An Evaluation Of Variations In The Cerebral Oxygenation and Cerebral Blood Flow Velocity Indices In Various Neurosurgical Positions (Supine, Prone & Lateral) In Elective Neurosurgical Procedures – An Observational Study

Dissertation submitted for the partial fulfilment for the Requirement of The degree

of

DM Neuroanesthesia

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DECLARATION

I hereby declare that this thesis titled "**An Evaluation Of Variations In The Cerebral Oxygenation and Cerebral Blood Flow Velocity Indices In Various Neurosurgical Positions (Supine, Prone & Lateral) In Elective Neurosurgical Procedures – An Observational Study**" has been prepared by me under the capable supervision and guidance of Dr Manikandan. S, Professor & Head, Division of Neuroanesthesia and Neurocritical Care, Department of Anesthesiology, Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram.

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This is to certify that this thesis titled **"An Evaluation Of Variations In The Cerebral Oxygenation and Cerebral Blood Flow Velocity Indices In Various Neurosurgical Positions (Supine, Prone & Lateral) In Elective Neurosurgical Procedures – An Observational Study"** is a bonafide work of Dr. Ashutosh Kumar, DM Neuroanesthesia Resident, and has been done under my direct guidance and supervision at Sree Chitra Tirunal Institute for Medical Sciences & Technology (SCTIMST), Thiruvananthapuram. He has shown keen interest in preparing this project.

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ABBREVIATIONS

ACA	**Anterior Cerebral Artery.**
ANOVA:	**Analysis of variance.**
ASA:	**American Society of Anesthesiology.**
CBF:	**Cerebral blood flow.**
CBV:	**Cerebral blood volume.**
CMR:	**Cerebral Metabolic Rate.**
CO:	**Cardiac output.**
COPD:	**Chronic obstructive pulmonary disease.**
CPAP:	**Continuous positive airway pressure.**
CPP:	**CPP: Cerebral perfusion pressure.**
CVP:	**CVP: Central venous pressure.**
DBP:	**Diastolic blood pressure.**
DCS:	**Diffusion correlation spectroscopy.**
DM:	**Diabetes mellitus.**
ECG:	**Electrocardiogram.**
ETCO2:	**End-tidal carbon-di-oxide.**
EVD:	**External ventricular drainage.**
FiO2:	**Fraction of inspired oxygen.**
FRC:	**Functional residual capacity.**
FV:	**Flow velocity.**
GCS:	**Glasgow coma scale.**
HOB:	**Head of bed.**
HHb:	**Deoxyhemoglobin.**
HbO2:	**Oxyhemoglobin.**
HR:	**Heart Rate.**
HDT:	**Head-down tilt.**
HUT:	**Head-up tilt.**
ICA:	**Internal carotid artery.**

ICP:	**Intracranial pressure**
ICU:	**Intensive care unit**
IJV:	**Internal jugular vein**
MAC:	**Minimum alveolar concentration.**
MAP:	**Mean Arterial Pressure.**
MCA:	**Middle cerebral artery.**
MFV:	**Mean flow velocity.**
MHZ:	**Megahertz.**
NIBP:	**Non-invasive blood pressure.**
NIR:	**Near-Infrared.**
NIRS:	**Near-Infrared Spectroscopy.**
NIRS (L):	**Near-Infrared Spectroscopy on left side.**
NIRS (R):	**Near-Infrared Spectroscopy on right side.**
NSICU:	**Neuro surgical intensive care unit.**
NICU:	**Neuro Intensive Care Unit.**
PaCO2:	**Partial pressure of carbon-dioxide in arterial blood.**
PbtO2:	**Partial pressure of Brain tissue oxygen.**
PCA:	**Posterior cerebral artery.**
PEEP:	**Positive end expiratory pressure.**
PET:	**Positron Emission Tomography.**
PI:	**Pulsatility index.**
PICC:	**Peripherally inserted central line.**
PIP:	**Peak airway pressure.**
PSV:	**Peak systolic velocity.**
PVR:	**Pulmonary vascular resistance.**
PWD:	**Pulsed wave doppler.**
rCBF:	**Relative Cerebral Blood Flow.**
rCMRg:	**Regional metabolic rate for glucose.**
RBC:	**Red blood cells.**
RI:	**Resistance index.**
PI:	**Pulsatality Index.**

RR: **Respiratory Rate.**

rSCO2: **Regional cerebral oxygen saturation.**

SAH: **Sub arachnoid hemorrhage.**

SaO2: **Arterial oxygen saturation.**

SBP: **Systolic blood pressure.**

ScO2: **Cerebral oxygen saturation.**

SjO2: **Jugular bulb oxygen saturation.**

SD: **Standard Deviation.**

SpO2: **Peripheral arterial oxygen saturation.**

SPSS: **Statistical package for the social sciences.**

SV: **Stroke volume.**

SVR: **Systemic vascular resistance.**

TCD: **Transcranial Doppler.**

TLC: **Total lung capacity.**

TSI: **Tissue saturation index.**

V/Q: **Ventilation-perfusion.**

VR: **Venous return.**

WHO: **World Health Organization.**

XeCT: **Xenon-enhanced computed tomography.**

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ABSTRACT

ABSTRACT

Background: Various positions used in neurosurgical procedures can compromise the cerebral oxygenation and cerebral blood flow velocity. This can cause perturbations in intracranial milieu in addition to the effects of anesthetic drugs. No previous study has evaluated effects of positioning on cerebral oxygenation as well as cerebral blood flow velocity concomitantly. We aimed to use near-infrared spectroscopy (NIRS) and Transcranial Doppler (TCD) Mean flow velocity (MFV) to study the effects of positioning (supine, prone, and lateral) on cerebral oxygenation and cerebral blood flow velocity in patients undergoing neurosurgical procedures.

Materials and Methods: After IEC approval, consented ASA I and II patients with GCS of 15 undergoing elective neurosurgery were included. NIRS & TCD MFV of MCA & ACA were recorded on both sides at three different time intervals (before induction of anesthesia (T0), 10 minutes after induction of anesthesia (T1), and 10 minutes after final positioning (T2)). A total of sixty patients were included in the three groups, that is, supine, lateral, and prone (20 each).

Results: All 3 groups were comparable in terms of baseline characteristics (demographics, systemic hemodynamics, NIRS and TCD MFV values). The NIRS values bilaterally were decreased insignificantly with maximum fall seen in lateral (3.89%) and prone (3.68%) positions and least with supine (1.14%). Post induction all 3 groups demonstrated a uniform reduction in MFVs in MCA & ACA. However, post final positioning MCA & ACA flow velocities showed a significantly greater decrease in prone group (41.6%), compared to supine (32%) and lateral (30.7%) groups. The decrease in TCD flow was very significant despite minimal decrease in Mean arterial pressure (MAP) or Heart rate (HR) (<10 % decrease in HR & MAP was seen).

Conclusion: Prone position causes significant changes in cerebral blood flow velocities compared to supine or lateral position under anesthesia. The TCD flow velocity changes were comparable in supine and lateral groups.

Thus, TCD can be a useful and feasible adjunct to NIRS for measuring positioning related changes in CBF during neurosurgical procedures, especially those done in prone position.

1.Introduction

Introduction

Successful conduct of neurosurgical procedures necessitates appropriate patient situating for better careful access. Appropriate surgical position is picked dependent on the area of the lesion, the careful surgical strategies and approach to the lesion of interest. Even however six essential body positions and their alterations are generally used to work with neurosurgical techniques three most regularly utilized situations in neurosurgery are, recumbent, horizontal, and prone. (1) The supine position is best utilized for better surgical access to anterior approach to spine and frontal lobe pathologies. (1) The lateral position being used commonly for lesions of temporal lobe, parieto-occipital cortex and posterior fossa surgeries especially involving cerebello-pontine angle tumors. The prone position is best utilized for posterior approach to the spine and posterior fossa surgeries (1).

The essential objective of perioperative neurosurgical positioning is to give better working conditions during the surgical procedure, providing adequate surgical exposure and maintaining cerebral hemodynamics and to keep up with typical body arrangement without causing injury. Numerous physical and physiological changes can happen to the patient due to surgical positioning. The position incited changes during neurosurgery can complexly affect the systemic and cerebral hemodynamics. (2)

CBF is monitored noninvasively using Transcranial doppler whereas cerebral oxygenation is measured using near infrared spectroscopy (NIRS) and brain tissue oxygen content (PbtO2). Cerebral oximetry is arising as a screen of cerebral perfusion with boundless application in many kinds of surgical procedure.

In neurosurgical patients these changes may be even more apparent since many of the patients frequently have increased ICP (intracranial pressure), which further reduces CPP. Venous

obstruction can be a major cause with significant consequences in lateral and prone positions, further exaggerated by acute flexion/rotation of arterial and venous channels like the jugular obstruction with head rotation. All these can have impact on CBF and ICP which can have bearing on cerebral oxygenation. (3)

Murphy et al. in their study found that the incidence of cerebral oxygen desaturation was higher in the patients undergoing shoulder surgery in beach chair position when compared to lateral decubitus position. (4) Similarly, Moerman et al. in their study in patients undergoing elective shoulder surgery under general anesthesia in the beach chair position found a relative decrease in cerebral oxygenation of more than 20% occurring in 80% of patients when the beach chair position was adopted. (5) They concluded a high prevalence of significant cerebral oxygen desaturation during shoulder surgery in the upright position in their study These studies have demonstrated the need for close monitoring for cerebral desaturation. NIRS might constitute a valuable technique to detect cerebral hypoperfusion in high-risk group of patients.

Just as NIRS monitors cerebral oxygenation, cerebral blood flow monitoring could be added to NIRS monitoring because NIRS is a regional cerebral oxygenation monitor, and it will be complemented by a global monitoring such as flow velocity (as a surrogate to cerebral blood flow) by Transcranial Doppler (TCD). Combining both modalities together can provide more information and useful to detect alterations during neurosurgery compared one individually. Previously done studies have demonstrated changes in TCD flow velocities of MCA (Middle Cerebral Artery) with positioning. (6) However, there is a paucity of data on the alterations in on cerebral oxygenation & blood flow velocities in neurosurgical population due to positioning under anesthesia. Since such alterations in CBF and oxygenation if occurs can impact the perioperative

management, it is required to evaluate the effects of commonly used neurosurgical positions namely supine, prone and lateral positions advocated in neurosurgery.

2.Review Of Literature

Review Of Literature

One of the essential objectives of neuroanaesthesia in the perioperative period is to give ideal position during neurosurgical procedures which would keep up with CBF, cerebral oxygenation and work with satisfactory venous drainage while forestalling any increment in ICP. A significant worry during positioning for the neuroanesthesiologist is aligning of the axes of neck and head with the body. Unintentional blockage of the cerebral venous drainage by influencing course through the internal jugular vein because of its crimping with neck rotation or flexion is a successive issue experienced. Subsequently, in clinical practice to forestall brain bulge, it's wise to prevent an ascent in ICP. Likewise, hindrance to cerebral venous drainage can cause cerebral infarct, airway and neck edema.

Likewise, in mechanically ventilated patients factors such as high airway pressures can increase ICP by elevating central venous pressure and by blocking cerebral venous return to the right chamber and diminishing mean arterial blood pressure, both bringing about diminished cerebral perfusion pressure. (7) Keeping sufficient cerebral perfusion, limiting elevation in ICP and forestalling position related complications is of principal significance to the neuro anesthesiologist during intraoperative period.

Neurosurgical positioning complexly affects the systemic and cerebral circulations which might possibly diminish cerebral blood flow and oxygenation. (1)

Systemic changes of Positioning

A) Supine position

It is the most ordinarily utilized position for neurosurgical procedures involving the anterior and middle cranial fossa. When compared to the horizontal supine position, lawn-chair (contoured) modification of the horizontal position is most commonly preferred in neurosurgery, with 15-degree angulation and flexion at the trunk-thigh-knee, and provides more physiological positioning of the lumbar spine, hips and knees. When compared to an upright position, supine position causes increase in venous return (VR) , stroke volume (SV) and hence cardiac output (CO). The heart rate (HR) and systemic vascular resistance (SVR) typically decreases. Systolic blood pressure (SBP) often remains unchanged, though mean arterial pressure (MAP) may occasionally decrease or may remain unchanged. Jugular venous flow may increase or remain unchanged. Jugular venous resistance remains unchanged or may decrease. Cerebral Perfusion pressure is usually maintained or may decrease slightly. Total lung capacity (TLC) and functional residual capacity

(FRC) reduces in supine position. Also intrapulmonary shunting and ventilation-perfusion (V/Q) mismatch increases in supine position. [1]

B) Prone position

The prone position is regularly utilized for dealing with the lesions in the posterior cranial fossa, suboccipital region, and also posterior approach to spine. (8) The advantages of this position are that it is a decent situation for posterior approaches, and there is a lower occurrence of venous air embolism contrasted to the sitting position. (9, 10)

When compared to the supine position in awake state, prone position causes an increase in intraabdominal pressure, decrease in venous return and stroke volume causing compensatory increase in heart rate. Also, SVR & Pulmonary vascular resistance (PVR) increases. SBP and MAP may remain unchanged or increase. With the patient anesthetized, there is a decrease in VR, SV and CO. The heart rate, SVR, PVR increase. SBP an MAP both decrease (or may remain unchanged). Ventilation-Perfusion mismatch and atelectasis decreases in prone position and leads to an improvement in oxygenation. (1)

C) Lateral position

The lateral position is utilized for patients requiring craniotomy of temporal lobe, skull base, and posterior cranial fossa tech. The advantages of this position are that it gives the best surgical pathway to temporal lobe pathologies. (1) The dangers incorporate damage to brachial plexus, stretch injuries, pressure paralyses, and mismatch in ventilation-perfusion.

