondly according to principle of error control, the situations that are not yet adapted have to be presented repeatedly in order to stimulate the required adaptation. Hence selection of maneuvers eliciting vertigo is imperative.

Types of vertigo which may get benefited by VRT

Peripheral and intermediate vestibular disorders

Table III: Cawthorne - Cooksey Exercises

(A) In bed	(1) Eye movements - at first slow, then quick (a) up and down (b) from side to side (c) focusing on finger moving from 3 feet to 1 foot away from face (2) Head movements at first slow, then quick; later with eyes closed (a) bending forwards and backwards (b) turning from side to side
(B) Sitting (in class)	(1) and (2) as in (A) (3) Shoulder shrugging and circling (4) Bending forwards and picking up objects from the ground
(C) Standing (in class)	(1) Exercises (1) and (2) in (A), and (3) in (B) (2) Changing from sitting to standing position with eyes open & shut (3) Throwing a small ball from hand to hand (above eye level) (4) Throwing ball from hand to hand under knee (5) Change from sitting to standing and turning round in between
(D) Moving about (in class)	(1) Circle round center person who will throw a large ball and to whom it will be returned (2) Walk across room with eyes open and then closed (3) Walk up and down slope with eyes and then closed (4) Walk up and down steps with eyes open and then closed (5) Any game involving stooping or stretching and aiming such as skittles, bowls or basket-ball

(Dix , 1976)

Table IV: Maneuvers of the VHT test battery*

Sequence Direction			Description Change of position		
			From	To	
General					
S	M1	Midline	Sitting	Supine	
	M2	Left	Supine	Left side	
	M3	Right	Left side	Right side	
	M4	Midline	Supine	Sitting	
Standing					
E	M5	S	Turning to right		
R			Turning to left		
E					
Sitting					
I	M7	Right	R	Nose on left knee	
E					M8
Right ear on the right shoulder					
Left ear on the left shoulder					
Movements					
S	M9	E	Turning head counterclockwise		
	M10		S	Turning head clockwise	
	M11			Bending forward	
I	M12	II	From sitting to erect standing position		
	M13		Moving head for and aft		
Head hanging & turned to left					
Head hanging and turned to right					
Head hanging to midline					
Again sitting					
*VHT indicates vestibular habituation training; M1-19, sequence of maneuvers in clinical application; series I, maneuvers with possible nystagmus; series II, maneuvers where nystagmus never occurred; right, maneuver directed to the right; left, maneuver directed to the left; and midline, maneuver executed in the midline.					
(Norre, 1988)					

Benign Paroxysmal Positional Vertigo (BPPV)

Patients with BPPV seem to benefit a lot from VRT (Telian 1996). This approach is effective and is recommended for patients with this diagnosis.

Labyrinthine injuries

This includes accidental trauma in connection with labyrinthine surgery. Following labyrinthectomy or vestibular nerve section, a stable unilateral vestibular lesion is created.

Due to incomplete or delayed compensation, vestibular symptoms are seen. A routine or selective use of VRT can help in restoring the balance (Dix, 1984). Any patient who shows particularly slow recovery should be referred to customized VRT. These patients should be motivated since they have a risk for poor recovery due to complicated CNS condition, sedating drugs etc.

Labyrinthine involvement following treated chronic suppurative otitis media

Endolymphatic hydrops This condition, not only idiopathic and synonymous with Meniere's disease, may occur in association with a number of other conditions such as congenital syphilis, trauma, allergy, stenosis of internal auditory artery and endocrine insufficiency.

Central causes which get benefited by VRT

Head injury

They include concussion and skull fractures. Since these conditions include central as well as peripheral involvement, VRT technique is used as supplement to comprehensive head injury program.

Drug Intoxication

Generally imbalance caused by ototoxic drugs is transitory and disappears as the effects of the drugs wear off. Some drugs like aminoglycoside antibiotic may cause irreversible damage. Imbalance following Ototoxic drugs may get benefited by VRT.

