

Clinical Screening of Dizziness in Vertebrobasillary Insufficiency (VBI) and Benign Paroxysmal Positional Vertigo (BPPV)

Shhalini Grover¹, G.L.Khanna²

¹Associate Professor, Department of Physiotherapy, ²Professor & Dean,
Faculty of Applied Science, Manav Rachna International University

Corresponding Author: Shhalini Grover

ABSTRACT

Dizziness/Vertigo is a symptom having diverse causes. The diagnosis and treatment of patients with dizziness consist of screening examination to rule out pathologies before intervention of physical therapy management. Patients with dizziness have some common musculoskeletal findings. Holistic approach is needed to treat the cervical dysfunction as well as dizziness.

Key words- Dizziness, Vertebrobasillary insufficiency, Benign Paroxysmal Positional Vertigo

INTRODUCTION

Dizziness is considered as an abnormal sensation that affects spatial perception and stability. Dizziness is a non specific term, patients complaints it as sensations of lightheadedness, imbalance, illusory feeling of movements or disorientation. ⁽¹⁾ Historically based on symptoms quality, dizziness is categorized into one of four grades: vertigo (illusion of motion, often spinning), near syncope (feeling of impending faint), disequilibrium (loss of equilibrium when walking), and nonspecific dizziness ^(2,3) with vertigo being the most common (40-50%). ⁽⁴⁾

Vertigo is described as a sensation of self spinning or of the surroundings results from an imbalance within the vestibular system, ⁽⁵⁾ although in panic disorders it can also be triggered. Pre-syncope is an condition where the symptoms of light-headedness occurs in episodic manner and

can results from diffuse temporary cerebral ischemia. According to Sloane, ⁽³⁾ disequilibrium or imbalance can be due to abnormal movement of the legs and trunk without the involvement of any sensation inside the head. Dizziness can also be produced due to some psychological disturbances and also be one of the symptom in multiple diseases in older persons for example in cardiovascular dysfunctions, neurosensory, and psychiatric conditions. Dizziness can also occur as a side effect of multiple medications.

Ryan and Cope ⁽⁶⁾ used the term “cervical vertigo” first time and is defined as vertigo associated with neck disorders. Furman and Cass ⁽⁷⁾ described cervicogenic dizziness as a non-specific sensation which originates from abnormal afferent activity from the neck leads to disequilibrium and altered orientation in space. ^(4, 8) Thus the symptoms of imbalance and dizziness can

be associated with neck pain. ⁽⁹⁾ However, many patients of vertigo having cervical pain and tender muscles are diagnosed with vestibular disorders. Therefore before making the diagnosis, it is necessary to exclude vestibular disorders through detailed history, examination and vestibular function tests. ⁽⁴⁾ The various causes of cervicogenic dizziness are cervical trauma, cervical degenerative conditions, inflammatory or mechanical problems in the cervical spine ^(9, 10) being whiplash injury is a common traumatic problem of dizziness. It is estimated that 0.1% of the population will experience whiplash injury every year, with 12-40% having continual problems ⁽¹¹⁾ where dizziness and unsteadiness are mostly experienced (40-70%). ⁽¹²⁾ Most commonly cervicogenic factors ⁽¹³⁾ and peripheral vestibular pathology ^(14,15) exist as the causes of dizziness.

Underlying mechanism for cervicogenic dizziness

To understand the underlying mechanism of cervicogenic dizziness, the proposed hypothesis is neurovascular hypothesis, the vascular hypothesis and the somatosensory input hypothesis. ⁽⁴⁾

- a) Neurovascular hypothesis was described by Jean-Alexander Barré in 1926 and by Yong-Choen Lieou in 1928. This hypothesis suggested that mechanical irritation or compression of the sympathetic plexus surrounding the vertebral arteries are due to degenerative changes or disease in the cervical spine which further results in vasoconstriction of these arteries. This give rise to ischemia and hence dizziness. But there is very minimal effect on the normal auto-regulation of cerebral blood flow. ⁽¹⁶⁾
- b) According to the vascular hypothesis, compression of the vertebral artery can cause episodic ischemia of the brain stem or inner ear leading to vertigo. ⁽⁴⁾ A rare vascular phenomenon named Bow hunter's syndrome can result from

mechanical occlusion of vertebra basilar system. ^(17,18) The cause of this occlusion may be degenerative changes in the cervical spine ⁽¹⁹⁾ or an instability at the occipitocervical junction. ⁽²⁰⁾ The compression of vertebral artery can also occur due to the scalenus anterior and the deep cervical fascia or by a hypertrophied ligament of the scalenus anterior and the longus colli muscle through which it passes. ⁽²¹⁾ According to Heikkila, ⁽⁴⁾ the main reason for vertebrobasilar insufficiency is arteriosclerotic vascular disease which further can lead to compromised cerebrovascular circulation and thus producing the symptoms like dizziness, vertigo, nystagmus, nausea, and loss of consciousness. ^(18,19)

