Non-Surgical Treatment of Vertigo

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Majority of the cases with dizziness can be treated medically and physiotherapeutically and very few require surgical interventions. In this report one hundred cases of dizziness have been investigated in a busy otolaryngology clinic, diagnosed and treated with drugs and head and neck exercises. The above have been rationalised on a pragmatic basis as to their modes and sites of actions, interactions with various neuropeptides and recalibration of the vestibular subsystem. Above 80% of the cases were benefited by the regimens laid down in this report. An interestingly significant outcome of this study is that the post-treatment neuro-otological findings have not altered much even in those patients who have improved and resumed their normal activities.

Introduction

There is constant interaction between the 'phasic' and the 'tonus' element and besides having an afferent system, it has also an effective efferent one, a 'feed-back' and regulating mechanism, that modify both the subjective sensation of vertigo/imbalance and the objective findings (Ghosh & Kacker, 1979). The complexity of the neurones and the synapses in the vestibular 'subsystem' in the central nervous system with the co-existence of more than one neurotransmitter in a single neurone, multiple pre and post-synaptic receptors, fast and slow post-synaptic potentials, omnopause neurones, reciprocal neuronal units and action at sites distant from that of release of the transmitters enormously multiply the problems of assigning distinct functions to different neurones and neuropeptides. It has been electronystagmographically shown that affection of quick and slow-discharging neuronal units in the paramedian pontine reticular formation are associated with different distinct findings on the electronystagmograph (Ghosh & Kacker, 1979; Ghosh, 1980 & 1981). The psychological aspect of the patient complicates it further.

As a clinician, this is a humble attempt at recovering from the

'stage of being dysig, meaning stupid or foolish and putting the pragmatic aspect of non-surgical treatment of dizziness on a rational basis as to the sites and modes of actions of different drugs, their modifying effects on the neuropeptides and the effect of head and balance exercises with or without medications.

Affection of the vestibular subsystem, starting from the peripheral labyrinth to the cerebral cortex (temporal lobe and post-central gyrus) leads to dizziness. Nystagmus is the most important manifestation of vestibular disease.

Referring to Fig. 1, one can see that the fibres from the labyrinth end in the vestibular nuclei and from there they relay in the reticular core, eye nuclei (III, IV and VI), dorsal nucleus of vagus, vomiting centre and chemoreceptor trigger zone, cerebellum (flocculo-nodular lobe and fastigial nuclei) and temporo-parietal lobes of cerebral cortex. Some fibres (mossy fibres) go directly from the labyrinth via the juxtarestiform body to the cerebellum. Conversely, cerebral and cerebellar fibres converge on the vestibular nuclei exerting an inhibitory influence. Damage to these enhances vestibular responses (Ghosh & Kacker, 1979).

The neurotransmitters, associated with vestibular mechanism, are nor-adrenaline, acetylcholine & Gamma Amino Butyric Acid (Fig. 1). Vestibular receptors are cholinergic. It is likely that a cholinergic transmission to the vestibular neurones cause vertigo. The cholinergic reticulo-vestibular pathways enhance the activity within the vestibulo-ocular reflex. The mono-aminergic reticulo-vestibular pathways activated by amphetamine, has an inhibitory in-

fluence on the activity of the vestibular nuclei. Thus there are two reticulo-vestibular pathways :

(1) excitatory cholinergic and (2) inhibitory monoaminergic. The distribution of nor-epinephrine in the central nervous system is principally in the hypothalamus and brainstem reticular system (Vogt, 1954). The overall activity depends upon the balance between these transmitters (Eveaton & Goodhill, 1968). So dizziness could be due to activation of both nor-epinephrine and cholinergic reticular areas. Moreover, the interplay between the quick-discharging and slow-discharging reticular neurones and vestibular nuclei is important (Ghosh and Sen, 1970; Ghosh, 1980, 1981). Dizziness would occur when acetylcholine activation exceeds the capacity of nor-epinephrine system. These are also under the regulation of the inhibitory effects of GABA.