When compared to the supine position and under anesthesia, lateral position causes a decrease in VR, SV, and consequently CO. An increase in HR, SVR and MAP is noted. This position is associated with a decrease in both SBP and MAP. The respiratory effects of this position include a decrease in FRC and TLC and a higher increase in intrapulmonary shunting. Atelectasis of the dependent lung occurs further increasing the V/Q mismatch. The park-bench modification causes decreased perfusion of the dependent arm. (1)

Monitoring of Cerebral oxygenation and Cerebral Blood Flow

Regional cerebral tissue oxygen saturation monitoring using NIRS is emerging as a surrogate monitor of cerebral perfusion and cerebral oxygenation with widespread application in many types of

surgery including neurosurgery. Cerebral Blood flow is monitored non-invasively using transcranial doppler (TCD).

A) Near-infrared spectroscopy (NIRS)

NIR light can be used to measure rSO2 (regional cerebral tissue oxygen saturation). (4) Near-infrared spectroscopy (NIRS) is a novel technique which uses the principles of optical spectrophotometry which makes use of the fact that the skull, is relatively transparent in the NIR range. It is non-invasive optical technology which is upcoming as a monitor of cerebral perfusion. This is based on the differential absorption of near-infrared light by oxygenated and deoxygenated hemoglobin.

Due to the poor signal-to-noise ratio because of the low intensity of transmitted light, most commercially available devices use reflectance-mode NIRS in which receiving optodes are placed ipsilateral to the transmitter. This arrangement exploits the fact that photons traverse an elliptical path when transmitted through a sphere in which the mean depth of penetration is proportional to the transmitter and receiver optode separation distance, figure 1.

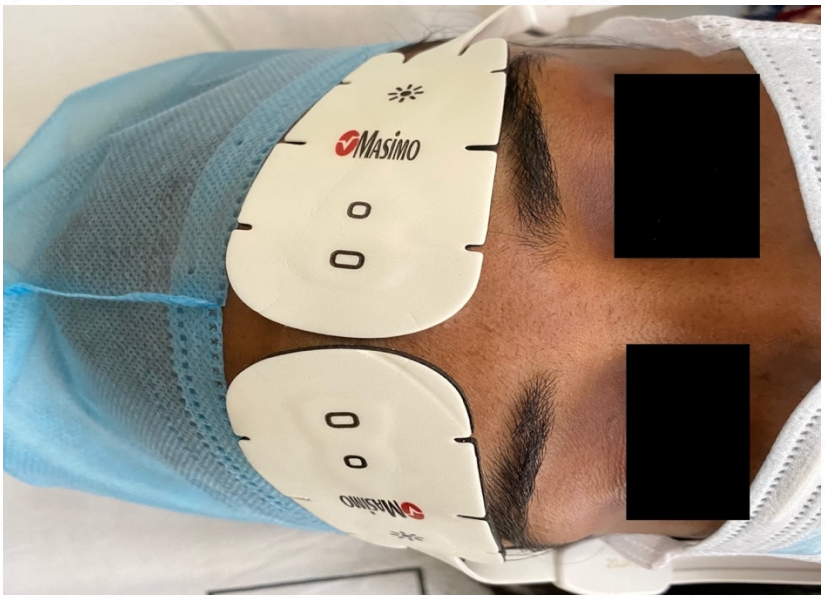


Figure 1: shows the NIRS electrodes position on the forehead.

NIRS is based on the Beer-Lambert law. Beer's law states that the intensity of transmitted light decreases exponentially as the concentration of a substance the light passes through increases, as shown in figure 2.

Lambert's law states that the intensity of transmitted light decreases exponentially as the distance travelled by the light through a substance increases, as shown in figure 3.

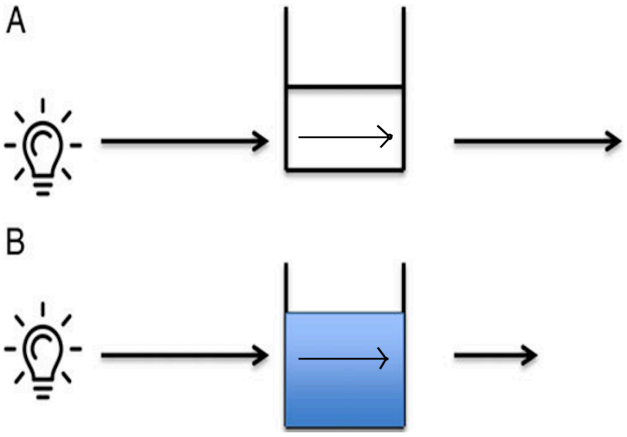


Fig 2: showing Beer's law (intensity of transmitted light decreases exponentially as the concentration of the solute increases)

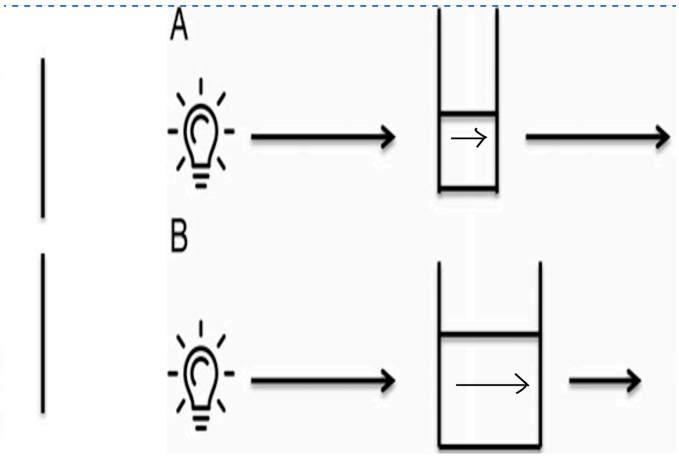


Fig 3: showing Lambert's law (intensity of transmitted light decreases exponentially as the distance travelled by the light increases)

Measurement of tissue oxygen saturation and tissue hemoglobin content is determined by the difference in intensity between the transmitted and received light delivered at specific wavelengths as stated by the Beer–Lambert law above.

In the 700–1300 nm range, NIR light penetrates several centimeters in the biological tissue. (11) Hemoglobin, bilirubin, and cytochrome are the primary light-absorbing molecules in tissue in the NIR range. The absorption spectra of deoxyhemoglobin (Hb) ranges from 650 to 1000 nm, oxyhemoglobin (HbO2) shows a broad peak between 700 and 1150, and cytochrome oxidase aa3 (Caa3) has a broad peak at 820–840 nm. (12) The commercial devices utilize wavelengths of NIR light between 700 and 850 nm where the absorption spectra of Hb and HbO2 are maximally separated and there is minimal overlap with H2O.

Correlation between NIRS and Cerebral Oxygenation:

Meeri et. al. did a study to assesses CBF by diffuse correlation spectroscopy (DCS) and oxy and deoxy- hemoglobin measurement by NIRS. DCS/NIRS based estimations of cerebral blood flow and cerebral oxygenation from frontal lobes were contrasted with simultaneous xenon-enhanced computed tomography (XeCT) during variations in carbon dioxide and blood pressure. (13) 7 patients were incorporated for the investigation. Relative CBF measured by DCS (rCBFDCS), and changes in oxy-hemoglobin (DHbO2), deoxy-hemoglobin (DHb), and absolute hemoglobin concentration (DTHC), estimated by NIRS, were persistently checked all through XeCT during both the scans, baseline and repeat scan after the intervention. $rCBF_{DCS}$ and $rCBF_{XeCT}$ showed good correlation among the patients. Moderate correlation was found between $rCBF_{DCS}$ and $DHbO_2$/DTHC. Both NIRS and DCS independently were able to differentiate the effects of xenon inhalation on CBF. The authors inferred that DCS estimations of CBF and NIRS estimations of tissue blood oxygenation could give a noninvasive, consistent monitoring for CBF in neurocritical care patients.

Pollard et. al. did a study to validate NIRS for brain oxygenation monitoring. (14) 22 conscious, healthy volunteers were studied, by breathing hypoxic gas mixtures. Using 12 subjects (referred as training group), they developed an algorithm based on the mathematic relationship which converts detected light from the field surveyed by the probe to cerebral hemoglobin oxygen saturation (CSfO2). The authors correlated the oximeter results with the estimated combined brain hemoglobin oxygen saturation CScombo2, where CScombO2=SaO2X 0.25+SjO2X0.75 and SjO2 = jugular venous saturation). The algorithm was then validated' in the 10 volunteers. A close association was found between CSfO2, and CScombO2. The conclusion of the study was that continuous monitoring with cerebral oximetry can accurately recognize decreasing cerebral hemoglobin oxygen saturation produced by systemic hypoxemia.

Marc et.al. did a study by using frequency domain near-infrared spectroscopy to determine the ratio of arterial and venous blood monitored by cerebral oximetry during three clinical situations: normoxia,

hypoxia, and hypocapnia. (15) They studied 20 anesthetized children aged < 8 years with congenital heart disease of varying arterial oxygen saturation (SaO_2) during cardiac catheterization. Cerebral oxygen saturation (ScO_2), SaO_2, and jugular bulb oxygen saturation (SjO_2) were measured by frequency domain NIRS and blood oximetry at normocapnia room air, normocapnia 100% inspired O_2, and hypocapnia room air. The study found arterial contribution of 16 ± 21% and venous contribution of 84 ± 21%, to cerebral oximetry. Though the contribution was similar among the three conditions of recording, significant variation was observed among the subjects (range, ~ 40:60 to ~ 0:100, arterial: venous). The authors concluded that because of biologic variation in cerebral arterial/venous ratios, use of a fixed ratio is not recommended. Though previous studies have demonstrated a predominant contribution from venous system (70%-80%). (16)

Regional cerebral oxygen saturation (ScO2) of the cerebrum is dictated by contrasting the particular absorbance patterns of oxygenated and deoxygenated hemoglobin to approach infrared light. (17) As CBF diminishes, tissue oxygen extraction will increase in order to keep up with cerebral metabolic demands with an inevitable reduction in hemoglobin saturation. Within the sight of a stable metabolic rate, ScO2 is accordingly an indirect index of CBF and gives an idea of organ ischemia. (18) Checking ScO2 permits clinically quiet episodes of cerebral ischemia to be distinguished, and the innovation has been utilized widely, going from neonatal to adult surgeries. Nonetheless, many elements might impact oxygen transport and ScO2 including hematocrit, affinity of hemoglobin-O2 binding (P50), inspiratory oxygenation and ventilation. Also of note is the fact that CBF can itself be influenced by position of the head, the posture adopted and, any anatomical variation. (19, 20)

Neurological monitoring of cerebral oxygenation with cerebral oximetry might refine the management strategies of hypoxia and hypotension in the intraoperative period. (21) Intraoperative ScO2 estimation gives a helpful endpoint to clinical intercession, for example, the utilization of vasopressor treatment to increase cerebral perfusion pressure (CPP). In any case, without direct estimation of cerebral perfusion, the discernment that blood pressure control alone is adequate to keep up with cerebral perfusion in all patients is restricted. The clinical supposition that CBF increases simultaneously with increase in blood pressure can't be made.

In another study, Iwasaki et al. studied the changes in HbO_2 and HbTot which occur after induction with different concentrations of sevoflurane (5%, or 8%) and combination of 67% N_2O and propofol. (22) The hemodynamic parameters of the patients were continuously recorded from baseline till 3 minutes after airway was secured.

The authors found critical increase in HbO2 and HbTot after 8% sevoflurane contrasted with 5% sevoflurane and propofol. The inference was that f NIRS is exceptionally delicate methodology to pick up changes caused by different concentrations of even the same anesthetic.

Advantages and Limitations of NIRS:

NIRS has numerous advantages such as being non-invasive, ability to simultaneously sample multiple regions of interest with high temporal resolution, and being user-friendly. Though NIRS has multiple advantages, it is not without limitations. NIRS values can be confounded by a number of factors, such as by contamination from extracerebral blood from scalp or subdural hematoma, presence of non-heme tissue chromophores etc. The spatial resolution provided by NIRS is restricted. It can sense the outer 1-4 mm of the cortical surface only. (23) Also, the area between light emitter and detector only contributes to the NIRS value. (24)

Transcranial Doppler

TCD is a non-invasive, bed side, real time monitoring and diagnostic tool of measuring blood flow velocity (FV) and other derived parameters in various intracranial arteries. A phased array probe with frequency of 1–2 MHz is used to insonate the intracranial arteries (Figure 6). The use of TCD has expanded drastically over the last three decades and has emerged as cost-effective tool for evaluating cerebral hemodynamics, detecting stenosis, collateral flow pattern, cerebral autoregulation and embolization. (25)

TCD works on the principle of Doppler shift. TCD probe emits sound waves which are generated by a piezoelectrical crystal located in the probe of the TCD. These waves are directed towards basal arteries through TCD ‘acoustic windows’ by positioning the probe appropriately. The red blood cells (RBCs) in the blood stream reflect the sound waves which are captured back by the TCD probe. (26, 27)

A positive deflection of the waveform indicates that the direction of blood flow in the vessel is towards the probe whereas, a negative deflection of the waveform suggests that the flow is away from the probe. As RBCs are moving particles, they change the frequency of reflected sound waves. The difference between the frequency of emitted and reflected waves measures FV of RBC and hence, the velocity of blood. It should be noted that TCD measures blood FV and not the cerebral blood flow (CBF). However, in a given condition, FV can be used as a surrogate marker for vessel diameter or CBF. TCD also measures vessel pulsatility and derives pulsatility index (PI) and resistance index (RI).