- c) Somatosensory hypothesis can include the cause of disturbed sensory input from the proprioceptors of the neck leading to a sensory mismatch between cervical, visual and vestibular inputs. ^(4,8) The proprioception receptors of neck which are densely present in the deep intervertebral muscles in the sub occipital region ⁽²²⁾ interact with signals of the vestibular and visual system to stabilize the eyes, the head and posture. ⁽⁴⁾ Neck pain can lead to the changed firing characteristics of these cervical proprioceptive receptors and causes dizziness.

Causes of Dizziness

There are two main causes of vertigo/dizziness, one can be central and the other is peripheral. There are many conditions which are the part of central vertigo .these may include tumors of brain stem or cerebellum, multiple sclerosis, demyelinating disease, arterio venous malfunction. And many conditions Bppv, cervicogenic vertigo, labyrinthitis, meniere's syndrome, semicircular canals trauma or infection, semicircular canal dehiscence syndrome, are in the category of peripheral vertigo. ⁽²³⁾ Vertebrobasilar insufficiency closely resembles BPPV in

terms of symptoms caused by cervical spine dysfunction in which the insufficiency is secondary to the osteophytic compression of vertebral artery. The symptoms due to VBI can be result from internal compression occurred in the cases of atherosclerosis and thromboembolism and also from external compression of cervical osteophytes, cervical fracture, dislocation, hypertonic muscle and from abnormal head posture. ⁽²⁴⁾

Clinical manifestation

Benign Paroxysmal Positional Vertigo (BPPV) is a clinical syndrome of peripheral vestibular system represented by recurrent, brief episodes of severe vertigo and rotary nystagmus triggered by specific positions of the head with respect to gravity. ⁽²⁵⁾ It is assumed that that BPPV is caused by the movement of free floating small crystals of calcium carbonate called “canaliths” localized in the semicircular canals (commonly the posterior semicircular canal) that create the sensation of vertigo through an asymmetrical activation of vestibular hair cells. ⁽²⁶⁾ During changing the position of their head rapidly, intense rotatory vertigo lasting for seconds occurs. It can be associated with episodes of nystagmus, nausea but no hearing loss or tinnitus. ⁽²⁷⁾ In recurrent cases, attacks are episodic and often occur in clusters over a period of weeks with remissions of months or years. ⁽²⁸⁾ But in VBI, the symptoms might results from degenerative cervical spine changes ⁽²⁹⁾ and cervical manipulation ⁽³⁰⁾ and the symptoms can be vertigo, unbalance, cloudy eyesight, loss of hearing, tinnitus, headache, nausea and vomiting. ⁽³¹⁻³³⁾ In this condition, there is recurrent symptomatic ischemia in the region supplied by the posterior circulation formed by vertebral arteries. ⁽³⁴⁾ The symptomology is divided into non-ischemic (local, somatic cause) and ischemic symptoms(hind brain ischemia). There is only ipsilateral posterior neck pain and occipital headache in case of non ischemic. ⁽³⁵⁾ One of the most common symptom of VBI is dizziness ⁽³⁶⁾ which occurs as an effect of neck rotation and does

not improve with repeated movement as seen in case of BPPV where there are seen reduction of vertigo with repeated neck turning. According to Lee& Kim, ⁽³⁷⁾ there are four types of BPPV, Posterior semicircular canal BPPV, Horizontal semicircular canal BPPV, Anterior semicircular canal BPPV & Mixed canal type. The main two causes for BPPV are one in which detached otolith debris could be either attached to the cupula (cupulolithiasis) or second it may be free-floating in the semicircular canals (canalithiasis). Pathological studies have shown that both of these conditions exist.

