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Original Research Article

Cardiovascular changes in patients presenting with vertigo

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ABSTRACT

Background: The term dizziness often used to refer to a host of complaints which present in a similar manner. Of these vertigo is the most common. A thorough knowledge of cardiovascular changes that occur in subjects presenting with peripheral vertigo will help the treating medical practioner to promptly identify the cause of symptoms and prevent any confusion that may alter the course of treatment. The aim of this study to identify the changes in cardiovascular parameters that occur in patients with vertigo.

Methods: Study was conducted at a tertiary care hospital during a period of two years during which 100 subjects where studied. Of these 50 where healthy adults which was taken as control group. Remaining 50 subjects where those who were admitted to hospital with a diagnosis of peripheral vertigo and termed as case group. Cardiovascular parameters where accessed in terms of pulse rate, rhythm, blood pressure and ECG Recordings among both the groups taken in static position and with 30 degree head tilt backwards and 45 degree angulation on either sides as is done during Dix-Hallpike's maneuver immediately, after 3 minutes and 10 minutes of positioning the patient.

Results: The study showed that there is no significant change in cardiovascular parameters in patients with vertigo when compared with healthy individuals in static position. Immediately following change in position of head there is sudden fall in pulse as well as blood pressure which then gradually comes back to baseline levels in healthy individuals. When there is a labyrinthine dysfunction in those who have hyper active labyrinth the pulse rate and Blood pressure increases sharply immediately after change in position then gradually comes back to baseline level. In hypo active labyrinth the pulse rate and blood pressure falls marginally compared to normal individuals and in dead labyrinths there is a significant fall in pulse rate and blood pressure immediately following change in position which then gradually reverts back to normal.

Conclusions: From our study it is quite clear that there are considerable changes in cardiac parameters in patients presenting vertigo which should be kept in mind while evaluating the patients to prevent misdiagnosis. The study also proves a close coordination between vestibular system and central cardiovascular regulatory mechanisms during positional changes to maintain hemodynamic stability.

Keywords: BPPV, Hemodynamic stability, Labyrinthine dysfunction, Peripheral vertigo

INTRODUCTION

Dizziness is one of the most common complaint with which patients present to health care practioners.[1] The term dizziness often used to refer to a host of complaints which present in a similar manner. This includes vertigo, light headedness and disequilibrium. Of these vertigo is the most common.[2] Vertigo by definition is a subjective perception that person himself or surroundings around him is rotating. Vertigo can be classified into two categories. 1) central vertigo and 2) peripheral vertigo. The common causes for peripheral vertigo are BPPV (

Benign paroxysmal positional vertigo), meniere's disease, vestibular neuronitis and labrynthitis.[3] Central causes for vertigo include CVA (cerebro vascular accidents), Vertebro-basilar insufficiency and space occupying lesions in the brainsteam. The general protocol for evaluation of a person presenting with vertigo includes thorough cardiovascular evaluation which includes monitoring of pulse rate and rhythm, respiratory rate, measuring Blood pressure as well as ECG recording to rule out cardio vascular causes for the presenting symptoms as sometimes cardiovascular causes can sometimes present in a similar manner.[4]

Maintenance of pulse and blood pressure during postural changes are known to involve a dynamic coordination of vestibular and cardiovascular regulatory mechanisms. The role played by the sensory cells of the vestibular system is very important in cardiovascular regulatory mechanisms during postural changes.[5] The sensory organs of the vestibular system namely macula of the utricle and saccule and the cupula of the semicircular canals detect changes in the body posture which are carried through the central pathways which in turn interplay with the central cardiovascular regulatory mechanisms to maintain a stable blood pressure and pulse rate during postural changes.[6] Studies have shown that when there is a peripheral vestibular dysfunction specially involving otolithic organ(utricle and saccule) these central regulatory mechanisms get distorted leading to changes in cardiovascular parameters which may sometime mimic a cardiovascular cause for presenting symptoms.[7] Hence a thorough knowledge of cardiovascular changes that occur in subjects presenting with peripheral vertigo will help the treating medical practioner to promptly identify the cause of symptoms and prevent any confusion that may alter the course of treatment. Hence the aim of this study to identify the changes in cardiovascular parameters that occur in patients with vertigo.