Recently transcranial color Doppler has also become available which makes insonation of various arteries easy. In some monitors, M-mode (motion mode) imaging is available which helps during the examination of the cerebral vessels.

Few areas of the skull bone are relatively thinner and allow penetration of ultrasound waves. This can be used to visualize the underlying cerebral blood vessels. The four commonly utilized acoustic windows are temporal, orbital, sub occipital, and submandibular windows. In many individuals (10–20%) it is not possible to get an adequate window. (28, 29, 30)

The most commonly used window for insonating the intracranial arteries is the trans-temporal window, since most of the important vessels are accessible through this window. This window is used for insonating internal carotid (ICA), middle cerebral (MCA), anterior cerebral (ACA) and posterior cerebral arteries (PCA). Trans-temporal acoustic window is an area which is delineated by a line drawn from tragus to the lateral canthus of the eye. The TCD probe is coated with an ultrasound gel and kept 2 cm above this line. The intracranial space is explored using the Power Motion (PM) mode. The movements during TCD examination have to be extremely fine, with the probe held as a pen between index finger and thumb and the base of palm resting on the patient's head. When the transducer is paced appropriately, the screen is filled with color signals between 30 to 80 mm depth. A red colour signal at the depth of 45 to 60 mm signifies the ipsilateral MCA (Figure 7). The Doppler spectral pattern provides important information about the flow characteristics in the arterial segment. (31)

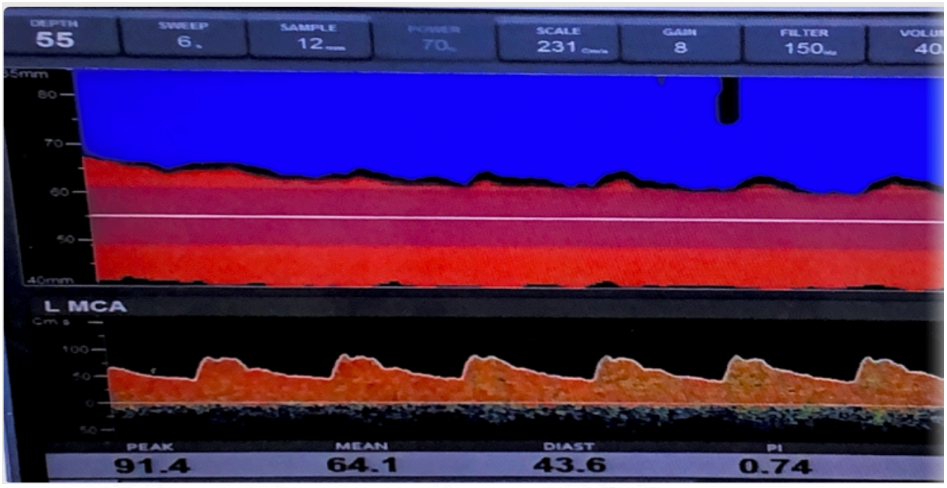


Figure 4 shows the TCD power motion mode (M-mode) in the upper half with the MCA tracing in the lower part of the figure along with the measured velocities shown in red color.

Once a band of frequency shifts is seen at any depth, the appropriate sample gate is selected by moving the gate selection line to the appropriate depth, and the doppler waveform is then visualized. Ideally, the doppler waveform should be a mix of colors with blue color (smaller number of RBCs) being near the spectral envelop and yellows/red color occupying the main body of the wave. A readjustment of probe position is needed if all colors are blue, indicating that the artery is not being insonated properly.

The artery which is most easily insonated from this window is the MCA, which can be insonated anywhere along its course from a depth of 35 up to 60 mm. It is seen as a broad red band in the PM mode window. When the MCA is traced deeper, a bidirectional flow can be detected at approximately 55 – 65 mm which appears as contiguous bands of red and blue color, with red being directed upwards (Figure 9). This location indicates the bifurcation of ICA into MCA & ACA. The ACA can be seen as the blue color band, from 60–70 mm. To insonate the ACA, the probe should be tilted slightly anteriorly.

The FV of the arteries measured using TCD is given by the formula:

FV (measured) = FV (actual) × cosine (angle of insonation)

Equation derived from Dopplers Principle: CBFV

- **v = [(c × *fd*)/(2 × *fo* × cos theta)]**
- c=speed of the US Wave emitted from probe,
- *fo* =emitted Wave pulse frequency
- *fr* =received Wave pulse frequency,
- *fd*=Doppler shift
- θ= angle of formed by reflected wave relatively to the initial US emission beam.

As can be seen from the formula above, measured FV will be less than actual FV. This raises two problems; firstly, it leads to inaccurate readings and secondly it brings inter-observer variation and makes the repetition of test erroneous. With the use of the temporal window, the first problem is easily minimized because MCA can only be insonated within a narrow-angle. The second problem can be avoided by fixing the probe when serial testing is required.

Using TCD, the variables that can be adjusted are the depth of insonation, power and filter ratio. The observed parameters are systolic FV, diastolic FV, mean FV (MFV), Pulsatility index (PI), and Resistivity index (RI).

The normal spectral waveform shows a sharp systolic upstroke and stepwise deceleration with positive end diastolic flow. Peak systolic velocity (PSV) is the first peak on the on a TCD waveform from each cardiac cycle. The end diastolic velocity (EDV) lies between 20 to 50% of the peak systolic velocity indicating the low resistance pattern of intracranial arteries. Mean flow velocity (cm/sec) is derived from PSV and EDV. MCA has the highest MFV. The mean flow velocity waveform shows least variation and is the commonly used parameter in TCD studies, given by the formula:

MFV = EDV+1/3(PSV-EDV)

In our study we mainly used Mean flow velocity (MFV) parameter, since mean flow velocity waveform shows least variation in TCD

Correlation between CBF and TCD flow Velocity

Bishop et. al. did a study to validate the technique of blood flow velocity measurement by TCD, and comparing it with intravenous Xenon based measurement of CBF. (32) They did the study in 17

symptomatic patients with cerebrovascular disease, and measured the blood flow velocity of middle cerebral artery (MCA) and intravenous Xenon based measurement of CBF. The measurements were taken at rest and during hypercapnia. They found that the variations in MCA flow velocity dependably associated with changes in CBF, however the total flow velocity can't be utilized as a marker of CBF. Though at rest, the authors found a poor correlation between the absolute measurement of MCA velocity and hemispheric CBF.

Leung et al., conducted a study to compare measurements of blood flow velocity (BFV) and BFV changes in the middle cerebral arteries (MCA) acquired from phase contrast magnetic resonance angiography (PCMRA) and transcranial Doppler ultrasound (TCD) during controlled manipulation of end-tidal partial pressure of carbon dioxide (PETCO2). (33) The study was conducted in 9 healthy volunteers. TCD and PCMRA velocity data were taken from the M1 segment of the MCA were acquired during precise targeting of PETCO2 induced by a computer-controlled gas delivery system. Doppler spectra and phase contrast data were processed into time-averaged peak-velocity (TAPV) values for comparison. Changes in velocity between baseline and hypercapnia were analyzed in terms of velocity-based cerebrovascular reactivity (CVR). The authors found good correlation between the pairs of velocity measurements acquired from the both TCD and PCMRA. The authors concluded that under the conditions of precise PETCO2 control, PCMRA appears to be more consistent than TCD, though TCD was qualitatively comparable to PCMRA measures of velocity in the MCA.

Brauer et al., conducted a study to investigate the correlation between changes in TCD-mean flow velocity (Vm) and changes in CBF in patients with various intracranial pathology undergoing cerebrovascular reactivity tests. (34) The study included 32 patients who were randomized into two groups for different reactivity tests. Patients in group 1 included 18 patients (awake or sedated) received a 1-g dose of acetazolamide intravenously, whereas group 2 included 14 patients wherein mechanical ventilation was adjusted to produce a 20% decrease in arterial CO2 tension compared with baseline CO2 value. Regional CBF was measured using xenon-enhanced computed tomography (Xe-CT) at the levels of the basal ganglia and the lateral ventricles. Bilateral MCA flow velocity was measured with pulsed TCD. Hemodynamic parameters (Mean arterial blood pressure, heart rate) and end-tidal CO2 were continuously recorded during the entire procedure. After baseline measurements and either alteration of CO2 or application of acetazolamide, the cerebrovascular reactivity was assessed at 20 min by a second measurement of CBF, TCD, and all other physiologic variables. A close correlation was seen between changes in CBF and Vm ($r=0.82$) though in subgroups formed of patients when classified according to the diagnosis, data dispersion suggested a weaker correlation. The authors concluded that the correlation between Vm and CBF may vary with intracranial pathology.

Another study by Sokoloff et al., evaluated whether blood flow velocity measurement with transcranial Doppler (TCD) could predict cerebral hypoxic episodes in moderate-to-severe TBI measured with a $PbtO_2$ probe. (35) The study recruited 17 patients. Measurements were done bilaterally on the middle cerebral artery (MCA) early after the insertion of $PbtO_2$ monitoring, daily for 5 days and during dynamic challenge tests. Physiological parameters were simultaneously collected (PaO_2, $PaCO_2$, hemoglobin [Hb] level, intracranial pressure, and cerebral perfusion pressure [CPP]). 85 TCD studies were conducted in 17 patients.Twenty-nine (34%) TCD measures were performed during an episode of cerebral hypoxia ($PbtO_2 \leq$ 20 mmHg). For early episodes of cerebral hypoxia (occurring ≤ 24 h from trauma), all Vmean < 40 cm/s were associated with an ipsilateral $PbtO_2 \leq 20$ mmHg (positive predictive value 100%). However, when considering all readings over the 5 days study period, however, no correlation was found between $PbtO_2$ and MCA's mean blood flow velocity (Vmean). Vmean is also positively correlated with $PaCO_2$, whereas PbtO2 is also correlated with PaO_2, CPP, and Hb level. The authors concluded that early TCD measurements compatible with low CBF (Vmean< 40 cm/s) can detect brain tissue hypoxia early after TBI (≤ 24 h) and could potentially be used as a screening tool before invasive monitoring insertion which could help to minimize secondary injury.

Kirkham et.al. did a study to evaluate the ability of cerebral blood flow velocity to monitor changes in MCA blood flow. (36) The study was conducted in 38 healthy volunteers. They evaluated the variation of TCD flow velocity with the changes in CBF produced by altering the arterial carbon dioxide tension (studied with arterial PaCO2 (partial pressure of Carbon-dioxide and end-tidal PCO2). The study found that the relationship between MCA doppler mean frequency, and either arterial or end-expiratory pCO2, was linear over the range of PaCO2 values of 20-60 mm Hg.

Advantages and Limitations of TCD:

TCD has numerous advantages. It is non- invasive, repeatability and does not involve exposure to ionizing radiations. Its portability is an added advantage in critical care and operation theatres. Also, it gives a real-time information regarding the flow velocities compared to other modalities of measurement of cerebral blood flow, which give value only at a fixed time point. TCD is not without limitations. (37) Major limitation of TCD being that it is highly operator dependent. In 10 to 15% of patients TCD acoustic windows are inadequate. TCD provides information only regarding global cerebral blood flow velocity rather than local blood flow velocity. (38).

Effects of Postural changes on CBF and Cerebral oxygenation

In the study by Arjen Mol et. Al, cerebral oxygenated hemoglobin (O_2Hb), deoxygenated hemoglobin (HHb) and tissue saturation index (TSI) were measured bilaterally on the forehead of 15 healthy young adults using NIRS. Sitting to standing, and slow and rapid supine to standing movements were assessed in their study. (39) Both oxygenated and deoxygenated hemoglobin dropped in the early phase after standing up, indicating a lower concentration of total hemoglobin, therewith reflecting a decrease of cerebral perfusion. Their study found that both the oxygenated and deoxygenated hemoglobin dropped in the early phase after standing up, indicating a lower concentration of total hemoglobin, therewith reflecting a decrease of cerebral perfusion.

Similar findings were also reported by van Lieshout et al and Thomas et al. (40, 41) in their study who demonstrated early perfusion drop after standing up assessed by transcranial Doppler. This perfusion drop indicates that cerebral autoregulation may not immediately compensate for blood pressure drops resulting from gravitational pooling after standing up, even in healthy adults. This perfusion drop in the brain having a constant oxygen demand can lead to the cerebral hemoglobin saturation decrease. The study concluded that in healthy individuals, NIRS-based cerebral oxygenation parameters are sensitive in detecting postural change and discriminate between standing up from supine and sitting position with minimum O_2Hb response as the most sensitive and reliable parameter.