Habituation to motion is due to an enhanced response of the nor-epinephrine mediated system. The increased organisation of the neural system or mobilization of the enzymes necessary for increased nor-epinephrine activity would be activated by repeated exposure to motion (Wood & Graybiel, 1970, 1973). This would alter the balance of activity in the central nervous system and prevent motion sickness by allowing nor-epinephrine system to predominate over the acetylcholine system. For the nervous system to recalibrate the relationship between visual, proprioceptive and vestibular signals, repeated head and eye movements are essential. If the level of nor-epinephrine activity is raised by drugs or habituation, or if acetylcholine activity is blocked by drugs, motion sickness may be prevented.

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Material and Methods and observations

One hundred cases of vertigo were studied and managed non-surgically. The aetiologies were as follows :

Group I : Cervical spine trauma sustained 1-2 years back. The age group was between 20-35 years. There were 50 cases.

Group II : Cervical spondylosis : most of the 25 cases were above 45 years.

Group III : Cupulolithiasis—3 cases.

Group IV : Vertebro-basilar insufficiency—9 cases.

Group V : Perilymphatic fistula in the round window—2 cases. Fistulae were closed with fat graft. Mild vertigo persisted for about 3 months and subsequently treated with drugs for 3 months.

Group VI : Undiagnosed : 11 cases. Age range was 45 to 60 years.

Since facilities for conducting electroculography or optokinetic nystagmography are not available in many institutions, reasonable diagnosis can be and were made by the following :

1. History.
2. Presence and character of spontaneous nystagmus.
3. Kopfschüttel (head-shaking) nystagmus.
4. Unterberger's test (stepping with hands clasped in front).
5. Positional test.
6. Audiometry.
7. Cold caloric test with ice-cold water.
8. X-ray cervical spines, internal auditory meatus and skull, when indicated.

Table I represents the anamnesia, which has been tabulated taking the help of available literature and personal experience, is helpful in differential diagnosis. In quite a good number of cases, history alone could point to the proper diagnosis. In the group with vertebro-basilar insufficiency, the patients had only tran-

Fig. 1. VG - Vestibular ganglion, VN - Vestibular nuclei, RF - Reticular formation, Supprn - Suppression, NA - Nor-adrenaline, ACH - Acetylcholine, Activa - Activation, CTZ - chemoreceptor Trigger Zone, VC - Vomiting centre, GABA - GammaAmino Butyric Acid, MR - Med. Rectus, LR - Lateral rectus, Ant - Anterior, Post - Posterior, III & VI - Eye nuclei, Fasti - Fastigial nucleus.

sient numbness and tingling of face, especially in the perioral region. None of the cases showed any neurological deficit.

Spontaneous nystagmus was looked for in sitting and head-erect position. This was present in 5 cases of cervical spine trauma, 2 cases of

TABLE I
Dizziness (aetiology and site of pathology)

