INTERNATIONAL JOURNAL OF CURRENT MEDICAL AND PHARMACEUTICAL RESEARCH
ISSN: 2395-6429, Impact Factor: 4.656
Available Online at www.journalcmpr.com
Volume 8; Issue 06(A); June 2022; Page No.282-286
DOI: http://dx.doi.org/10.24327/23956429.ijcmpr20220066

Research Article

A TRANSCRAINIAL DOPPLER STUDY IN 80 YOUNG HEALTHY FEMALES TO UNDERSTAND CEREBRAL HEMODYNAMICS DURING GRADED VALSALVA MANEUVERS

Jeewandeep Kaur[1] and Arvinderpal Singh Batra[2]

[1]Physiology BPSGMC for Women Khanpur Kalan, Sonepat. Haryana Pin 131305
[2]Department of Anatomy BPSGMC for Women Khanpur Kalan, Sonepat. Haryana Pin 131305

ARTICLE INFO

Article History:
Received 4th March, 2022
Received in revised form 25th April, 2022
Accepted 18th May, 2022
Published online 28th June, 2022

Key words:
Transcranial hemodynamic, young females, Valsalva maneuver, middle cerebral artery

ABSTRACT

Background: The Valsalva maneuver is commonly performed during everyday activities such as lifting, defecation and coughing, and is characterized by changes in intrathoracic pressure that have a pronounced effect on venous return, cardiac output and blood pressures. purpose of this study was to examine the cerebral hemodynamic response to graded valsalva maneuver

Objectives: To assess cerebral hemodynamics during graded valsalva maneuvers study in 80 young healthy females using transcranial haemodynamics.

Material and Methods: 80 young female participants were recruited for the study. All participants groups were free from disease and were not taking any medication. Participants were instructed to abstain from strenuous exercise, alcohol and caffeine for at least 24 h before the experimental trial. During the experimental session each participant first stood for 5min, during which baseline measures were obtained, then completed a maximal VMs for 10 second. Following recovery, relative VMs of 30 and 90% of the maximal Valsalva pressure was performed for 10 s, the order of which was randomized between participants. These relative pressures were used to demonstrate graded cerebral blood flow velocity restriction. Each VM was separated b 5 min or until values had returned to baseline. Participants were verbally instructed what pressure and duration to obtain, immediately before the performance

***Observation and Results**:* time to peak (Phase-I) corresponding to group statistics baseline-90% in all three variables MCAv_{mean}, systolic MCA, Diastolic MCA is statistically not significant. In group statistics baseline-after VM (Phase-III) nadir phase in Diastolic MCAv is statistically significant. Mean arterial pressure in group statistics baseline-90% (Phase-I) is not statistically significant but group statistics baseline-after VM (Phase-III) is statistically significant ($P<0.05$). Time to peak MCAv_{mean} and peak MAP during Phase-I of the MV was unaffected by Valsalva pressure ($P=0.410$, $P=0.107$). Time to nadir (Phase-III) falling the VM for MCAv_{mean} and MAP showed significant co-relation between intensity of theVM.

Conclusion: The study is useful to understand the mechanism cerebral hyperemic reponses may have potential implications for patient care under many clinical conditions of brain injury.

INTRODUCTION

Transcranial Doppler sonography is proved to be a suitable non invasive technique for measuring cerebral blood flow (CBF) velocity in the large cerebral basilar arteries[1]. Direct visualization of the large basilar arteries, recently performed during neurosurgical procedures, has excluded pronounced changes in caliber during fluctuating $PaCO_2$ and blood pressure. Interpretation of these results, however, has been somewhat limited, owing to study design[2]. Transcranial Doppler sonography of the basilar arteries has become a well-established method for measuring blood flow velocity[1]. So far, MR flow measurements have been used primarily for the determination of flow velocity in normal or pathologically altered carotid arteries, and have shown excellent correlation with Doppler sonographic results[3, 4]. Higher velocities obtained with MR angiography compared with transcranial doppler sonography have been reported[5]. The VM may be viewed as eliciting undesirable cardiovascular and cerebrovascular responses, but there is also evidence that it may indeed protect the cerebral circulation during phase I of the maneuver[6, 7]. Furthermore, the increased central venous pressure (CVP) experienced during a VM may reduce the pressure difference across the cerebral vascular bed[8]. Nevertheless, while these mechanical mechanisms may limit the increase in MCAv, an elevation is observed during phase I[7].This reflects the high-pass filter characteristics of the cerebral circulation[9].