Jakob et.al, in their investigation found that during positive pressure breathing, the prone position with head turned sideways lead to reduction in mean flow velocity of MCA by nearly 10% even when MAP is elevated. (42) Prone position with turned head influences both CBF and venous drainage of brain showing that ideal cerebral perfusion requires centering of the head. In healthy awake individuals, the prone position with positive pressure breathing reduced MCA MFV especially when the head was rotated to the side. These results may have implications for the anesthetized and ventilated patient.

In the study by Gulsah & Sevgi, they studied the effects of head & neck positions on the cerebral blood flow velocity by transcranial Doppler ultrasound in patients who underwent cranial surgery. (43) The MCA mean flow velocity of the patients was measured in supine position with 0° and 30° head elevations, right and left lateral positions, right and left lateral positions with head flexion and extension. The measurements were taken before surgery and postoperatively within 72 hours after surgery. The study found that the MCA mean flow velocity was increased in head elevations from 0° to 30°, in right and lateral positions with 30° head elevations, but the velocity was decreased in head flexion and extension positions in preoperative as well as the postoperative periods.

Ledwith et. Al, studied the effects of body position on cerebral oxygenation. (44) They studied the effects of 12 different body positions on neurodynamic and hemodynamic outcomes in 33 patients. The patient population include patients affected by traumatic brain injury, subarachnoid hemorrhage, or craniotomy for tumor. Cerebral oxygenation changes were evaluated with brain tissue oxygen ($PbtO_2$) monitoring. Simultaneously ICP was also calculated. The study evaluated 12 different positions, which were as follows: supine, supine with knee bent, left lateral position, and right lateral position, and in each of these positions, the head of bed was then elevated to 15^0, 30^0, or 45^0. Changes from baseline to the 15-minute postposition assessment mean change scores showed a downward trend for PbtO2 for all positions with statistically significant decreases observed for supine head of bed (HOB) elevated 30^0 and 45^0 ($p < .01$) and right and left lateral positioning HOB 30^0 ($p <.05$). ICP decreased with supine HOB 45^0 ($p < .01$) and knee elevation, HOB 30^o and 45^o ($p < .05$), and increased ($p <.05$) with right and left lateral HOB 15^o. Hemodynamic parameters were comparable in the various positions. The lateral position was found to have the most adverse effect on intracranial physiology.

Effects of anesthetic drugs on CBF and cerebral oxygenation

Anesthetic agents can affect the cerebral blood flow and oxygenation through direct and indirect actions. Since we have planned to conduct the current study under anesthesia intraoperatively with Propofol as an induction agent and Sevoflurane for maintenance, it is important to know the effects of the agents used on cerebral hemodynamics.

Effects of Propofol on Cerebral Circulation:

Propofol produces dose-related reduction in global CBF by 50 to 60%. (45, 46) This variation in regional cerebral blood flow (rCBF) reduction has been confirmed with Positron Emission Tomography (PET) studies which demonstrated large preferential decrease in the medial thalamus, cuneus and precuneus, and posterior cingulate, orbitofrontal, and right angular gyri. (47) The CBF decrease with propofol is mainly due to its metabolic depressant effect. Propofol depresses CMR (Cerebral Metabolic Rate) and does this differently according to region. (48) The metabolism in the cerebral cortex decreases (by 58%) more than the sub- cortical brain areas (by 48%). In patients with intracranial tumors, Intracranial Pressure (ICP) was shown to be lower and Cerebral Perfusion Pressure (CPP) higher in patients anesthetized with propofol when compared with those anesthetized with volatile anesthetics (isoflurane or sevoflurane). (49) MAP usually decreases with administration of propofol, which necessitates strict maintenance of MAP/ CPP, even more important in patients with elevated ICP.

Effects of Sevoflurane on Cerebral Circulation:

The study by Kaisti et.al. using PET showed either a decrease or no change in global CBF occurred with sevoflurane. (45, 50) MAP decreased significantly with sevoflurane in the study by Kaisti et.al, whereas MAP was found to be unchanged in the study by Schlunzen et.al. Thus, as long as MAP is maintained, global CBF remains unchanged with use of sevoflurane, though heterogeneity of the rCBF response was seen; with a increase in rCBF seen in the anterior cingulate and a reduction in the cerebellum.

During propofol-induced isoelectric EEG, sevoflurane at both 0.5 and 1.5 MAC produced a smaller increase in Vmca (MCA mean flow velocity) than isoflurane, suggesting lesser vasodilating potential of sevoflurane than isoflurane. (51) Sevoflurane at 1 MAC was reported to decrease rCMRg (regional metabolic rate for glucose) in multiple regions of the brain, with the maximum decrease seen in the lingual gyrus (by 71%), followed by occipital lobe (by 68%), and thalamus (by 68%). (52) Sevoflurane, with or without N_2O, produces no or small increases in ICP seen in animal studies as well as in humans. These effects reinforce the fact that sevoflurane does not affect the CBV (Cerebral Blood Volume) though it causes reduction in CBF. (53)

Effect of anesthesia induction has been previously demonstrated in NIRS based studies. Lovell et al. used functional NIRS (fNIRS) in their study and documented small changes in cerebral oxygenation after induction of anesthesia with intravenous anesthetics such as propofol, thiopentone and etomidate. (54) They studied the hemodynamic changes of the patients from preinduction untill three minutes after giving intravenous (IV) anesthetic induction drugs. The changes observed in oxygenation were substantiated as differences in the relative oxygenated hemoglobin (HbO2) and total hemoglobin concentration (HbTot). The authors concluded that IV induction agents such as thiopentone and propofol cause an increase in both HbO2 and HbTot compared to the baseline period. This investigation uncovered that fNIRS has the ability to gauge drug dependent changes in cerebral oxygenation seen with anesthetic induction.

Similar study with NIRS was done by Fleck et al. (55) This study was conducted in pediatric population with congenital heart disease. They measured the changes in cerebral oxygenation (by NIRS) and blood supply (by cardiac output using electrical velocimetry) in children with congenital heart disease after Propofol infusion. Propofol sedation caused a significant drop in mean arterial pressure (MAP) whereas cerebral tissue oxygenation index, showed an increased. The authors postulated that the increase in cerebral oxygenation with propofol sedation despite a decrease in cardiac index and MAP may be due to decreased oxygen consumption of the sedated brain in the presence of an intact cerebral auto-regulation.

3. HYPOTHESIS

Hypothesis

We hypothesize that patient positions adopted for performing various neurosurgical procedures from awake supine state to either lateral or prone positions causes significant changes in cerebral oxygenation and cerebral blood flow velocities compared to neurosurgical procedures performed in supine posture under anesthesia.

4. AIMS AND OBJECTIVES

Aims And Objectives

Primary Aim:

1. To assess the effects of change in position from supine to lateral and supine to prone positions on cerebral oxygenation and cerebral blood flow velocity compared to patients operated in supine during elective neurosurgery.

Secondary Aim:

1. To assess the significance of position induced changes in cerebral oxygenation and cerebral blood flow velocity during patient positioning in neurosurgery.

2. To compare the changes in cerebral oxygenation and cerebral blood flow velocity from awake state to anesthesia and final positioning during elective neurosurgery.

3. To assess the correlation between cerebral and systemic hemodynamic changes following final patient positioning in neurosurgery.

5. MATERIALS AND METHODS

Materials And Methods

We planned a prospective observational study to assess the changes in cerebral oxygenation and cerebral blood flow with respect to different surgical positions in patients who underwent elective craniotomy for primary brain tumors in the Neuro-Surgical Operation Theatre (NSOT) of Sree Chitra Tirunal Institute of Medical Sciences and Technology (SCTIMST), Trivandrum, which is a specialized tertiary referral center.

Institute Ethics Committee Approval: Institutional Ethics Committee approved our study. (SCT/IEC/1401/JULY-2019, dated 12.08.2019 Annexure- 2)

The study was conducted between August 2019 to May 2021.

Study Design: Prospective observational study.

Patient enrolment: The patients for the study were randomly selected from elective neurosurgical operation theatre list and were recruited after fulfilling the inclusion and exclusion criteria.

Inclusion criteria:

Consenting adult patients with age more than 18 years undergoing elective neurosurgical procedures

- ASA (American society of Anesthesiology) physical status I and II patients.
- Age 18-60 years.
- GCS of 15.

Exclusion criteria

- Patient refusal.
- Age less than 18 years and more than 60 years.
- GCS <15
- Emergency surgeries

- Longstanding diabetes mellitus, hypertension, vascular diseases, COPD (chronic obstructive pulmonary disease).
- Known case of cardiac illness (coronary heart disease, valvular heart disease, congenital heart disease, congestive heart failure).
- Patients with previous history of stroke, transient ischemic attacks, carotid disease, severely increased ICP.
- Obesity.
- ASA physical status III, IV, V patients.
- Patients with sub-arachnoid hemorrhage, vasospasm.
- Pregnant & nursing mothers.
- Poor TCD window

Informed written consent was obtained from those who were willing for the study participation. The total number of patients recruited were 60 based on the sample size.

Grouping of the patients (20 subjects in each group) was done according to the final surgical positioning as follows;

Group S: Patients who were operated in supine position.

Group P: Patients who were operated in prone position.

Group L: Patients who were operated in lateral position.

Patients were kept nil per oral of 6 hours for solids, 4 hours for liquids and 2 hours for clear fluids. No sedative/opioid premedication were given. Patients were administered their preoperative medications like antiepileptic drugs, steroids and anti-aspiration prophylaxis in the form of tablet pantoprazole 1mg/kg as per our institute protocol.

After completing the WHO (World Health Organization) Safety check list the patients were wheeled into the operation theater. Patients were positioned in supine position with small head support. Preinduction monitoring lines consisting of five lead electrocardiogram (ECG), pulse oximetry, non-invasive blood pressure (NIBP) were attached. A wide bore intravenous line was

secured under local anesthesia and intravenous fluid in the form of lactated Ringer's solution was started at 2 ml/kg. Before induction of anesthesia, baseline hemodynamic parameters including pulse rate, blood pressure, and oxygen saturation were noted. For recording the NIRS values, the forehead was cleaned and dried thoroughly. Then baseline NIRS reading was taken in supine position from the NIRS electrodes placed at the frontal area bilaterally.

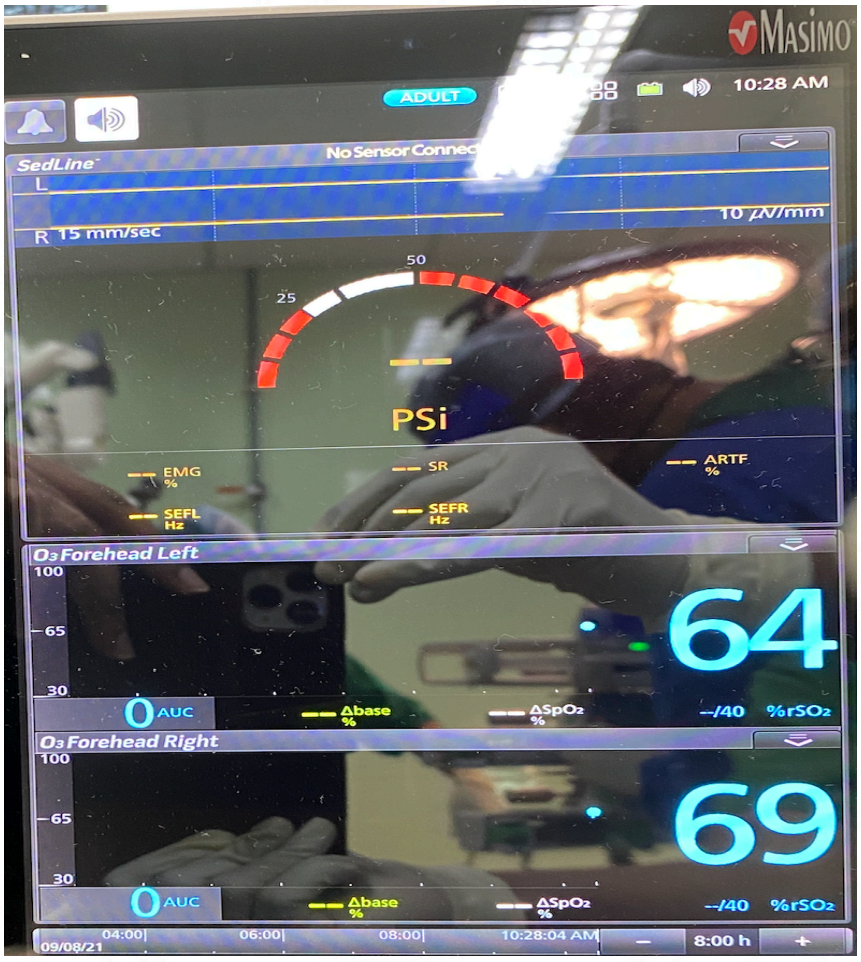


Figure 5: showing NIRS machine (MASIMO ROOT with O3 Regional Oximetry).

The machine used in our study was MASIMO ROOT (with O3 Regional Oximetry) as shown in Figure 5. This was followed by recording baseline flow velocities of MCA & ACA bilaterally in supine position with TCD. The TCD machine used in our study was DOLPHIN *IQ* VIASONIX as shown in figure 6.