of sudden onset		Chronic			
Below 50 yrs. of age		Above 50 yrs.	Episodic	Without cochlear symptoms	Non-episodic
Without cochlear symptoms	With cochlear symptoms	(a) vascular disorders involving labyrinthine blood flow from the vertebro-basilar system.	With cochlear symptoms	Without cochlear symptoms	1. Associated with fainting or unconsciousness :—
(a) vestibular neuronitis of bacterial, viral or metabolic origin.	(a) Viral labyrinthitis (common)	(b) Strokes :—cerebral, brain-stem and cerebellar.	(a) CSOM and mastoiditis.	1. Positional :	(a) V. B. insufficiency (T.I.A.)
(b) Tabes dorsalis.	(b) Bacterial labyrinthitis (rare)	(c) Arteriosclerosis.	(b) Meniere's disease, syphilis and Cogan's disease	(a) Cupulolithiasis (BPPV)	(b) Epilepsy
	(c) Meningitis	(d) Hypertensive cardiac disease.	(c) Acoustic neurinoma & other CP-angle lesions.	(b) (Idiopathic, following head injury & vascular episodes involving the anterior vestibular artery).	(c) Orthostatic hypotension.
	(d) May follow :	(e) diabetes mellitus.	(d) Multiple sclerosis	(c) injury to cervical spines.	(d) Carotid arteriosclerosis and carotid sinus sensitivity.
	(i) Physical activity e.g., extreme exertion, sexual intercourse etc.			(d) injury to brain stem and other stem lesions including metastatic cancer.	(e) Paroxysmal cardiac arrhythmias.
	(ii) Middle ear pressure changes e.g. in flying, scuba diving etc.			(e) V.B. insufficiency usually associated with numbness of face, sensory disturbance of one or both sides of body or slurred speech.	(f) Post-inf. cerebellar artery thrombosis.
	(iii) emotional stress			(f) Non-Positional	2. Not associated with fainting/unconsciousness—
	(iv) rupture of the round window membrane and perilymphatic fistula in oval window as following stapedectomy.			(a) Drugs.	(a) psychoneurosis.
				(b) Hyperventilation.	(b) Diabetes mellitus.
				(c) Cervical spine trauma.	(c) Hypothyroidism.
				(d) Menopause.	(d) Anaemia.
				(e) Subclavian steal syndrome.	(e) Hypertension.
				(f) Migraine.	(f) Leukaemia.
					(g) Drugs (ototoxics, sedatives, tranquilisers etc.)
					(h) Motion sickness.
					(i) Dysequilibrium of ageing
					(j) Eye muscle imbalances.
					(k) Refractive error.

cervical spondylosis and 3 cases of vertebro-basilar insufficiency. This was not found to be of any localising value. Two cases of Group IV showed vertical (upwards) nystagmus implicating involvement of both MLFs (Medial Longitudinal Fasciculi). Then the head was (a) tipped backwards, (b) to the right and (c) left, over the shoulders, and (d) forwards for about one minute. Two cases in Group I and 3 cases in Group II showed horizontal rotatory nystagmus fading away within about 15-20 seconds, in b and c positions. The direction of nystagmus was not helpful in localising the lesion. Kopfschüttel nystagmus (on shaking the head sideways around a vertical axis for 20 times) was present in 25 cases with cervical spondylosis (Group II), 2 cases of V.B. insufficiency (Group IV) and 3 of the undiagnosed cases (Group VI). Head shaking produced giddiness similar to the one the patients had

been having in 5 of the above cases in Group I ; 2 of Group II ; 2 of Group III ; 2 of Group V and 2 of Group VI (both had Kopfschüttel nystagmus) and dissimilar type in 33 of Group I (including those who had Kopfschüttel nystagmus) and 4 of Group VI, (including those who had Kopfschüttel nystagmus), corroborating the widely accepted impression that the latter is suggestive of central lesion while the former, of peripheral lesion.

Unterberger's test was not of much help in localising the lesion ; but it indicated the presence of pathology in the vestibular subsystem. Positional test revealed the following : (a) non-fatiguable nystagmus with direction towards the upper ear was present in 13 cases in Group I (25% approximately). Out of these, three cases showed decline in nystagmus after fifth positioning and total fatigue on the ninth to

tenth ones. The rest showed fatiguable nystagmus towards the undermost ear, (b) in Group II, 10 cases (40%) showed non-fatiguable type nystagmus and out of these, four cases showed decline after the sixth positioning and total decline after the ninth ones, (c) in Group III, all showed fatiguable nystagmus towards the undermost ear, (d) in Group IV, five cases (about 50%) showed classical non-fatiguable type of nystagmus towards the upper ear, (e) Group V, did not show any positional nystagmus and (f) in Group VI three cases (about 25%) showed fatiguable type of nystagmus towards the undermost ear.