METHODS

Study was conducted at a tertiary care hospital during a period of two years during which 100 subjects where studied. Of these 50 where healthy normal adults which was taken as control group. Remaining 50 subjects where those who were admitted to hospital with a diagnosis of peripheral vertigo and termed as case group. These included patients with BPPV, Meniere's disease, vestibular neuronitis and labyrinthitis. Those with preexisting cardiovascular/ CNS disease or those who had other co morbid conditions and those already on treatment for peripheral vestibular disease where excluded from the study. Informed consent was taken from the subjects. Activity of the labyrinth in the case group was assessed by caloric stimulation. Cardiovascular parameters where accessed in terms of pulse rate, rhythm, blood pressure and ECG Recordings among both the groups taken in static position and with 30 degree head tilt backwards and 45 degree angulation on either sides as is done during Dix-Hallpike's maneuver immediately, after 3 minutes and 10 minutes of positioning the patient. The values where then compared for any difference in pulse rate, rhythm, blood pressure and ECG changes. Firstly, between case and control groups, also between the effected and non effected sides of the case study group.

RESULTS

Total 100 subjects where studied. Out of 100 subjects 50 where normal adults between age group of 20-50 years. Control group comprised of 25 males and 25 females. Case group comprised 50 subjects who where diagnosed with peripheral vertigo. Out 50 subjects in case group 28 where males and 22 where females (Table 1).

Table 1: Sex distribution.

Group	Male	Female
Control	25	25
Case	28	22

Table 2: Case group.

Condition	Male	Female	Total
BPPV	10	8	18
Vestibular Neuronitis	9	5	14
Menier's disease	4	6	10
Labrynthitis	5	3	8
Total	28	22	50

Table 3: Labyrinthine status.

Condition	Hyper reflexia	Hypo reflexia	Areflexia
BPPV	18	0	0
Vestibular neuronitis	14	0	0
Menier's disease	8	2	0
Labrynthitis	4	2	2
Total	44	4	2

Table 4: Pulse rate in static position.

Group	Mean pulse rate	Mean systolic blood pressure	Mean diastolic blood pressure
Control	78	124	84
Case	79	126	86

Table 5: Pulse rate with head turned to non affected side.

Group	Mean pulse rate immediately	Mean pulse rate after 3 minutes	Mean pulse rate after 10 minutes
Control	77	76	77
Case	78	78	79

Out of 50 subjects in case group 18 (36%) had BPPV, 14 (28%) had vestibular neuronitis, 10 (20%) had Menier's disease and 8 (16%) had labrynthitis (Table 2).

Out 50 subjects in case group that were subjected to caloric testing 44 had hypereflexia, 4 had hyporeflexia and 2 had areflexia (Table 3).

Mean pulse rate and Mean Blood pressure readings in static position in control and case group was comparable without significant difference (Table 4).

Mean pulse rate with 30 degree head tilt and head turned to 45 degree on the non affected side among case group was comparable to that in the static position without significant change immediately after positioning, after 3 minutes as well as after 10 minutes of positioning. Also, there was no significant difference in the readings between case and control group (Table 5).

Mean pulse rate with 30 degree head tilt and head turned to 45 degree on the affected side among case group showed a significant rise among the subjects who showed hypereflexia on caloric test immediately as well as after 3 minutes of positioning and the pulse rate returned to that of static position after 10 minutes of positioning.

Whereas in subjects with hyporeflexia and areflexia on caloric test there was significant fall in pulse rate immediately as well after 3 minutes of positioning and returned to baseline level after 10 minutes of positioning (Table 6).

Table 6: Pulse rate with head turned to affected side.