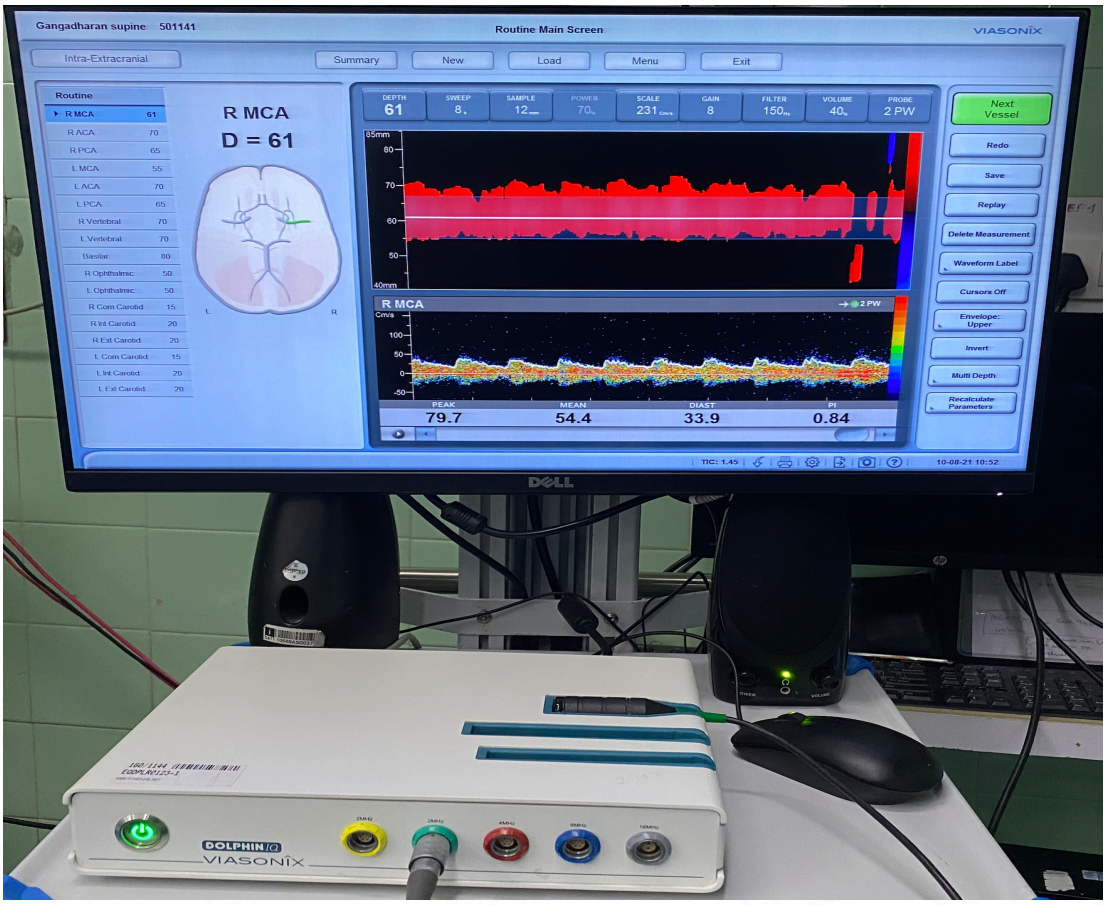


Figure 6: The Transcranial Doppler machine and the probe used in our study.

Technique for MCA & ACA visualization by TCD

All the patients placed comfortable in supine with head positioned with support and in neutral position. The 2 Mhz TCD probe was placed in front of tragus above the imaginary line drawn from outer cantus of the eye to the tragus on the right side. The depth was preset to 40-60 mm with gain setting of 8 and power of 50%. The middle cerebral artery (MCA) flow was identified in the M-mode window showing the red color. The doppler flow was obtained from the dedicated window for the same and the values were recorded. Then the probe is tilted anteriorly to obtain to the blue color in the M-mode representing anterior cerebral artery flow at a depth of approximately 60-80 mm. The doppler wave was obtained for recording the velocities. The peak flow, mean flow velocity and the pulsatality index (PI) were recorded. The similar technique was used for the left side.

After recording of the baseline (T0) parameters, general anesthesia was induced after preoxygenation for 3-5 minutes with Inj. Fentanyl, 2 mcg/kg, Inj. Propofol, 2mg/kg in titrated doses. After checking the adequacy of mask ventilation, Inj.Vecuronium (0.1 mg/kg) was administered and patients were intubated with appropriate size endotracheal tube via direct laryngoscopy/ video laryngoscopy/ fiberoptic bronchoscopy as deemed appropriate. Anesthesia was maintained using a combination of Oxygen (50% FiO2: Air) and Sevoflurane at an end tidal concentration equivalent to 1 MAC. Patients were ventilated in volume-controlled mode with a tidal volume of 8 ml/kg. Respiratory rate was adjusted to maintain an $ETCO_2$ value between 32-35 mmHg. Radial artery was cannulated for invasive arterial BP monitoring whereas central venous line was inserted via internal jugular vein or Peripherally inserted central line (PICC). Nasopharyngeal temperature probe was attached and the patient's temperature was maintained throughout the procedure between 35^0-$36\ ^0$ C.

Mean arterial blood pressure (MAP) was maintained within 10% of baseline throughout the procedure with use of intravenous fluids $\pm$ vasopressors. Once the patient's vitals were stable for 10 minutes after induction of anesthesia, hemodynamic variables, bilateral regional cerebral oxygen saturation were recorded. and cerebral blood flow velocity of MCA & ACA were recorded in supine position similar to the technique described earlier and recorded as **T1** time point.

Adequate padding was applied to pressure points and all the patients were applied mechanical compression device for both the legs. With appropriate safety checks and precautions, all patients were placed in the final neurosurgical position (supine, lateral or prone) by the team of neurosurgeons and anesthesiologists and the head was clamped in three-pin Mayfield head frame. To blunt hemodynamic response to skull pinning, adequate depth of anesthesia was ensured and additional dose of Inj. Fentanyl 2mcg/kg iv was administered.

Once the patient was placed in the final surgical position, and after 10 min of hemodynamic stability, the hemodynamic, respiratory variables, NIRS and TCD measurements were repeated again as described above. This time point was recorded as T2.

In the Supine group the patients were positioned with a reverse Trendelenberg of 10 to 15 degrees from horizontal to provide optimal cerebral venous drainage as well as slight flexion at hip and

knee by placing a pillow for thigh support and with leg elevation in order to improve venous return to the heart. (1) (Fig. 7) It was ensured that there is no excessive head and neck rotation or tilt that will compromise the vessels and airway.

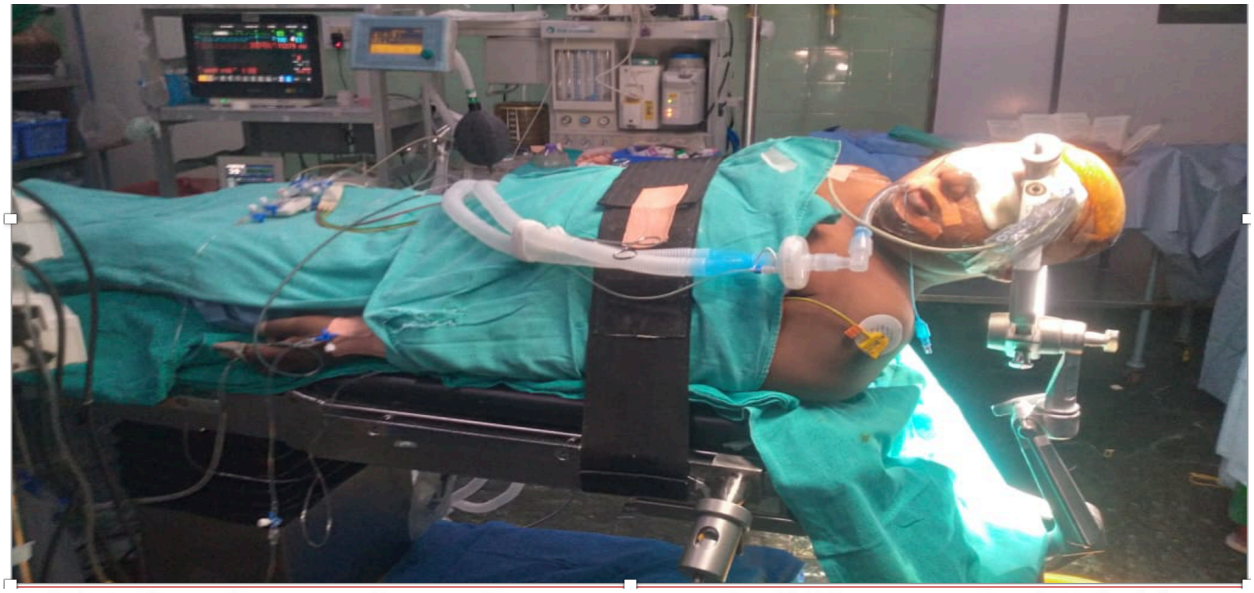
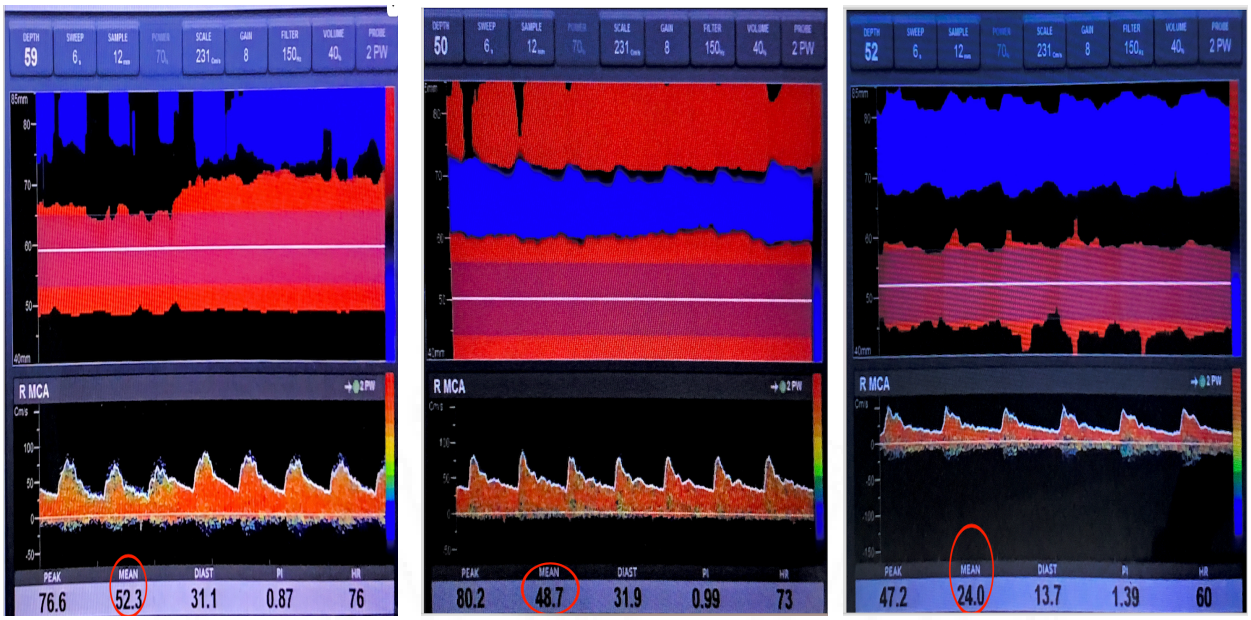


Figure 7: showing supine position with 30° head elevation

T0 T1 T2

Figure 8: showing NIRS values recorded at T0, T1 and T2 time points in supine position.



T0 T1 T2

Figure 9: showing TCD indices recorded at T0, T1 and T2 time points in supine position.