Audiometry was done in all the cases. The following were the positive findings : Group I— Only 3 cases showed high frequency sensorineural loss. Dynamic ranges between comfortable and uncomfortable levels were not compressed and tone-decay

test was negative. Group II—40% of them showed high frequency sensori-neural loss in both ears, suggestive of presbycusis. 30% of them showed compressed dynamic range, indicative of presence of recruitment. Group III—Audiometry showed normal hearing. Group IV—8 of them showed high frequency sensori-neural loss and 3 out of them showed bilateral tone decay, indicating neural lesion. Group V—Both cases had high frequency sensorineural loss of 30-40 dB. Group VI—8 cases showed bilateral high frequency sensori-neural loss

who were above 50 years, indicating presbycusis.

Cold caloric test was conducted on all the cases, which showed the following : Group I—17 cases showed hyperactivity, 3 hypoactivity, and 30, normal responses. Group II—11 cases showed hyperactivity and out of these 2 showed perversion of nystagmus. The rest were normal. Group III—Normal reaction., Group IV—6 cases were hyperactive and out of these, 3 showed dissociation and/or perversion of nystagmus, 2 cases showed normal reaction and

1 case showed bilateral hypofunction. Group V—Normal reaction. Group VI—6 were hypofunctioning and 5 normally functioning.

Therapy consisted of drugs and head and neck exercises. The drugs that are used in a case of vertigo can be rationally tabulated (Table-II and Fig.1). The drugs, commonly used in the present study were :

- (a) Pyridoxine 40 mg TDS.
- (b) Vasodilators : Cyclandelate - 200 mg. TDS or QID. depending on the intensity of dizziness and

TABLE—II

ANTIVERTIGINOUS DRUGS

(Based on Avery's Classification)

CLASS	ACTION	DRUGS
A. LABYRINTHINE SUPPRESSANTS		
1. Antihistamines	(1) Vest. Suppressants (Endogran). Anticholinergic (Central). Sedative + Tranquilliser. Some enhances sympathetic activity. Flunarizine acts as a Calcium — Channel Blocking Agent. Intracere- bular Calcium is regarded as The physiological link in excitation-responses coupling. Flunarizine dampens this by inhibiting Influx of Ca-Ion Into the Cells of Crista And Vestibular Neurones (i.e. It Has Both Peripheral and Central Actions)	(a) Cinnarizine 15mgm Q 6H (b) Flunarizine 15mgm Q 6H (c) Dimenhydrinate 50mgm TDS (d) Promethazine Hydrochlor 25mgm BD (e) Meclezine Hydrochlor 25mgm BD (f) Astemizole 10mgm OD
2. Antiemetics Phenothiazines	2. Suppress Central Vest. Pathways. Dopaminergic Blocking Effect. Weak Antihistamine and Anti-Cholinergic. Suppress CTZ ₁ (Esp. Prochlorperazine) Block Arousal (Reticular) by vest. Stimulation.	(a) Prochlorperazine 10mgm TDS (b) Cinnarizine (c) Promethazine Hydrochlor (d) Trifluoperazine Hydrochlor 5-10mgm. TDS (e) Trimiprazine Tartarate (f) Cyclizine (Marzine)
3. Anticholinergic	(3) Inhibit Central and Peripheral Cholinergic Pathways, Leading to Blocking of Para- Sympathetic System, Depression of Smooth Muscle Activity, Cerebral and medullary Centres.	(a) Atropine 0.4mgm I.M. (b) Scopolamine 0.6mgm I.M. (Transdermal for Prophylaxis).
4. Adrenergic	(3a) Depress CTZ + VC. Activate Reti-Vestibular Pathways. Depresses Vest. Nuclei.	(a) d-Amphetamine 10mgm BD (b) Ephedrine 25mgm TDS.
5. GABA-Mediated Inhibition	(9) Inhibitory Transmitter. GABA Formation Governed by Pyridoxine-Dependent Enzymes (Increase in Cerebellum and Brain Stem). Sodium Valproate Increases The Level of GABA in CNS by Inhibiting the Enzymatic Break Down of GABA. Good in Head Trauma.	(a) Pyridoxine 50mgm BD (b) Valproic Acid (Depakene) 15mgm/Kg/daily to 30mgm/Kg/daily in 3 divided doses.
B. VASODILATORS	(4) Improve Blood Circulation (Peripheral and Central). Nicotinic Acid Reduces Atherosclerosis Also.	(a) Histamine Diphosphate 2.75mgm n 250 ML.N/Saline, 30-60 Drops PeriMin. (b) 5% CO ₂ +95% O ₂ , Breathe For 20 mins, 2-4 Times A Day. (c) Betahistine 30-48mgm./day. (e) Cyclandelate 200mgm 6 hourly (e) Nylidrin 6mgm 8 Hourly