Corresponding author:* **Jeewandeep Kaur
Physiology BPSGMC for Women Khanpur Kalan, Sonepat. Haryana Pin 131305

Work in animals has shown that brief cerebral ischemia (5 s) can lead to a near-maximal hyperaemic response. However, studies in healthy conscious humans exhibiting cerebral reactive hyperaemia are scarce. Whilst a hyperemic response has been suggested during the phase IV response [10], no concurrent beat-to-beat measures of CBF and oxygenation have been reported during a Valsalva maneuver.

Therefore, the purpose of this study was to examine the cerebral hemodynamic response to graded valsalva maneuver.

MATERIAL AND METHODS

80 young female participants of age ranging from 18 to 25 years were recruited for the study. Participants were informed of the likely risks and experimental procedures. Informed written consent was obtained from all the participants. A prior Ethical Approval was taken from Institutional Ethics Committee (IEC) of the BPS Govt. Medical College for women, Khanpur Kalan,Sonepat. All participants groups were free from disease and were not taking any medication. Participants were instructed to abstain from strenuous exercise, alcohol and caffeine for at least 24 h before the experimental trial. A common familiarization session was arranged for the participants. During the familiarization session the participants were familiarized with all experimental procedures and equipment, including practicing Valsalva maneuvers (VMs).

During the experimental session each participant first stood for 5min, during which baseline measures were obtained, then completed a maximal VMs for 10 second.Following recovery, relative VMs of 30 and 90% of the maximal Valsalva pressure was performed for 10 s, the order of which was randomized between participants. These relative pressures were used to demonstrate graded cerebral blood flow velocity restriction. Each VM was separated by 5 min or until values had returned to baseline. Participants were verbally instructed what pressure and duration to obtain, immediately before the performance.

Inclusion Criteria

Young Healthy Females age ranging from 18 to 25 years were included in present study.

Exclusion Criteria

Subjects with history of diabetes, cardiopulmonary disease, hypertension and chronic respiratory illness, vasovagal attack, fainting, syncope and on any medications like anti hypertensive were excluded from the study. Blood flow velocity was measured in the right middle cerebral artery (MCAv) using a 2-MHz Transcranial Doppler machine. Transcranial Doppler sonography has proved to be a suitable non invasive technique for measuring cerebral blood flow (CBF) velocity in the large cerebral basilar arteries. Blood pressure was measured manually by digital sphygmomanometer during graded valsalva maneuver (VMs). Mean blood flow velocity (MCAvmean) and mean arterial blood pressure (MAP) was calculated as the integral for each cardiac cycle divided by the corresponding pulse interval. An index of cerebral vascular conductance (CVCi) then was calculated via the equation MCAvmean/MAP. The statistical analyses of dependent variables were performed using a Student 't' Test. Data were assessed for approximation to a normal distribution and sphericity, with no corrections required. Linear regression analysis was used to determine the correlation between the phase dependent changes in MCAv, and MAP. All the data was analyzed using SPSS statistical software (Version 22), with a statistical significance set at P≤ 0.05. All data were presented as the mean ± SD.

OBSERVATION AND RESULTS

Following observations were made in group statics:
Baseline- after VM, Baseline-90% VM, Baseline-30% VM, 90%-30% VM

Baseline - 90% VM corresponds to Phase-I of the VM (Peak Phase) and Baseline-after VM corresponds to Phase-III (nadir phase). Study variables are MCAvmean, Systolic MCA, Diastolic MCA, MAP (Mean Arterial Pressure), CVC (Cerebrovascular Conductance).