Group	Mean pulse rate in static position	Mean pulse rate immediately	Mean pulse rate after 3 minutes	Mean pulse rate after 10 minutes
Hyper-reflexia	68	79	75	68
Hypo-reflexia	84	70	74	84
Areflexia	85	60	67	85

Table 7: Blood pressure among controls.

Group	Mean blood pressure in static position	Mean blood pressure immediately	Mean blood pressure after 3 minutes	Mean blood pressure after 10 minutes
Control	124/84	126/84	126/84	124/84

Table 8: Blood pressure among case group with head tilted to normal side.

Group	Mean blood pressure in static position	Mean blood pressure immediately	Mean blood pressure after 3 minutes	Mean blood pressure after 10 minutes
Case	126/86	124/86	124/86	126/86

Table 9: blood pressure with head tilted to affected side in labyrinthine dysfunction.

Group	Mean blood pressure(Systolic/diast-olic) in static position	Mean blood pressure (Systolic/diastolic) immediately	Mean blood pressure (Systolic/diastolic) after 3 minutes	Mean blood pressure (Systolic/diastolic) after 10 minutes
Hyper-reflexia	122/84	138/90	130/88	122/84
Hypo-reflexia	124/88	110/68	112/70	124/88
Areflexia	130/86	106/64	110/68	130/86

Mean blood pressure in the control group with 30 degree tilt and head turned to 45 degree turned to either side showed no significant change in blood pressure compared to the blood pressure at static position (Table 7).

Mean blood pressure in the case group with 30 degree head tilt and head turned 45 degree on the non affected side was comparable to that in the static position with significant change in the blood pressure (Table 8).

Mean blood pressure with 30 degree head tilt and head turned to 45 degree on the affected side among case group showed a significant rise in subjects who showed hypereflexia on caloric test immediately as well as after 3 minutes of positioning and the blood pressure returned to that as in static position after 10 minutes of positioning. Whereas in subjects with hyporeflexia and areflexia on caloric test there was significant fall in blood pressure immediately as well after 3 minutes of positioning and returned to baseline level after 10 minutes of positioning (Table 9).

ECG findings in static position in control and case group was comparable without significant difference. No ectopics where seen (Table 10).

Table 10: ECG parameters in static position.

Group	PP interval	PR interval	RR interval	QRS complex width
Control	0.82sec	60milli sec	0.82 sec	86milli sec
Case	0.84sec	60milli sec	0.84 sec	88milli sec

Table 11: ECG parameters immediately after positioning on non affected side.

Group	PP interval	PR interval	RR interval	QRS complex width
Control	0.80 sec	80milli sec	0.80sec	86milli sec
Case	0.82 sec	80milli sec	0.82sec	88milli sec

Table 12: ECG parameters After 3 minutes of positioning on non affected side.

Group	PP interval	PR interval	RR interval	QRS complex width
Control	0.80 sec	80milli sec	0.80sec	86milli sec
Case	0.82 sec	80milli sec	0.82sec	88milli sec

Table 13: ECG parameters after 10 minutes of positioning non affected side.

Group	PP interval	PR interval	RR interval	QRS complex width
Control	0.82 sec	60milli sec	0.82sec	86milli sec
Case	0.84 sec	60milli sec	0.84sec	88milli sec

ECG recordings with 30 degree head tilt and head turned to 45 degree on the non affected side among case and control group showed significant variability in ECG parameters immediately (Table 11) as well as after 3 minutes of positioning (Table 12) compared to static position which then returned to baseline levels after 10 minutes of positioning (Table 13). There was prolonging of PR interval and a minimal reduction of RR and PP interval. Width of QRS complex remained unchanged and no ectopics where noted.

Table 14: ECG parameters immediately after positioning on affected side.

Group	PP interval	PR interval	RR interval	QRS complex width
Hyper-reflexia	0.84 sec	0.1sec	0.84sec	86milli sec
Hypo-reflexia	0.74sec	90milli sec	0.74sec	88milli sec
Areflexia	0.64 sec	80milli sec	0.64sec	86milli sec

Table 15: ECG parameters after 3 minutes of positioning on affected side.