CLASS	ACTION	DRUGS
		(f) Papaverine 150mgm Capsule BD (.5 mgm/Kgm Body Wt.) (g) Duvadilan Retard (Isoxsuprine) 40mgm BD. (h) Nicotinic Acid (75mgm TDAC). (i) Perdilatol (Buphemine Hydrochlor) 6mgm Tab. 2TDS (j) Thymoxamine 10 mgm I.V. or 40-100 mgm Orally. (k) Caffeine 150-250mgm TDS. (l) Nafridofuryl (Praxiene) 100mgm TDS.
C. DIURETICS	(5) Decrease of Intralabyrinthine Pressure. Carbonic Anhydrase Inhibitor. Decrease Reabsorption of Na, K, CL and-HCO ₃	(a) Acetazolamide 250mgm OD For 2 Days in every 3 days. (b) Hydrochlorthiazide 25mgm 12 Hrly. (K Supplemented if Used for a Long Time. 20 M.Mol/Day).
D. PSYCHOTROPICS	(6) Modify Subjective Response to Vertigo.	
1. Antianxiety Agents	Decrease Resting Activity in Vest. Nuclei Reticular Facilitatory system. Diazepam Increases GABA-Mediated Inhibition.	(a) Diazepam 2-5mgm 8 Hourly. (b) Phenobarb 15-45 mgm 8 Hourly.
2. Antidepressants	Affect Brain Amine Levels by Intefering With Their Uptake Into Binding Sites (Tricyclic Antidepressants)	(a) Amitriptaline 25mgm TDS. (b) Nortriptyline 10mgm TDS.
E. CORTICOSTEROIDS	(7) Decrease Labryinthine Oedema + Swelling Due To Virus Infection. Lessen Damage of Acute Viral Labyinthitis if Given Within 24-48 Hrs.	Prednisolone 25-100mgm OD. Dexamethasone
F. ANTIBACTERIAL	(8) Combat Infection	Antibiotics
G. CNS-STIMULANTS	Cortex & Reticular Activity	Xanthine Derivatives e.g. Caffeine-150-250 mgm TDS.
H. CNS-METABOLIC STIMULANT	Uptake & Utilisation of Glucose & O ₂ increased in Cortex & Brainstem. Good after Head Trauma.	Pyritinol 300-600 mgm daily

Good Combinations :

- (1) Scopolamine + Dextroamphetamine—**Strongest** Antivertiginous Combination.
.6 mgm 10 mgm BD
- (2) Promethazine + „
25 mgm
- (3) „ + Ephedrine
25 mgm TDS.

Keywords :

CTZ — Chemoreceptor Trigger Zone.
VG — Vomiting Centre
CNS — Central Nervous System.