All variables at the attainment of the "peak" for both the MCAvmean and MAP phase I responses were used in the analysis (i.e., a data point for each individual peak). Time to peak was taken from the start of the VM to the MCAvmean and MAP peaks independently. Each VM was analyzed for MCAvmean and MAP times and magnitude of nadir (phase III), and time to recovery from the end of the strain.

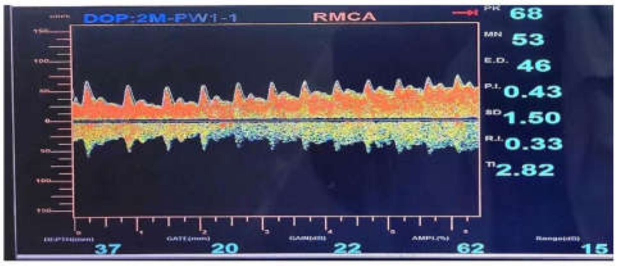

Fig 1 Recording of the Transcranial Doppler

Table no. 1 and figure no.2 is depicting changes in MCAv (Middle Cerebral Artery Blood flow Velocity) from baseline during valsalva maneuver for 30% and 90% VM and the mean difference between baseline (53.38 ± 16.38) and 30% VM (60.47 ± 21.28) is statistically significant in MCAv mean, similarly, the mean difference between 90% VM (54.99 ± 18.16) and 30% VM (60.47 ± 21.28) is statistically significant (P≤ 0.05).

Table no 1 Changes in mcav (middle cerebral artery blood flow velocity) from baseline during valsalva maneuver for 30% and 90% vm

Variables	Groups	Mean	Std. Deviation	P-Value
MCA (Mean) MN	Baseline	53.38	16.38	0.12
	after VM	49.01	12.50	
	Baseline	53.38	16.38	1.00
	90 % VM	54.99	18.16	
	Baseline	53.38	16.38	0.028
	30 % VM	60.47	21.28	
	90 % VM	54.99	18.16	0.029
	30 % VM	60.47	21.28	
Systolic MCA(PK)	Baseline	80.22	23.63	1.0
	after VM	77.89	18.56	
	Baseline	80.22	23.63	1.0
	90 % VM	78.56	25.01	
	Baseline	80.22	23.63	1.0
	30 % VM	79.62	25.99	
	90 % VM	78.56	25.01	1.0
	30 % VM	79.62	25.99	
Diastolic MCA(ED)	Baseline	38.91	15.12	0.033
	after VM	34.16	10.99	
	Baseline	38.91	15.12	1.0
	90 % VM	41.42	17.19	
	Baseline	38.91	15.12	0.525
	30 % VM	42.49	16.82	
	90 % VM	41.42	17.19	1.0
	30 % VM	42.49	16.82	

The mean difference between all group statistics in systolic MCAv (PK) is statistically non significant (P≤ 0.05). The mean difference between baseline (38.91 ± 15.12) and after VM (34.16 ± 10.99) is statistically significant (P<0.05) in Diastolic MCAv (ED).

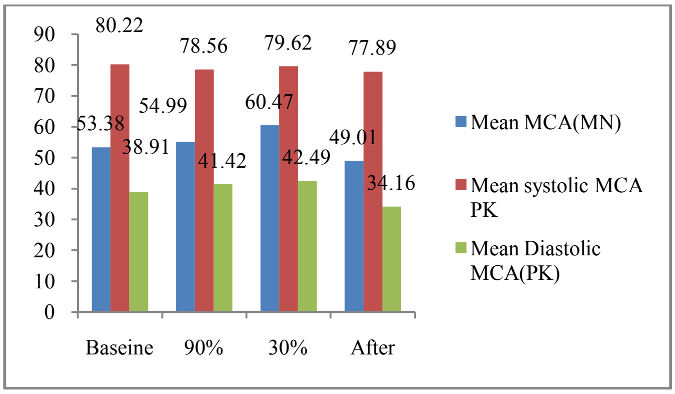

Fig. No 2 Changes in MCAv (Middle Cerebral Artery Blood flow Velocity) from baseline during valsalva maneuver for 30% and 90% VM