Psychogenic disorders

Vertigo and imbalance often occurs in the absence of underlying vestibular disorder. With perseverance and encouragement successful recovery of vestibular disorder may occur by VRT and thus prolonged psychiatric treatment can be avoided.

Drug treatment with VRT

Drug treatment of vertigo has 3 goals: (1) Elimination of vertigo, (2) the enhancement or at

least non-compromise of the process of vestibular compensation, (3) Decrease in neuro-vegetative symptoms

Medication for vertigo should facilitate (or at least not hinder) vestibular compensation. Drug treatment or medications that are typically used for acute vertigo such as vestibular suppressants cause sedation and sometimes CNS depression. They may be useful in initial stages to control an acute attack of vertigo. But if used for prolonged periods, they may be counterproductive with respect to central vestibular compensation.

Some of these drugs induce decompensation or cause overcompensation in an already compensated case. Sedative effect has to be avoided when compensation and habituation have to be developed. The sedative effect of any drug is counteradvised in VRT.

Current knowledge about central compensation indicates that traditional practice of prescribing vestibular suppressants for such patients may be counterproductive. Hence once acute attacks are controlled, these drugs should be stopped.

Vestibular suppressants

Anticholinergics

- ♦ Homatropine
- ♦ Scopolamine

Benzodiazepines

- ♦ Lorazepam
- ♦ Diazepam

Antihistamines

- ♦ Diphenhydramine
- ♦ Cyclizine
- ♦ Cinnarizine
- ♦ Flunarizine
- ♦ Dimenhydrinate

Effect of many drugs on vestibular compensation so far has only been studied mainly in animals (frogs, guinea pigs, cats etc) by means of experimental model of vestibular compensation following unilateral peripheral vestibular lesions. The antivertigo drug can delay compensation, cause decompensation or facilitate compensation. Objective data in humans is generally extrapolated from animals.

- ♦ Non-Selective anticholinergics exhibit vestibular suppressive properties and also affect vestibular compensation. Reversible overcompensation has been reported in animals after anticholinergics administration. (Rascol, 1995)

Antihistamines like Cinnarizine, Flunarizine are generally classified as Calcium channel antagonists. Sedation is a common adverse effect with these drugs, which delays compensation. Cinnarizine and Flunarizine slow down vestibular compensation (Norre, 1990).

Benzodiazepines presumably act centrally to suppress vestibular response. Benzodiazepines like most sedative drugs may impair vestibular compensation. This assumption is not supported by all studies (Rascol, 1995)

- ♦ Certain drugs cause decompensation of a previously compensated vestibular lesion (Cholinomimetics, adrenergic agonists, GABA agonists). The equivalent in human pathology is poorly understood. (Rascol, 1995).
- ♦ Betahistine, the most preferred antivertigo drug worldwide effectively controls vertigo and offers dose dependent effect.

Betahistine is a histamine analogue and does not have sedative effect like the antihistamines.

"Another effect of Betahistine in balance-disorder patients is its facilitatory effect on vestibular adaptation which is very vital for speedy rehabilitation of patients who have acute peripheral vestibular failure." (Biswas, 1998).

The effect of Betahistine on compensation was studied in cats by Tighilet et al (1995). It was observed that oral Betahistine (50 or 100 mg/kg bodyweight) significantly accelerated recovery in posture and balance following transection of the left side vestibular nerve. Treated cats exhibited near normal values 20 - 25 days after vestibular lesions, compared with 35 - 40 days in a control group which did not receive Betahistine. Thus it was shown that Betahistine significantly facilitates vestibular compensation in cats.

VRT is now widely accepted as an integral part of the clinical management of vertigo. When drug treatment is to be simultaneously continued with VRT, it is important to consider the effect of antivertigo drug on vestibular compensation.

Fig 1

Fig 1: Flow diagram to illustrate compensation and decompensation in Peripheral Vestibular pathology
(Adapted from Luxon 1997)

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