Diagnostic testing

The vertebral artery test (VAT) is the most common test which determines tolerance to cervical extension and rotation prior to manipulation or to differentiate between dizziness caused by VBI from dizziness caused by other conditions, such as benign paroxysmal positional vertigo (BPPV), an inner ear pathology where Hallpike-Dix test is performed first. In the Hallpike-Dix test and Vertebral artery test, the end position of the patient is similar and also both conditions produce dizziness, therefore differential diagnosis is necessary. So a modified VAT (mVAT) has been used to assist in the differential diagnostic process of dizzy patients and is performed in sitting. ⁽³⁸⁾ The VAT movements of coupled rotation and extension of the cervical spine can affect blood flow velocities through the vertebral artery. ⁽³⁹⁾ The vertigo in BPPV is provoked by change of head position called as positioning vertigo as compared to the vertigo of VBI where the vertigo is due to maintaining a head posture in a particular position known as positional vertigo. ⁽⁴⁰⁾ The onset of symptoms reported early in VBI lasting upto 40-50 seconds while in bppv the onset is immediate. Moreover the symptoms increases in intensity with the maintenance of head position while in BPPV there is decrease in symptoms within seconds. ⁽⁴¹⁾ BPPV, which is thought to be due to the

presence of free floating otoliths in the endolymph -stimulating fibers of the posterior semicircular canal, usually does not require imaging studies. A variety of tests have been used for determining the effect of spinal motion on vertebral artery patency. In George's test, bilateral blood pressure, pulse rates are measured and auscultate the subclavian and carotid arteries are auscultated with rotation of head right and left rotation and then in lateral side bending and the last in extension of neck in seated position (Maigne's test) and in the supine position also (DeKleijn's test). Signs of nausea, tinnitus, vertigo, light headaches, slurring of speech, dizziness or nystagmus may indicate vascular compromise or stenosis of the carotid or vertebral arteries. (42) A diagnosis of BPPV can be made by correlating historical data and clinical findings. (43,44) Patients typically complain of episodes of severe dizziness of sudden onset, precipitated by changes in head position, lasting less than a minute. Attacks may be provoked by lying down or rising out of bed, rolling over, suddenly turning the head to one side, bending forward and straightening up, or by throwing the head back and to one side as when reaching for something on a shelf. Nausea and vomiting rarely accompany the attacks. (45) There is another clinical test called "dizziness test" which distinguishes between the vertebral artery and the semicircular canals of the inner ear. In this test, the patient seated with the shoulders stabilized, now the patient rotate neck actively to left and right to the end range. Next, the head is stabilized in neutral by the examiner and the shoulders are actively rotated as far right and left as possible. If the patient experiences dizziness in both cases, the vertebral arteries may be involved. If dizziness is experience only when the head is rotated, the semicircular canals are probably involved. (46) Various techniques like MRA (magnetic resonance angiography), EMF (electromagnetic flow meter) and CDU (color Doppler Ultrasound) are used for the diagnosis of hemo-dynamic status in vertebrobasillary artery system.

Currently Color Doppler ultrasonography is the first choice in the examination because it is easy to apply, noninvasive and cheap. (32) Doppler sonography is a accepted valid and reliable tool for the evaluate of extracranial vessels and used to assess physiological alteration in blood flow. Mann and Refshauge (47) showed decrease in vertebral blood flow with neck rotation and also concluded that manual therapy affects the neck positioning hence the blood flow.

Manual Therapy

Firstly it is essential to distinguish the side effect and adverse effect of manual therapy in vertigo patients associated with VBI and BPPV. Paroxysmal positional vertigo is a self-limiting disorder and therefore considered benign. (48,49) Spontaneous recovery can be expected within weeks to months. (48) In 20-30% of patients, BPPV persists or recurs for years when untreated. (49,50) The position of cervical rotation places tension on the vertebral artery and causes relatively high proportion of injuries. Coulter 1998 recommended that cervical thrust rotation procedure is abandoned to avoid the injuries of vertebral arteries. A study by Haynes (2002) indicated that cervical rotation produced more mechanical stress on the vertebral artery than cervical lateral flexion. The results coincide with the results of various cadaver studies which also demonstrated that head rotation causes narrowing of the contralateral vertebral artery at the C1-C2 level. (51) Alternate to the rotatory manipulation, Krauss (52) have described translatoric spinal manipulation (TSM) techniques which does not affect the vertebral artery because these manipulations are small amplitude using straight line impulse parallel or perpendicular to the facet joints of vertebral motion segment. Thus the manual therapy along with vestibular rehabilitation has a great effect in improving the patients symptoms. (7) Karlberg (53) and Reid (54) also concluded in the same way that physiotherapy has an effective role in treating the vertigo and