Table no. 2 and figure no. 3 is showing changes in Mean Arterial Pressure (MAP) from baseline during valsalva maneuver for 30% and 90% VM. The mean difference between baseline (87.06± 11.60) and after VM (80.59 ± 10.97) isStatistically Significant (P≤ 0.05) in MAP.

Table no.-2 Changes in Mean Arterial Pressure (MAP) from baseline during valsalva maneuver for30% and 90% VM

Variable	Groups	Mean	Std. Deviation	P-Value
MAP (Mean ArterialPressure)	baseline	87.06	11.60	0.684
	90%	87.87	13.99	
	baseline	87.06	11.60	0.640
	30%	84.02	14.20	
	baseline	87.06	11.60	0.001
	after	80.59	10.97	
	90%	87.87	13.99	0.280
	30%	84.02	14.20	

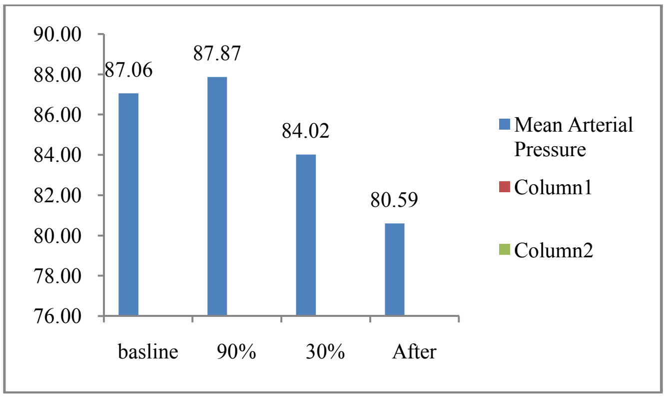

Fig No 3 Changes in Mean Arterial Pressure (MAP) from baseline during valsalva maneuver for 30% and 90% VM

Table no. 3 and figure no. 4 changes of Cerebro vascular conductance (CVC) from baseline during valsalva maneuver for 30% and 90% VM. The mean difference between baseline (0.59 ± 0.17) and after VM (0.62 ± 0.15) is statistically significant (P≤ 0.05) in Cerebro Vascular Conductance (CVC).

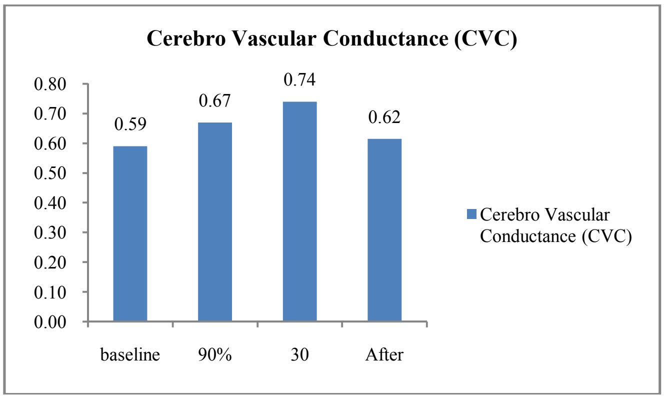

Fig No 4 Changes of Cerebro vascular conductance (CVC) from baseline during valsalva maneuver for 30% and 90% VM

Table no.-3 Changes of Cerebro vascular conductance (CVC) from baseline during valsalvamaneuver for 30% and 90% VM

Variables	Groups	Mean	Std. Deviation	P-value
Cerebro vascular conductance (CVC)	Baseline	0.59	0.17	0.003
	after VM	0.62	0.15	
	Baseline	0.59	0.17	0.070
	90 % VM	0.67	0.33	
	Baseline	0.59	0.17	0.860
	30 % VM	0.74	0.68	
	90 % VM	0.67	0.33	0.207
	30 % VM	0.74	0.68	

Comparison between Mean Arterial Pressure (MAP) and MCAvmean during Valsalva Maneuver is shown in Table no.4.