In prone group the patients were kept over two customized bolsters placed under the upper chest and pelvis with adequate padding and support for all pressure points. The head position was kept neutral. Positioning of the head was done with 10^0 elevation with respect to torso. (56) (Fig. 10)

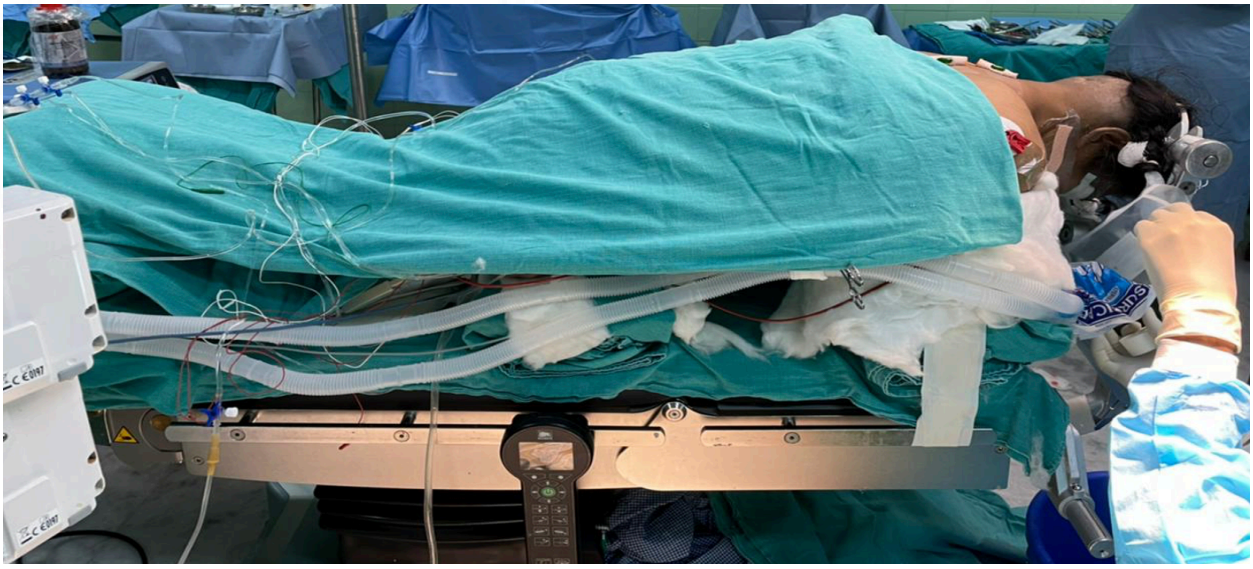
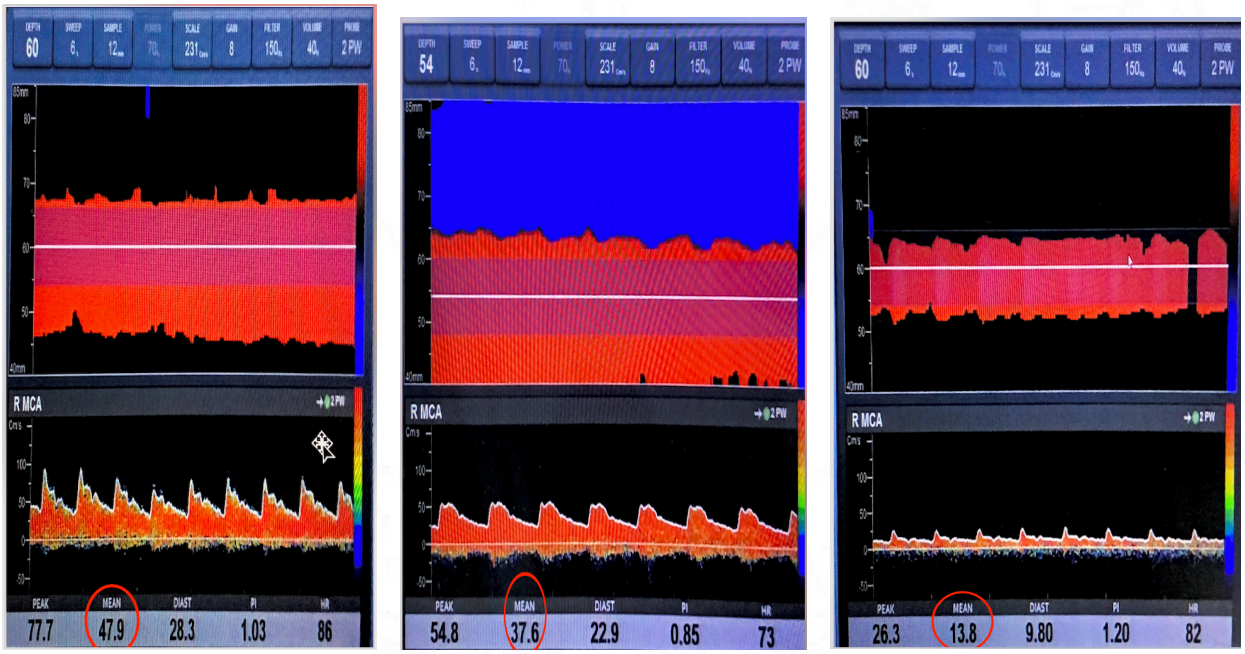


Figure 10: showing prone position.

T0 T1 T2

Figure 11: showing NIRS values recorded at T0, T1 and T2 time points in prone position.



T0 T1 T2

Figure 12: showing TCD indices recorded at T0, T1 and T2 time points in prone position.

All the patients in the lateral group underwent surgery in the park-bench modification of lateral position which gives the surgeon a better surgical access to the structures in posterior fossa, in contrast to the lateral position. At our institute we use park-bench position for surgeries requiring lateral position. Head is flexed at the neck and then turned to look toward the floor of operation theatre (120^0 from vertical & laterally flexed 20^0).

At all times most extreme consideration ought to be required to forestall complications in the post-operative period by avoiding excessive neck extension, flexion or rotation. (57) (fig.13)

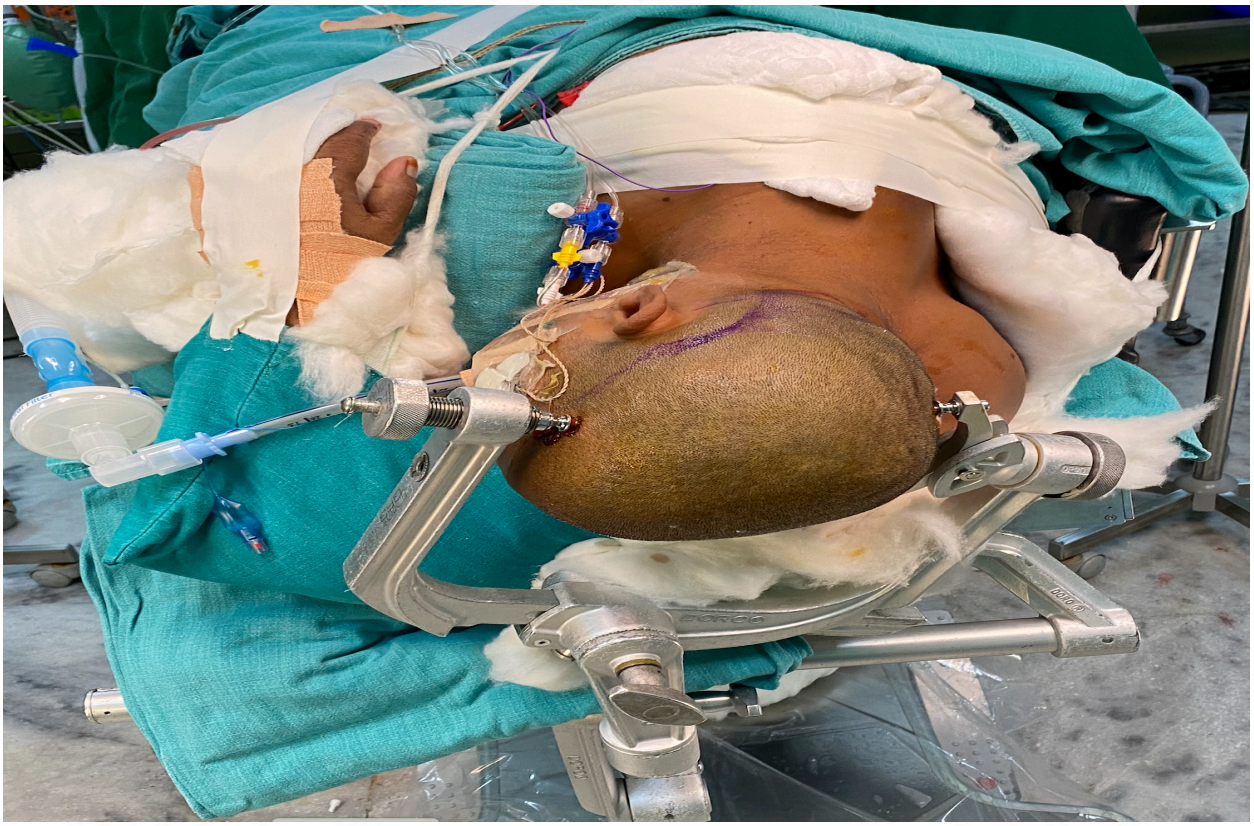


Figure 13: showing park-bench position.

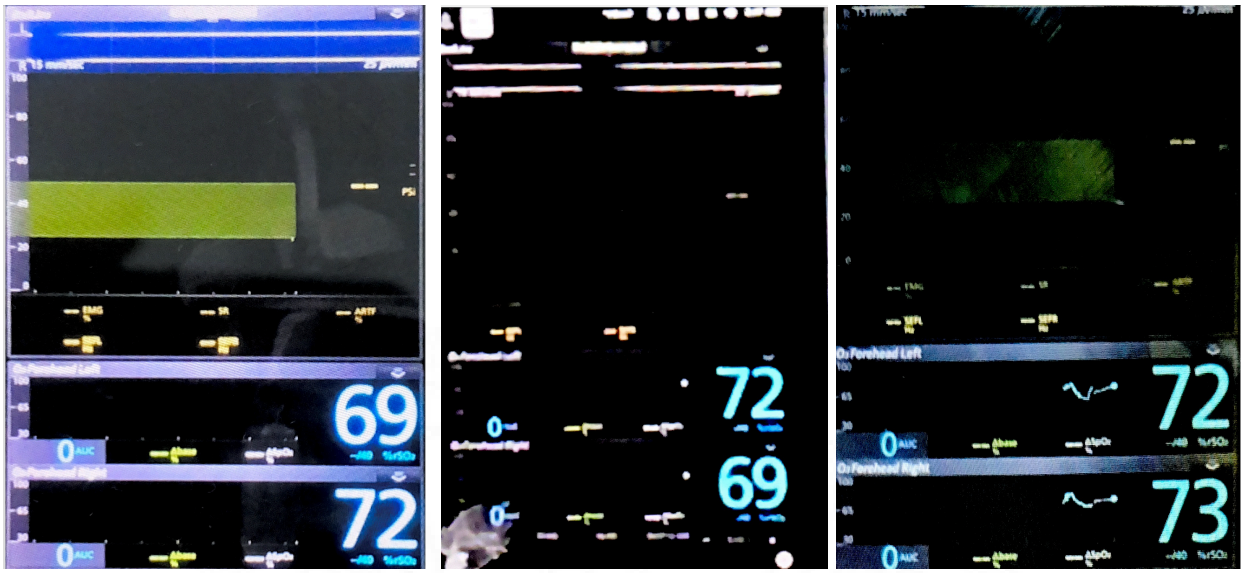
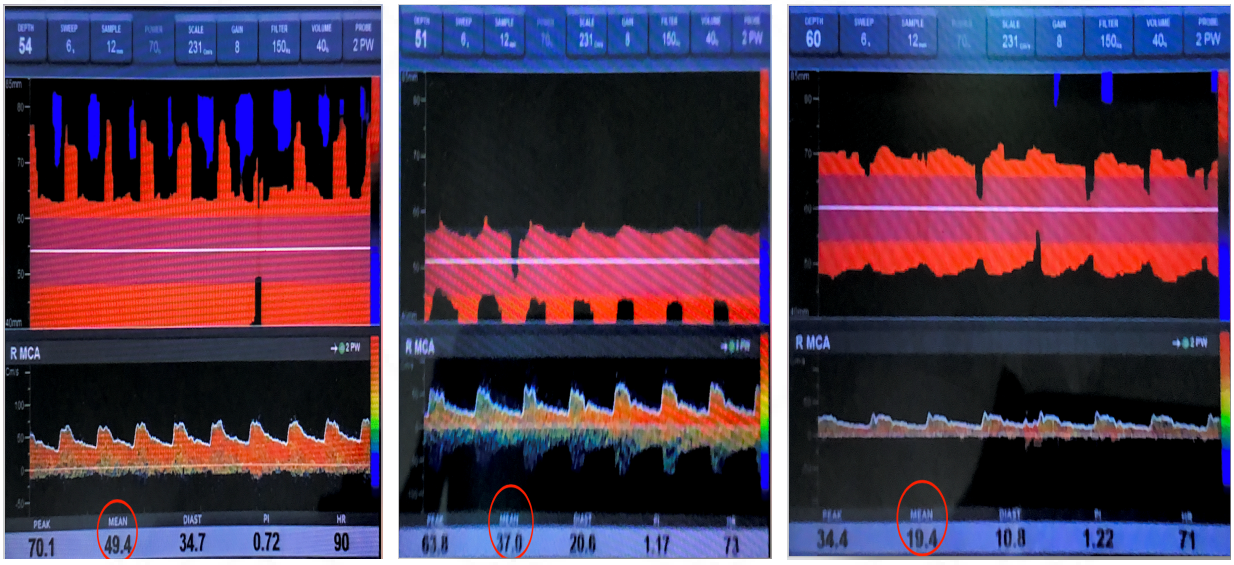


Figure 14: showing NIRS values recorded at T0, T1 and T2 time points in lateral position.



T0 T1 T2

Figure 15: showing TCD indices recorded at T0, T1 and T2 time points in lateral position.

If there was any significant drop in hemodynamic variables or NIRS values (10 % change from baseline), the cause will be identified and appropriate steps will be taken to correct the same.

Data Collection

Baseline Heart rate, systolic and diastolic blood pressure, oxygen saturation (SpO2), peak airway pressure (PIP), End tidal carbon dioxide (ETCO2) and Minimum alveolar concentration (MAC) of the inhalational agent were recorded at baseline (T0), after induction of anesthesia (T1) and 10 minutes after final positioning (T2)

NIRS, TCD velocity of MCA & ACA bilaterally in supine position before and after anesthesia induction and after the final surgical positioning were noted down. Data entry was done manually by the Principal Investigator into the study proforma.