cervicogenic dizziness symptoms. In BPPV, particle repositioning manoeuvre (PRM) based on anatomy of inner ear and pathophysiology of the BPPV has an effect on symptoms and the overall response rates are from 30% to 100%. A study conducted by Steenerson and Cronin⁽⁵⁵⁾ compared 20 BPPV patients between particle repositioning maneuver intervention with no treatment and found after 3 months that all patients relieved from symptoms after PRM as compared with no treatment group where only 25% of patients got relieved with the symptoms. The symptoms of dizziness arise from upper cervical spine require focused treatment at cervical region. Numerous manual therapy treatment including cervical mobilization, range of motion exercises, strengthening exercises, proprioceptive exercises, soft tissue mobilization and therapeutic agents are effective in improving the cervical mobility and reducing the pain due to joint restriction, muscle tightness, trigger points, increased muscle tone, poor cervical posture and impaired cervical kinesthesia. O'Leary⁽⁵⁶⁾ highlighted the relationship of pain intensity and superficial muscle activity in mechanical neck pain thus the role manual therapy comes where the manual therapy intervention affects the cervical motor control and also has impact on pain. A study done by Sterling⁽⁵⁷⁾ showed that mobilization at the level of C5/C6 decrease the activity of superficial neck muscle and facilitates the deep neck flexors which control the cranio-cervical region.

So, vertigo is symptoms and not a disease thus understanding the diagnosis on the basis of presentation of symptoms, effective management can be incorporated. The need is to distinguish between the various causes of dizziness and manage accordingly.

REFERENCES

1. Chan Y. Differential diagnosis of dizziness. Current opinion in otolaryngology & head and neck surgery. 2009 Jun 1;17(3):200-3.

2. Drachman DA, Hart CW. An approach to the dizzy patient. Neurology. 1972 Apr.
3. Sloane PD, Coeytaux RR, Beck RS, Dallara J. Dizziness: state of the science. Annals of Internal Medicine. 2001 May 1;134:823-32.
4. Heikkila H. Cervical vertigo. Grieve's modern manual therapy, the vertebral column. Edinburgh, UK: Elsevier Churchill Livingstone. 2004.
5. Baloh RW. Vertigo. The Lancet. 1998 Dec 5;352 (9143):1841.
6. Ryan GM, Cope S. Cervical vertigo. The Lancet. 1955 Dec 31;266(6905):1355-9.
7. Furman JM, Cass SP. Balance disorders: a case-study approach. Oxford University Press, USA; 1996 Jun 15.
8. Brandt T, Bronstein AM. Cervical vertigo. Journal of Neurology, Neurosurgery & Psychiatry. 2001 Jul 1;71(1):8-12.
9. Wrisley DM, Sparto PJ, Whitney SL, Furman JM. Cervicogenic dizziness: a review of diagnosis and treatment. Journal of Orthopaedic & Sports Physical Therapy. 2000 Dec;30(12):755-66.
10. Reid SA, Rivett DA. Manual therapy treatment of cervicogenic dizziness: a systematic review. Manual therapy. 2005 Feb 28;10(1):4-13.
11. Barnsley L, Lord S, Bogduk N. Comparative local anaesthetic blocks in the diagnosis of cervical zygapophysial joint pain. Pain. 1993 Oct 1;55(1):99-106.
12. Rubin AM, Woolley SM, Dailey VM, Goebel JA. Postural stability following mild head or whiplash injuries. Otolaryngology & Neurotology. 1995 Mar 1;16(2):216-21.
13. Treleaven J, Jull G, Sterling M. Dizziness and unsteadiness following whiplash injury: characteristic features and relationship with cervical joint