Table no.-4 Comparison between Mean Arterial Pressure (MAP) and MCAvmean during ValsalvaManeuver

Group	Variable	Mean	Std. Deviation	P-Value
baseline	MAP	87.06	11.60	0.001
	MCAvmean	53.38	16.38	
90%	MAP	87.78	13.99	0.001
	MCAvmean	54.99	18.16	
30%	MAP	84.02	14.2	0.001
	MCAvmean	60.47	21.28	
after	MAP	80.59	10.97	0.001
	MCAvmean	49.01	12.5	

- The mean difference between MAP & MCAv mean (87.06 ± 11.60) is statistically Significant (P≤ 0.05) during baseline.
- The mean difference between MAP & MCAvmean (87.78 ± 13.99) is statistically Significant (P≤ 0.05) during 90%VM.
- The mean difference between MAP & MCAvmean (84.02 ± 14.02) is statistically significant (P≤ 0.05) during 30%VM.
- The mean difference between MAP & MCAv mean (80.59 ± 10.9) is statistically significant (P≤ 0.05) after VM.

In Table-1, time to peak (Phase-I) corresponding to group statistics baseline-90% in all three variables MCAvmean, systolic MCA, Diastolic MCA is statistically not significant. In group statistics baseline-after VM (Phase-III) nadir phase in Diastolic MCAv is statistically significant. In Table-2, Mean arterial pressure in group statistics baseline-90% (Phase-I) is not statistically significant but group statistics baseline-after VM (Phase-III) is statistically significant (P<0.05). Time to peak MCAvmean and peak MAP during Phase-I of the MV was unaffected by Valsalva pressure (P=0.410, P=0.107). Time to nadir (Phase-III) falling the VM for MCAvmean and MAP showed significant co-relation between intensity of the VM. In Table-3, Cerebrovascular conductance in group statistics baseline-90% (Phase- I) is non significant and in group statistics baseline-after VM (Phase-III) is significant. All variables in group statistics 90%-30% VM were statistically non significant. In Table-4, comparison between MAP and MCAvmean of baseline, 90%, 30% and after VM were done and in all variables, it was found to be statistically significant.

DISCUSSION

We used transcranial Doppler ultrasound as a surrogate for cerebral blood flow, which provides a measure of blood flow velocity rather than absolute flow. The change in flow velocity has been found to accurately reflect changes in absolute flow as long as conduit artery diameter is unchanged[11].