Group	PP interval	PR interval	RR interval	QRS complex width
Hyper-reflexia	0.8 sec	0.9sec	0.84sec	86milli sec
Hypo-reflexia	0.78sec	88milli sec	0.78sec	88milli sec
Areflexia	0.7 sec	80milli sec	0.7sec	86milli sec

Table 16: ECG parametrs after 10 minutes of positioning on affected side.

Group	PP interval	PR interval	RR interval	QRS complex width
Hyper-reflexia	0.72sec	0.58sec	0.72sec	86milli sec
Hypo-reflexia	0.90sec	58milli sec	0.90sec	88milli sec
Areflexia	0.90sec	64milli sec	0.90sec	86milli sec

ECG recordings with 30 degree head tilt and head turned to 45 degree on the affected side among case and control group showed significant variability in ECG parameters immediately (Table 14) as well as after 3 minutes of positioning (Table 15) compared to static position which then returned to baseline levels after 10 minutes of positioning (Table 16).

DISCUSSION

This is a rare study which aims at studying the role of vestibular system in regulating cardiac parameters with respect to changes in posture, and the changes in cardiac parameters when there is an abnormality in the vestibular system. This study shows that Benign Paroxysmal Positional Vertigo (BPPV) is the most common type of vestibular dysfunction (Table 2). Incidence was 36% as per this study. This result is comparable with a study conducted by Hanley K and O'Dowd T where they identified Benign Paroxysmal positional vertigo (BPPV) as the most common cause for Vertigo in general practice.[7]

As evidenced by the study there is no significant change in heart rate, blood pressure or ECG parameters in patients with vertigo when compared with healthy individuals in static position. (Table 4). Immediately following change in position of head there is sudden fall in pulse as well as blood pressure which then gradually comes back to baseline levels in healthy individuals (Table 5).

When there is a labyrinthine dysfunction in those who have hyper active labyrinth the pulse rate and Blood pressure increases sharply immediately after change in position then gradually comes back to baseline level. In hypo active labyrinth the pulse rate and blood pressure falls marginally compared to normal individuals and in dead labyrinths there is a significant fall in pulse rate and blood pressure immediately following change in position which then gradually reverts back to normal.(Table 6,7,8,9).

In similar studies conducted by Emma Hallgren et al, and Baojian Xue et al, there were significant changes in cardiac parameters particularly in those having a suppressed vestibular system.[5,8]

In static position ECG parameters where comparable in normal individuals and study group (Table 10), where as there was considerable change in various ECG parameters with change in position in both study and control groups which correlated with the changes in pulse and blood pressure changes that occur with change in position (Table 11,12,13). In diseased labyrinths this change in ECG parameters was much more pronounced in all groups including hyperactive, hypoactive and dead labyrinths (Table 14,15,16). These findings are in unison with various studies showing considerable alteration in ECG patterns in patients with vestibular dysfunction.[5,8,9]

This sudden change in pulse rate and blood pressure immediately is because of the role of vestibular system which through its connection with the central cardio regulatory centers brings about immediate changes in cardiac parameters on sensing changes in position to maintain hemodynamic stability immediately after change in position as evidenced by exaggerated correction in hyperactive labyrinths, decreased activity in hypoactive labyrinths and failed compensation in dead labyrinths.[10] The baseline returning of the cardiac parameters after considerable time is due to the activity of baroreceptor mediated response to main hemodynamic stability which takes considerable time until actual change of blood pressure occurs in carotid arteries.[5]

CONCLUSION

From our study it is quite clear that there are considerable changes in cardiac parameters in patients presenting vertigo which should be kept in mind while evaluating the patients to prevent misdiagnosis. The study also proves a close coordination between vestibular system and central cardiovascular regulatory mechanisms during positional changes to maintain hemodynamic stability.

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