- position error. *Journal of Rehabilitation Medicine*. 2003 Jan 1;35(1):36-43.
14. Toglia JU. Acute flexion-extension injury of the neck. Electronystagmographic study of 309 patients. *Neurology*. 1976 Sep 1;26(9):808-.
 15. Oosterveld WJ, Kortschot HW, Kingma GG, De Jong HA, Saatci MR. Electronystagmographic findings following cervical whiplash injuries. *Acta oto-laryngologica*. 1991 Jan 1;111(2):201-5.
 16. Foster CA, Jabbour P. Barre-Lieou syndrome and the problem of the obsolete eponym. *The Journal of Laryngology & Otology*. 2007 Jul 1;121(07):680-3.
 17. Fleming JB, Vora TK, Harrigan MR. Rare case of bilateral vertebral artery stenosis caused by C4-5 spondylotic changes manifesting with bilateral bow hunter's syndrome. *World neurosurgery*. 2013 Jun 30;79(5):799-e1.
 18. Zaidi HA, Albuquerque FC, Chowdhry SA, Zabramski JM, Ducruet AF, Spetzler RF. Diagnosis and management of bow hunter's syndrome: 15-year experience at Barrow Neurological Institute. *World neurosurgery*. 2014 Nov 30;82(5):733-8.
 19. Stręk P, Reroń E, Maga P, Modrzejewski M, Szybist N. A possible correlation between vertebral artery insufficiency and degenerative changes in the cervical spine. *European archives of oto-rhino-laryngology*. 1998 Oct 8;255(9):437-40.
 20. Safain MG, Talan J, Malek AM, Hwang SW. Spontaneous atraumatic vertebral artery occlusion due to physiological cervical extension: case report. *Journal of Neurosurgery: Spine*. 2014 Mar;20(3):278-82.
 21. Dadsetan MR, Skerhut HE. Rotational vertebrobasilar insufficiency secondary to vertebral artery occlusion from fibrous band of the longus coli muscle. *Neuroradiology*. 1990 Dec 1;32(6):514-5.
 22. Kulkarni V, Chandy MJ, Babu KS. Quantitative study of muscle spindles in suboccipital muscles of human fetuses. *Neurology India*. 2001 Oct 1;49(4):355.
 23. Hain TC. Cervicogenic causes of vertigo. *Current opinion in neurology*. 2015 Feb 1;28(1):69-73.
 24. Huijbregts P, Vidal P. Dizziness in orthopaedic physical therapy practice: Classification and pathophysiology. *Journal of Manual & Manipulative Therapy*. 2004 Oct 1;12(4):199-214.
 25. Katsarkas A, Outerbridge JS. Nystagmus of paroxysmal positional vertigo. *Annals of Otology, Rhinology & Laryngology*. 1983 Mar 1;92(2):146-50.
 26. Epley JM. New dimensions of benign paroxysmal positional vertigo. *Otolaryngology--Head and Neck Surgery*. 1980 Sep 1;88(5):599-605.
 27. Lee AT. Diagnosing the cause of vertigo: a practical approach. *Hong Kong Med J*. 2012 Aug 1;18(4):327-32.
 28. Brandt T. Benign paroxysmal positioning vertigo. In *Vertigo 2003* (pp. 251-283).
 29. Vates GE, Wang KC, Bonovich D, Dowd CF, Lawton MT. Bow hunter stroke caused by cervical disc herniation: Case report. *Journal of Neurosurgery: Spine*. 2002 Jan;96(1):90-3.
 30. Arnold M, Bousser M. Clinical manifestations of vertebral artery dissection. In *Handbook on Cerebral Artery Dissection 2005 Sep 2* (Vol. 20, pp. 77-86). Karger Publishers.