In our study we found that across the range of graded Valsalva maneuver increase in Middle Cerebral Artery Velocity (MCAv) with graded Valsalva maneuver, resulted in a greater reduction in both MCA Vmean and MAP upon release of Valsalva maneuver in phase III. It is consistent with our hypothesis that the more intense Valsalva maneuver would lead to greater reduction in both MCA blood flow velocity and cortical oxygenation and that the reducation in oxygenation would be matched by an increased flow velocity in phase IV of the Valsalva maneuver. The intrathoracic pressure is rapidly translated to the cerebrospinal fluid at the onset of the Valsalva maneuver such that ICP rises[12, 13] and reduces the transmural pressure within the cerebral arteries[14]. The reduction in transmural pressure may restrain the passive dilation in response to the acute increases in cerebral perfusion pressure during phase I of the Valsalva maneuver and subsequently increases in MCAv are restrained. Furthermore, right atrial pressure increases linearly with expiratory pressure[15] and may attenuate the pressure difference across the cerebral circulation. The MAP response to Valsalva maneuver was found to be intensity dependent in phase III . The MCA vmean response also matched this response to MAP in phase III. Despite similar increases in MAP during phase I and IV, the MCAv increase is greater during phase IV[7] where ICP would be expected to be declining to near baseline levels[13.] Other mechanisms of regulation, such as the autonomic nervous system, may operate during these latter phases (phase IV) of the Valsalva maneuver to regulate cerebral blood flow[10.] This shows that Valsalva maneuver both challenges and contribute to the regulation of cerebal blood flow. MAP and MCAV changes seen in phase III were dependent on intensity. More intense Valsalva maneuvers produce a greater reduction in cerebral blood flow velocity[16]. This rapid reduction in MAP is likely attributable to the passive effect of intrathoracic pressure on the arteries[7, 17]and the refilling of the distended pulmonary vessels[8]. The elevated venous pressure during a Valsalva maneuver will increase cerebral blood volume[8, 18] and reduce venous outflow and contribute to the reduction in MCAv. This reduced flow, however, will be partially mitigated via an increase in arterial oxygen extraction[19]. The brain demonstrates high-pass filter characteristics, with high frequency oscillations in MAP being translated to the cerebral circulation[9]. Whilst the increase in CBF during phase I may be restrained by the mechanical increase in ICP and subsequent reduction in transmural pressure[13] changes in flow velocity only when conduit artery diameter is unchanged[11] which appears to be true during moderate changes in MAP[8]. Further, the retest reliability has been shown to be strong during repeated VMs using transcranial Doppler[20]. The Valsalva maneuver is the part of our everyday physiological activities and it causes changes in the cerebral hemodynamics, hence a detailed understanding of Valsalva maneuver may provide information to identify brain disease and their risk factors.

SUMMARY

The valsalva maneuver is a normal activity performed normally by every individual during every day physical activity (lifting, coughing etc.). During valsalva maneuver intrathoracic pressure increases which causes changes in cerebrovascular perfusion. In our study we found that valsalva maneuver is a very useful activity and it can be applied clinically in conditions where decreased cerebrovascular perfusion is needed (Strock, Embolism). We assessed the role of various parameters like Mean Arterial Pressure (MAP), Middle Cerebral Artery mean blood flow velocity (MCAvmean). CerebroVascular Conductance (CVC) on graded valsalva maneuver using transcranial Doppler machine (2 - MHz). It was found that graded valsalva maneuver would result in greater reduction in both middle cerebral artery blood flow velocity and cortical oxygenation and it can protect from brain injury. This information can be applied clinically and may be useful to prevent severe brain damage and injury.

CONCLUSION

In our study we examined the cerebral hemodynamic response to graded Valsalva maneuver and we found that the more intense Valsalva maneuvers would result in greater reductions in both middle cerebral artery blood flow velocity and cortical oxygenation, and that the reduction in oxygenation would be matched by an increased flow velocity in phase IV of the valsalva maneuver. So, Valsalva maneuver may protect the brain from hyper perfusion injury. The study is useful to understand the mechanism cerebral hyperemic reponses may have potential implications for patient care under many clinical conditions of brain injury. The information obtained may be utilized for assessment and management of neurological disorders.

References

1. Aaslid R, Markwalder TM, Nornes H. Non-invasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 1982;57:769–774.
2. Giller CA, Bowman G, Dyer H, Mootz L, Krippner W. Cerebral arterial diameter duringchanges in blood pressure and carbondioxide during craniotomy. Neurosurgery1993;32:737–742.
3. Fu¨rst G, Sitzer M, Hofer M, Steinmetz H, et al. Quantification of carotid blood flow velocity using MR phase mapping. J Comput Assist Tomogr 1994;18:688–696.
4. Bendel P, Buonocore E, Bockisch A, Besozzi MC. Blood flow in the carotid arteries:quantification by using phase-sensitive MR imaging. AJR Am J Roentgenol 1989;152:1307–1310.
5. Mattle H, Edelman RR, Wentz KU, Reis MA, Atkinson

DJ, Ellert T.Middle cerebralartery: determination of flow velocities with MR angiography. Radiology 1991;181:527–530.