STATISTICAL ANALYSIS

Sample Size Calculation

Assuming a maximum variation of within 10% from the recorded baseline value of change in NIRS and TCD flow velocity from supine to the final position, to be equivalent, with alpha as 5% and beta as 80%, the calculated average sample size for this study was 17 in each of the Group S, Group P and Group L. Considering the dropouts due to poor TCD window, a total of sixty patients were recruited with 20 patients in each group.

Statistical Methods

Statistical analyses were done using a statistical software package SPSS, version 20.0

Categorical and quantitative variables were expressed as frequency (percentage) and mean ± SD respectively. One way ANOVA test was used to compare quantitative parameters among categories. Repeated measure ANOVA test was used to compare quantitative parameters among

different time interval for each of the categories. Chi-square test was used to find association between categorical variables. Karl Pearson correlation was used to find out the relationship between quantitative variables. For all statistical interpretations, $p<0.05$ was considered the threshold for statistical significance. Statistical analyses was performed by using a statistical software package SPSS, version 20.0.

6. RESULTS & OBSERVATIONS

Results & Observations

The study was successfully completed in all the sixty patients without any dropouts. The results of our study are as follows:

1) Demographic Data

Table 1 shows the comparison of the demographic variables between the three groups.

Variable	Group S	Group P	Group L	p-value
	Total Number =20	Total Number =20	Total Number =20	
Age (years) Mean±SD	41.9±9.5	40.5±12.3	44.2±11.6	0.584
BMI (kg/m^2) Mean±SD	25.7±5.9	24±3.5	23±2.7	0.150
Gender (Male:Female) Number (%)	9:11 (45:55)	7:13 (35:65)	5:15 (25:75)	0.415
ASA Physical status (I:II:III:1V) Number (%)	(16:4) (80:20:0:0)	(16:4) (80:20:0:0)	(18:2) (90:10:0))	0.619
Comorbidity No (%) Yes (%)	16 (80) 4 (20)	16 (80) 4 (20)	18 (90) 2 (10)	0.619

Group S= Supine, Group P= Prone, Group L= Lateral, $P<0.05$ is Significant. Analysis by χ^2 test.

The demographic details of 60 patients enrolled in the study are shown in Table 1. The mean age was 41.9±9.5 years in Group S, 40.5±12.3 years in Group P and 44.2±11.6 years in Group L. There was no statistical difference between the groups.

The weight of the patients between the groups was comparable with a mean weight of 61.1±15.7 kg in Group S, 63.2±8.2 kg in Group P and 62.9±8.2 Kg in Group L. The Group S had mean BMI of 25.7±5.9, Group P had 24±3.5, and Group L had 23±2.7. There was no statistical difference between the groups.

There were 11 females out of 20 (55 %) in Group S and 13 females (65%) in Group P, and 15 females (75 %) in Group L indicating that most patients were females in all 3 groups. There was no statistical difference between the groups.

Group S, had 16 patients (80%) in ASA I, 4 patients (20%) in ASA II. Group P had 16 patients (80%) in ASA I, 4 patients (20%) in ASA II. Group L had 18 patients (90%) in ASA I, 2 patients (10%) in ASA II, indicating most patients belonging to ASA I in all three groups. None of the patients were in ASA III or IV. There was no statistical difference between the groups.

In Group S, 4 patients (20%) had comorbidity (one each having Diabetes Mellitus, Systemic hypertension, hypothyroidism, bronchial asthma). In Group P also 4 patients (20%) had comorbidity (one each having Diabetes Mellitus, Systemic hypertension, hypothyroidism, bronchial asthma). In Group L, 2 patients (10%) had comorbidity (one each having Diabetes Mellitus, Systemic hypertension).

2) Tumor characteristics of patients in three study groups

Table 2 shows the tumor characteristics of enrolled 20 patients in supine position (Group S).

Diagnosis	Number (N=20)	Percentage (%)
Meningioma	7	35
Glioma	6	30
Cavernoma	3	15
Focal cortical dysplasia	1	5
Colloid Cyst	1	5
Trigeminal Neuralgia	1	5
Metastasis	1	5

In the supine position, 7 patients were operated for meningioma (35%), 6 for glioma (30%), these two accounting for the majority in this group. 3 patients underwent cavernoma resection (15%).

The other tumors were focal cortical dysplasia (5%) Colloid Cyst (5%), Trigeminal Neuralgia (5%).

Table 3 shows the tumor characteristics of enrolled 20 patients in prone position (Group P).

Diagnosis	Number (N=20)	Percentage (%)
Meningioma	4	20
Schwannoma	4	20
Prolapsed Intervertebral disc (PIVD)	3	15
Arnold-Chiari malformation	3	15
Vertebral Body Lesion	2	10
Cerebellar Hemangioblastoma	2	10
Cavernoma	1	5
Pineal region tumor	1	5

In the prone position, 4 patients each were operated for meningioma (20%) and Schwannoma (20%), these two accounting for the majority in this group. 3 patients were operated for prolapsed intervertebral disc (15%), 3 patients underwent foramen magnum decompression for Arnold-Chiari malformation (15%). 2 patients each underwent vertebral body lesion (10%) and cerebellar hemangioblastoma (10%) excision. The other tumors included cavernoma (5%) and pineal region tumors (5%).

Table 4 shows the tumor characteristics of enrolled 20 patients in Lateral/Park-Bench position (Group L).

Diagnosis	Number (N=20)	Percentage (%)
Cerebellar-pontine angle Schwannoma	16	80
Cerebellar-pontine angle Meningioma	2	10
Cerebellar-pontine angle Epidermoid	1	5
Thalamic Glioma	1	5

Most common tumor in Group L was Cerebellar-pontine angle schwannoma with 16 patients (80%), Cerebellopontine angle meningioma 2 patients (10%), 1 patient each of epidermoid (5%) and glioma (5%).

3) Changes in Supine group (Group S)

1) Hemodynamic parameters (Heart rate, Mean arterial pressure) in supine group is shown in table 5.

Table 5 shows the changes in Heart Rate & MAP at different time intervals in Supine group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
Heart rate (beats/min) (Mean±SD)	71.3 ± 8.91	76 ± 9.71	75.6 ± 7.85	6.59	<0.01	-0.53	1.000	6.03	0.002
MAP (mmHg) (Mean±SD)	94.85 ± 8.39	88.3 ± 7.57	89.65 ± 7.96	-6.91	<0.01	1.53	0.666	-5.48	0.001

Repeated measure ANOVA. P<0.05 is Significant.

In Group S, the mean baseline heart rate was 71.3±8.91 per minute which increased to 76±9.71 per minute after induction of anesthesia and decreased slightly to 75.6±7.85 per minute after the final positioning. The change in heart rate from T0 to T1 was statistically significant but overall changes were less than 10 %. The percentage change from baseline (T0 to T2) was~ 6%, though it was statistically significant.

The MAP at baseline was 94.85 ± 8.39 mm Hg, which decreased to 88.3 ± 7.57 mm Hg and settled at 89.65 ± 7.96 mm Hg after the final positioning. The change in MAP from T0 to T1 (~7%) was statistically significant but overall changes were less than 10 %. The percentage change from baseline (T0 to T2) was~ 5.5%, though it was statistically significant.

2) **Ventilatory parameters (Tidal volume, Peak airway pressure, Respiratory rate, EtCO2) in Supine group is shown in table 6**

Table 6 shows the Ventilatory parameters at different time intervals in Supine group

	T1	T2	p-value*
Tidal Volume (ml) (Mean±SD)	459.8 ± 58.9	459.8 ± 58.9	NS
Airway Pressure (cmH2O) (Mean±SD)	19.5 ± 1.8	21.2 ± 2.2	p<0.01
Respiratory Rate (per minute) (Mean±SD)	12.2 ± 0.9	12.2 ± 0.9	NS
EtCO2 (mmHg) (Mean±SD)	34.4 ± 0.8	34.4 ± 0.8	NS

*Paired t-test, NS-Not significant. P<0.05 is Significant.

The baseline tidal volume in the supine group was 459.8 ± 58.9 ml, and was maintained the same till T2. The baseline airway pressure was 19.5 ± 1.8 cm H2O after induction which became 21.2 ± 2.2 mmHg after final positioning in Group S. Though this change in airway pressure was statistically significant, the percentage change was 9 ± 4.8 %. The baseline respiratory rate at T1 was 12.2 ± 0.9 per minute which again was maintained same at time point T2. The baseline EtCO2 was 34.4 ± 0.8 mmHg after induction which was remained same to 34.4 ± 0.8 mmHg after final positioning in Group S.

3) Cerebral Oxygenation data with NIRS.

Cerebral oxygenation data with NIRS is shown in table 7.

Table 7 shows the changes in NIRS (L) & NIRS (R) at different time intervals in Supine group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
NIRS (L) (Mean±SD)	67.6 ± 7.43	68.2 ± 5.53	68.35 ± 5.83	0.89	1.000	0.22	1.000	1.11	1.000
NIRS (R) (Mean±SD)	69.9 ± 5.7	70.05 ± 4.88	70.7 ± 5.55	0.21	1.000	0.92	1.000	1.14	1.000

Repeated measure ANOVA. P<0.05 is Significant.

The baseline NIRS on the left side was 67.6 ± 7.43 in group S before induction of anesthesia which minimally increased to 68.2 ± 5.53 after induction and 68.35 ± 5.83 after the final positioning. The baseline NIRS on the right side was 69.9 ± 5.7 in group S before induction of anesthesia which minimally increased to 70.05 ± 4.88 after induction and 70.7 ± 5.55 after the final positioning. All the NIRS (L and R) changes in supine group were statistically insignificant.

4) Cerebral Blood Flow Data with TCD.

Cerebral Blood Flow Data (Mean flow velocity of MCA & ACA) with TCD is shown in table 8 & 9.

Table 8 showing the changes in MCA MFV (L) & ACA MFV (L) at different time intervals in Supine group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
MCA (L) MFV (cm/sec) (Mean±SD)	45.35 ± 16.31	35.82 ± 11.81	30.83 ± 10.95	-21.01	0.016	-13.94	0.135	-32.03	0.006
ACA (L) MFV (cm/sec) (Mean±SD)	35.25 ± 9.82	31.64 ± 17.48	28.17 ± 8.75	-10.24	1.000	-10.98	1.000	-20.10	0.067

Repeated measure ANOVA. P<0.05 is Significant.

The baseline MCA MFV on left side was 45.35 ± 16.31 cm/sec, which decreased significantly to 35.82 ± 11.81 cm/sec. The decrease was to the tune of 21 %. And it was statistically significant as well (p -value 0.016). After final positioning the MCA MFV further decreased to 30.83 ± 10.95 cm/sec. This decrease was ~14 % from T1 but was not statistically significant. When considering the change from baseline to T2, it was nearly 32% decrease, and statistically significant as well (p -value 0.006).

The baseline ACA MFV on left side was 35.25 ± 9.82 cm/sec, which decreased significantly to 31.64 ± 17.48 cm/sec. The decrease seen was around 10.24 %, and it was not statistically significant. After final positioning the ACA MFV further decreased to 28.17 ± 8.75 cm/sec. This decrease was ~11 % from T1 and was also not statistically significant. When comparing from baseline, the change to T2 was nearly 20% decrease.

Table 9 showing the changes in MCA MFV (R) & ACA MFV (R) at different time intervals in Supine group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
MCA (R) MFV (cm/sec) (Mean±SD)	42.6 ± 10.98	37.2 ± 14.21	30.19 ± 11.47	-12.67	0.365	-18.84	0.061	-29.12	0.001
ACA (R) MFV (cm/sec) (Mean±SD)	36.05 ± 10.62	31.91 ± 13.62	28.12 ± 12.48	-18.70	0.025	-8.17	1.000	-25.34	0.018

Repeated measure ANOVA. P<0.05 is Significant.

The baseline MCA MFV on the right side was 42.6 ± 10.98 cm/sec, which decreased to 37.2 ± 14.21 cm/sec. The decrease was to the tune of 12.67 %, but it was not statistically significant. After final positioning the MCA MFV further decreased to 30.19 ± 11.47 cm/sec. This decrease was 18.84 % from T1 but was not statistically significant. When considering the change from baseline (T0) to T2, it was nearly 29% decrease, which was statistically significant (p-0.001).

The baseline ACA MFV on right side was 36.05 ± 10.62 cm/sec, which decreased significantly to 31.91 ± 13.62 cm/sec. The decrease seen was around 18.7 %, and it was statistically significant (p-value 0.025). After final positioning the ACA MFV further decreased to 28.12 ± 12.48 cm/sec. This decrease was nearly 8.17 % from T1 and was not statistically significant. When comparing from baseline, the change to T2 was nearly 25% decrease, which was statistically significant (p-0.018).

4) Changes in Prone Group (Group P)

1) Hemodynamic parameters (Heart rate, Mean arterial pressure) in prone group is shown in table 10.

Table 10 shows the changes in Heart Rate & MAP at different time intervals in Prone group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
Heart rate (beats,min) (Mean±SD)	73.8 ± 9.85	79.1 ± 10.36	77.6 ± 8.79	7.18	p<0.01	-1.90	0.502	5.15	0.005
MAP (mmHg) (Mean±SD)	92.05 ± 5.16	86.8 ± 5.55	86.85 ± 4.17	-5.70	p<0.01	0.06	1.000	-5.65	p<0.01

Repeated measure ANOVA. P<0.05 is Significant.