31. Albuquerque FC, Dashti SR, Hu YC, Newman CB, Tebeb M, McDougall CG, Rekate HL. Intracranial venous sinus stenting for benign intracranial hypertension: clinical indications, technique, and preliminary results. *World neurosurgery*. 2011 Jun 30;75(5):648-52.
32. Özdemir H, Berilgen MS, Serhatlıoğlu S, Polat H, Ergün U, Barışçı N, Hardalaç F. Examination of the effects of degeneration on vertebral artery by using neural network in cases with cervical spondylosis. *Journal of medical systems*. 2005 Apr 1;29(2):91-101.
33. Caplan LR. Migraine and vertebrobasilar ischemia. *Neurology*. 1991 Jan 1;41(1):55-.
34. Ausman JI, Shrontz CE, Pearce JE, Diaz FG, Crecelius JL. Vertebrobasilar insufficiency: A review. *Archives of neurology*. 1985 Aug 1;42(8):803-8.
35. Millikan CH, Siekert RG. Studies in cerebrovascular disease. I. The syndrome of intermittent insufficiency of the basilar arterial system. In *Proceedings of the staff meetings*. Mayo Clinic 1955 Feb 23 (Vol. 30, No. 4, p. 61).
36. Asavasopon S, Jankoski J, Godges JJ. Clinical diagnosis of vertebrobasilar insufficiency: resident's case problem. *Journal of Orthopaedic & Sports Physical Therapy*. 2005 Oct;35(10):645-50.
37. Lee SH, Kim JS. Benign paroxysmal positional vertigo. *Journal of Clinical Neurology*. 2010 Jun 1;6(2):51-63.
38. Alshahrani A, Johnson EG, Cordett TK. Vertebral artery testing and differential diagnosis in dizzy patients. *Physical Therapy and Rehabilitation*. 2014 Jun 5;1(1):3.
39. Johnson EG, Houle S, Perez A, Lucas SS, Papa D. Relationship between the duplex Doppler ultrasound and a questionnaire screening for positional tolerance of the cervical spine in subjects with suspected vascular pathology: a case series pilot study. *Journal of Manual & Manipulative Therapy*. 2007 Oct 1;15(4):225-30.
40. Alvarenga GA, Barbosa MA, Porto CC. Benign paroxysmal positional vertigo without nystagmus: diagnosis and treatment. *Brazilian journal of otorhinolaryngology*. 2011 Dec 31;77(6):799-804.
41. van der Velde GM. Benign paroxysmal positional vertigo Part II: A qualitative review of non-pharmacological, conservative treatments and a case report presenting Epley's "canalith repositioning procedure", a non-invasive bedside manoeuvre for treating BPPV. *The Journal of the Canadian Chiropractic Association*. 1999 Mar;43(1):41.
42. Greenstein GM, editor. *Clinical assessment of neuromusculoskeletal disorders*. Mosby Incorporated; 1997.
43. Barany R. The clinical aspects and theory of train nystagmus. *Arch Augenbulk*. 1921;88:139-45.
44. Norre ME. Relevance of function tests in the diagnosis of vestibular disorders. *Clinical Otolaryngology*. 1994 Oct 1;19(5):433-40.
45. Lempert T, Gresty MA, Bronstein AM. Benign positional vertigo: recognition and treatment. *BMJ: British Medical Journal*. 1995 Aug 19;311(7003):489.
46. Magee DJ. *Orthopaedic physical examination*. 1992
47. Mann T, Refshauge KM. Causes of complications from cervical spine manipulation. *Australian Journal of Physiotherapy*. 2001 Jan 1;47(4):255-66.
48. Dumas G, Charachon R, Ghozali S. Vertige positionnel paroxystique benin. A propos de 51 observations. In *Annales d'oto-laryngologie et de*

- chirurgie cervico-faciale 1994 (Vol. 111, No. 6, pp. 301-313). Elsevier Masson.
49. McClure J, Lycett P, Rounthwaite J. Vestibular dysfunction associated with benign paroxysmal vertigo. *The Laryngoscope*. 1977 Sep 1;87(9):1434-42.
50. Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo Clinical and oculographic features in 240 cases. *Neurology*. 1987 Mar 1;37(3):371-.
51. Haynes MJ, Cala LA, Melsom A, Mastaglia FL, Milne N, McGeachie JK. Vertebral arteries and cervical rotation: Modeling and magnetic resonance angiography studies. *Journal of manipulative and physiological therapeutics*. 2002 Aug 31;25(6):370-83.
52. Krauss J, Creighton D, Ely JD, Podlowska-Ely J. The immediate effects of upper thoracic translatoric spinal manipulation on cervical pain and range of motion: a randomized clinical trial. *Journal of Manual & Manipulative Therapy*. 2008 Apr 1;16(2):93-9.
53. Karlberg M, Magnusson M, Eva-Maj M, Agneta M, Moritz U. Postural and symptomatic improvement after physiotherapy in patients with dizziness of suspected cervical origin. *Archives of Physical Medicine and Rehabilitation*. 1996 Sep 1;77(9):874-82.
54. Reid SA, Rivett DA, Katekar MG, Callister R. Sustained natural apophyseal glides (SNAGs) are an effective treatment for cervicogenic dizziness. *Manual therapy*. 2008 Aug 31;13(4):357-66.
55. Steenerson RL, Cronin GW. Comparison of the canalith repositioning procedure and vestibular habituation training in forty patients with benign paroxysmal positional vertigo. *Otolaryngology--Head and Neck Surgery*. 1996;114(1):61-4.
56. O'Leary S, Falla D, Jull G. The relationship between superficial muscle activity during the cranio-cervical flexion test and clinical features in patients with chronic neck pain. *Manual therapy*. 2011 Oct 31;16(5):452-5.
57. Sterling M, Jull G, Wright A. The effect of musculoskeletal pain on motor activity and control. *The Journal of Pain*. 2001 Jun 30;2(3):135-45.

How to cite this article: Grover S, Khanna GL. Clinical screening of dizziness in Vertebrobasillary insufficiency (VBI) and Benign Paroxysmal positional vertigo (BPPV). *Int J Health Sci Res*. 2017; 7(6):301-308.