6. Niewiadomski, W., W. Pilis, D. Laskowska, A. Gazsiorowska, G. Cybulski, and A. Strasz. 2012. Effects of a brief Valsalva manoeuvre on hemodynamic response to strength exercises. Clin. Physiol. Funct. Imaging 32:145–157.
7. Tiecks, F. P., Lam, A. M., Matta, B. F., Strebel, S., Douville, C., and Newell, D. W. (1995). Effects of the Valsalva maneuver on cerebral circulation in healthy adults: a transcranial Doppler study. Stroke 26, 1386–1392.
8. Pott, F., van Lieshout, J. J., Ide, K., Madsen, P., and Secher, N. H. (2000). Middle cerebral artery blood velocity during a Valsalva maneuver in the standing position. J.Appl. Physiol. 88, 1545–1550
9. Zhang, R., J. H. Zuckerman, C. A. Giller, and B. D. Levine.1998. Transfer function analysis of dynamic cerebral autoregulation in humans. Am. J. Physiol. Heart Circ.Physiol. 274:233–241.
10. Zhang, R., Crandall, C. G., and Levine, B. D. (2004). Cerebral hemodynamics during the Valsalva maneuver insights from ganglionic blockade. Stroke 35, 843–847.
11. Valdueza JM, Balzer JO, Villringer A, Vogl TJ, Kutter R, and Einhaupl KM. Changes in blood flow velocity and diameter of the middle cerebral artery during hyperventilation: assessment with MR and transcranial Doppler sonography. AJNR AmJ Neuroradiol 18: 1929–1934, 1997.
12. Hamilton, W., R. Woodbury, and H. Harper. 1944. Arterial, cerebrospinal and venous pressures in man during cough and strain. Am. J. Physiol. 141:42–50
13. Greenfield, J. C., J. C. Rembert, and G. T. Tindall. 1984. Transient changes in cerebral vascular resistance during the Valsalva maneuver in man. Stroke 15:76–79.
14. Haykowsky, M. J., Eves, N. D., Warburton, D. E., and Findlay, M. J. (2003). Resistance exercise, the Valsalva maneuver, and cerebrovascular transmural pressure.Med. Sci. Sports Exerc. 35, 65–68.
15. Korner, P., A. Tonkin, and J. Uther. 1976. Reflex and mechanical circulatory effects of graded Valsalva maneuvers in normal man. J. Appl. Physiol. 40:434–440.
16. Van Lieshout, J. J., Wieling, W., Karemaker, J. M., and Secher, N. H. (2003). Syncope, cerebral perfusion, and oxygenation. J. Appl. Physiol. 94, 833–848.
17. Dawson, S. L., R. B. Panerai, and J. F. Potter. 1999. Critical closing pressure explainscerebral hemodynamics during the Valsalva maneuver. J. Appl. Physiol. 86:675–680.
18. Gisolf, J., Van Lieshout, J., Van Heusden, K., Pott, F., Stok, W., and Karemaker.J. (2004). Human cerebral venous outflow pathway depends on posture and central venous pressure. J. Physiol. 560, 317–327.
19. Trangmar, S. J., Chiesa, S. T., Stock, C. G., Kalsi, K. K., Secher, N. H., and Gonzálezp–Alonso, J. (2014). Dehydration affects cerebral blood flow but not its metabolic rate for oxygen during maximal exercise in trained humans. J. Physiol.
20. Wallasch, T. M., and Kropp, P. (2012). Cerebrovascular response to valsalva maneuver: methodology, normal values, and retest reliability. J. Clin. Ultrasound 40, 540–546.

How to cite this article:

Jeewandeep Kaur and Arvinderpal Singh Batra (2022) ' A Transcrainial Doppler Study In 80 Young Healthy Females To Understand Cerebral Hemodynamics During Graded Valsalva Maneuvers', *International Journal of Current Medical and Pharmaceutical Research,* 08(06), pp 282-286.

****