(c) pyritinol 300-600 mg. daily. Cinnarizine 25 mg. TDS was tried on 10 cases. The above were administered for two to three months. Acute exacerbations during the treatment period were managed by dimenhydrinate, prochlorperazine, and tranquilizers for a few days, depending on the responses. Head and Neck exercises were prescribed for six months or more to all the patients. Initially about 70% of the patients complain of some dizziness on performing the exercises; but they tolerate them subsequently with pleasure without any induced vertigo. Two patients had even vomiting

initially and had to discontinue the manoeuvres.

No psychotropic drugs viz. diazepam, was administered routinely since in our experience we found that this interferes with and delays the recalibrating or learning process that takes place in the reticular/centrencephalic system during the above exercises. (Ghosh and Kacker, 1979).

The results of treatment were quite encouraging and are as follows :

Group I : 40 of the cases were back to normal activities and did not

complain of dizziness; 5 cases showed improvement and the rest 5 continued to have symptoms as before, Group II : Out of 25 cases, 20 cases showed remarkable improvement. The remaining 5 cases, had improvement to the extent that they could lead reasonably normal life.

Group III : All of them become normal.

Group IV : 6 of them improved and became reasonably active. 3 did not show much improvement though they felt somewhat better.

Group V : Both the cases became normal.

Group VI : 9 of them improved very well and are leading normal life. The remaining two maintained status quo.

Post-treatment neuro-otological findings :

Spontaneous horizontal - rotary nystagmus, where present earlier, disappeared in 70% of the cases. But vertical nystagmus, noted in Group IV cases, persisted though symptomatically they improved remarkably. Initially and later they did not have any neurological deficit. In about 25% of the cases Kopfschüttel nystagmus disappeared. In rest of the cases, though there was subjective improvement, Kopfschüttel nystagmus persisted. Patients with both similar and dissimilar types of induced vertigo, following head-shaking, benefited i.e. there was no difference in their responses to the treatment between the groups with peripheral and central lesions. Similar was the behaviour with regards to fatiguable and non-fatiguable nystagmus on positioning. Both varieties persisted in all the cases, even in those who had subjective improvement. The significant observation is that the neuro-otological findings do

not show any remarkable change after non-surgical treatments, even though there is remarkable subjective improvement.

Discussion

From the present study it is obvious that the lesions in patients with dizziness quite often cannot be precisely localised, though one may have reasonable idea as to their being peripheral or central (suprasegmental) in nature. Nevertheless, disturbances eg. upset homeostasis in the blood circulation and neurotransmitters in the vestibular 'subsystem', incoming abnormal afferent impulses from the periphery (as in cervical spondylosis) to this subsystem and cerebellovestibular incoordination etc. can be effectively managed by non-surgical treatment. The drugs and their rationalised usages are legion (Fig. 1 and Table II); but one can choose a few and treat the patients with gratifying results. The modes of action have been tabulated for ready reference (Table-II) and the drugs can be safely instituted. The rationale behind head and neck exercises lie in: i) recalibrating the functional units in the central nervous system and ii) enhanced responses in the non-

epinephrine mediated system as a result of exercise. The former is substantiated by the fact that tranquilisers reduce and delay this process by depressing the reticular/centerencephalic system and hence we do not use these drugs while the patient is undergoing exercises (Ghosh and Kacker, 1979). Any patient complaining of dizziness should not be indiscriminately treated with Stemetil, Diligan, Valium etc. as a blanket cover, since this practice is not only not helpful in many cases; but contrarily, it does harm to many in the form of delaying the recalibration process and at times perpetuating the symptoms. Pyridoxin enhances the enzymatic reactions that increase the concentration of GABA which inhibits the vestibular subsystem leading to subjective improvement. Pyritinol increases the uptake and utilisation of glucose and oxygen in the cerebral cortex and brainstem and is very good for head trauma. Vasodilators bring in more nutrition to the cell of the peripheral and central vestibular apparatus. Cinnarizine inhibits the influx of calcium-ions into the vestibular cells, thus, dampening the physiological link in excitation. It also improves the blood flow to the vestibular structures, both peripheral and central.

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