In Group P, the mean baseline heart rate was 73.8 ± 9.85 per minute which increased to 79.1 ± 10.36 per minute after induction of anesthesia and decreased slightly to 77.6 ± 8.79 per minute after the final positioning. The change in heart rate from T0 to T1 was statistically significant (p<0.01) though the changes were less than 10 %. Percentage changes from T1 to T2 (1.9 % change) was not statistically significant. The percentage change from T0 to T2 was ~5 % and statistically significant (p-0.005).

The MAP at baseline was 92.05 ± 5.16 mm Hg, which decreased to 86.8 ± 5.55 mm Hg and settled at 86.85 ± 4.17 mm Hg after the final positioning. The change in MAP from T0 to T1 (5.7%) was

statistically significant (p<0.01) and T1 to T2 (0.06 %) was not statistically significant. The percentage change from T0 to T2 was 5.65 % and statistically significant (p<0.01).

2) **Ventilatory parameters (Tidal volume, Peak airway pressure, Respiratory rate, EtCO2) in prone group is shown in table 11**.

Table 11 shows the Ventilatory parameters at different time intervals in Prone group

	T1	T2	p-value*
Tidal Volume (ml) (Mean±SD)	438.5 ± 42.8	438.5 ± 42.8	NS
Airway Pressure (cmH2O) (Mean±SD)	19.3 ± 1.5	21.2 ± 2.2	p<0.01
Respiratory Rate (per minute) (Mean±SD)	12 ± 0	12 ± 0	NS
ETCO2 (mmHg) (Mean±SD)	34.4 ± 0.7	34.4 ± 0.7	NS

*Paired t-test, NS-Not significant.

The baseline tidal volume in the prone group was 438.5 ± 42.8 ml, and was maintained the same till time point T2. The baseline airway pressure was 19.3 ± 1.5 cm H2O after induction which became 21.2 ± 2.2 mmHg after final positioning in Group P. Though this change in airway pressure was statistically significant, the percentage change was 13 ± 7.3 %. The baseline respiratory rate at T1 was 12 per minute which again was maintained same at time point T2. The baseline EtCO2 was 34.4 ± 0.7 mmHg after induction which remained same to 34.4 ± 0.7 mmHg after final positioning in Group P.

3) Cerebral Oxygenation data with NIRS.

Cerebral Oxygenation data with NIRS is shown in table 12.

Table 12 shows the changes in NIRS (L) & NIRS (R) at different time intervals in Prone group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
NIRS (L) (Mean±SD)	67.8 ± 6.58	66.85 ± 5.21	64.65 ± 6.03	-1.40	1.000	-3.29	0.190	-4.65	0.076
NIRS (R) (Mean±SD)	67.9 ± 5.69	65.5 ± 5.86	65.4 ± 5.61	-3.53	0.162	-0.15	1.000	-3.68	0.291

Repeated measure ANOVA. P<0.05 is Significant.

The baseline NIRS on the left side was 67.8 ± 6.58 in group S before induction of anesthesia which minimally decreased to 66.85 ± 5.21 after induction and further dropped to 64.65 ± 6.03 after the final positioning. The baseline NIRS on the right side was 67.9 ± 5.69 in group S before induction of anesthesia which minimally increased to 65.5 ± 5.86 after induction and 65.4 ± 5.61 after the final positioning. All the NIRS (L and R) changes in prone group were statistically insignificant.

4) Cerebral Blood Flow Data with TCD.

Cerebral Blood Flow Data (Mean flow velocity of MCA & ACA) with TCD is shown in table 13 & 14.

Table 13 showing the changes in MCA MFV (L) & ACA MFV (L) at different time intervals in prone group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
MCA (L) MFV (cm/sec) (Mean±SD)	40.87 ± 12.04	32.54 ± 11.37	25.71 ± 9.93	-20.38	0.009	-20.98	0.046	-37.09	p<0.01
ACA (L) MFV (cm/sec) (Mean±SD)	36.05 ± 10.62	31.91 ± 13.62	28.12 ± 12.48	-11.48	0.804	-11.89	0.986	-22.01	0.025

Repeated measure ANOVA. P<0.05 is Significant.

The baseline MCA MFV on left side was 40.87 ± 12.04 cm/sec, which decreased significantly to 32.54 ± 11.37 cm/sec. The decrease was to the tune of 20.3 % and it was statistically significant as well (p -value 0.009). After final positioning the MCA MFV further decreased to 25.71 ± 9.93 cm/sec. This decrease was ~21 % from T1 and was statistically significant (p-value 0.046). When considering the change from baseline to T2, it was nearly 37.09 % decrease which was statistically significant (p<0.01).

The baseline ACA MFV on left side was 36.05 ± 10.62 cm/sec, which decreased significantly to 31.91 ± 13.62 cm/sec. The decrease seen was around 11.48 %, and it was not statistically significant. After final positioning the ACA MFV further decreased to 28.12 ± 12.48 cm/sec. This decrease was 11.8 % from T1 and was also statistically insignificant. When comparing from baseline, the change to T2 was nearly 22% decrease which was statistically significant (p-0.025).

Table 14 showing the changes in MCA MFV (R) & ACA MFV (R) at different time intervals in prone group

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
MCA (R) MFV (cm/sec) (Mean±SD)	40.87 ± 11.31	35.27 ± 8.89	24.9 ± 8.37	-13.70	0.052	-29.41	0.001	-39.08	p<0.01
ACA (R) MFV (cm/sec) (Mean±SD)	40.09 ± 15.11	29.99 ± 10.42	24.13 ± 7.12	-25.19	0.015	-19.54	0.083	-39.81	<0.001

Repeated measure ANOVA. P<0.05 is Significant.

The baseline MCA MFV on the right side was 40.87 ± 11.31 cm/sec, which decreased to 35.27 ± 8.89 cm/sec. The decrease was around 13.7 %, but it was not statistically significant. After final positioning the MCA (R) MFV further decreased to 24.9 ± 8.37 cm/sec. This decrease was 29.41 % from T1 and was statistically significant (p-value 0.001). When considering the change from baseline to T2, it was nearly 39% decrease, and statistically significant (p-value <0.01).

The baseline ACA MFV on right side was 40.09 ± 15.11 cm/sec, which decreased significantly to 29.99 ± 10.42 cm/sec. The decrease seen was around 25 %, and it was statistically significant (p-

value 0.015). After final positioning the ACA MFV further decreased to 24.13 ± 7.12 cm/sec. This decrease was nearly 19.5 % from T1. When comparing from baseline, the change to T2 was nearly 39% decrease and statistically significant (p<0.001).

5) Changes in Lateral/Park Bench Group (Group L)

1) Hemodynamic parameters (Heart rate, Mean arterial pressure) in Lateral/Park bench group is shown in table 15.

Table 15 shows the changes in Heart Rate & MAP at different time intervals in Group L

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
Heart rate (beats/min) (Mean±SD)	71.95 ± 8.77	77.35 ± 9.04	76.6 ± 7.89	7.51	p<0.01	-0.97	1.000	6.46	p<0.01
MAP (mmHg) (Mean±SD)	90.7 ± 9.89	86.55 ± 8.95	88.35 ± 8.07	-4.58	0.004	2.08	0.358	-2.59	0.508

Repeated measure ANOVA. P<0.05 is Significant.

In Group L, the mean baseline heart rate was 71.95 ± 8.77 per minute which increased to 77.35 ± 9.04 per minute after induction of anesthesia and decreased slightly to 76.6 ± 7.89 per minute after the final positioning. The percentage change in heart rate from T0 to T1 was statistically significant (p-value <0.01) though T1 to T2 was statistically insignificant. When comparing from baseline, the change from T1 to T2 was nearly 6.46 % decrease and statistically significant (p<0.01).

The MAP at baseline was 90.7 ± 9.89 mm Hg, which decreased to 86.55 ± 8.95 mm Hg and settled at 88.35 ± 8.07 mm Hg after the final positioning. The change in MAP from T0 to T1 (-4.5%) was found to be statistically significant (p-0.004) but T1 to T2 (2.08 %) was not statistically significant. When comparing from baseline, the change from T1 to T2 was nearly 2.59 % decrease and was not statistically significant.

2) **Ventilatory parameters (Tidal volume, Peak airway pressure, Respiratory rate, EtCO2) in Lateral/Park bench are shown in table 16**.

Table 16 shows the Ventilatory parameters at different time intervals in Group L

	T1	T2	p-value*
Tidal Volume (ml) (Mean±SD)	433.8 ± 43.8	433.8 ± 43.8	NS
Airway Pressure (cmH20) (Mean±SD)	19.4 ± 1.7	21.4 ± 2.2	p<0.01
Respiratory Rate (per minute) (Mean±SD)	12 ± 0	12 ± 0	NS
ETCO2 (mmHg) (Mean±SD)	34.1 ± 0.8	34.1 ± 0.8	NS

*Paired t-test, NS-Not significant. P<0.05 is Significant.

The baseline tidal volume in the prone group was 438.5 ± 42.8 ml, and was maintained the same till time point T2. The baseline airway pressure was 19.4 ± 1.7 cm H2O after induction which became 21.4 ± 2.2 mmHg after final positioning in Group L. This change in airway pressure was statistically significant (p<0.01), and the percentage change was 10 ± 4%. The baseline respiratory rate at T1 was 12 per minute which was maintained same at time point T2. The baseline EtCO2 was 34.1 ± 0.8 mmHg after induction which remained same to 34.1 ± 0.8 mmHg after final positioning in Group L.

2) Cerebral Oxygenation data with NIRS.

Cerebral Oxygenation data with NIRS is shown in table 17.

Table 17 shows the changes in NIRS (L) & NIRS (R) at different time intervals in Group L

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
NIRS (L) (Mean±SD)	66.3 ± 6.41	66.15 ± 5.63	66.3 ± 6.25	-0.23	1.000	0.23	1.000	0.00	1.000
NIRS (R) (Mean±SD)	66.8 ± 4.62	65.2 ± 5.85	64.2 ± 6.09	-2.40	0.658	-1.53	1.000	-3.89	0.341

Repeated measure ANOVA. P<0.05 is Significant.

The baseline NIRS on the left side was 66.3 ± 6.41 in group S before induction of anesthesia which was nearly same to 66.15 ± 5.63 after induction and remained nearly same to 66.3 ± 6.25 after the final positioning. The baseline NIRS on the right side was 66.8 ± 4.62 in group S before induction of anesthesia which minimally decreased to 65.2 ± 5.85 after induction and 64.2 ± 6.09 after the final positioning. The changes in NIRS were statistically insignificant.

3) Cerebral Blood Flow Data with TCD.

Cerebral Blood Flow Data (Mean flow velocity of MCA & ACA) with TCD is shown in table 18 & 19.

Table 18 showing the changes in MCA MFV (L) & ACA MFV (L) at different time intervals in Group L

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
MCA (L) MFV ((cm/sec)) (Mean±SD)	44.23 ± 12.99	36.24 ± 11.46	31.09 ± 12.6	-18.06	0.067	-14.21	0.279	-29.71	0.002
ACA (L) MFV ((cm/sec)) (Mean±SD)	34.72 ± 9.37	32.16 ± 10.48	27.17 ± 9.09	-7.37	0.889	-15.52	0.131	-21.75	0.012

Repeated measure ANOVA. P<0.05 is Significant.

The baseline MCA MFV on left side was 44.23 ± 12.99 cm/sec, which decreased significantly to 36.24 ± 11.46 cm/sec. The decrease was in the range of 18.06 %, but it was statistically insignificant. After final positioning the MCA MFV further decreased to 31.09 ± 12.6 cm/sec. This decrease was 14.2 % from T1 and was again statistically not significant. When considering the change from baseline T0 to T2, it was nearly 29.71 % decrease, which was statistically significant (p-0.012).

The baseline ACA MFV on left side was 34.72 ± 9.37 cm/sec, which decreased to 32.16 ± 10.48 cm/sec. The decrease seen was around 7.37 %, and it was not statistically significant. After final positioning the ACA MFV further decreased to 27.17 ± 9.09 cm/sec. This decrease was 15.52 % from T1 and was also statistically insignificant. When comparing from baseline T0 to T2, the change was 21.7% decrease which was statistically significant (p-0.012).

Table 19 showing the changes in MCA MFV (R) & ACA MFV (R) at different time intervals in Group L

	T0	T1	T2	(T0 -T1)		(T1-T2)		(T0-T2)	
				% Change	p-value	% Change	p-value	% Change	p-value
MCA (R) MFV ((cm/sec)) (Mean±SD)	38.61 ± 10.24	35.69 ± 11.86	27.3 ± 8.12	-7.55	0.991	-23.52	0.005	-29.30	0.001
ACA (R) MFV ((cm/sec)) (Mean±SD)	37.4 ± 10.19	32.47 ± 12.21	28.19 ± 14.62	-13.18	0.299	-13.18	0.528	-24.63	0.038

Repeated measure ANOVA. P<0.05 is Significant.

The baseline MCA MFV on right side was 38.61 ± 10.24 cm/sec, which decreased to 35.69 ± 11.86 cm/sec. The decrease was around 7.5 %, and it was not statistically significant. After final positioning the MCA MFV further decreased to 27.3 ± 8.12 cm/sec. This decrease was nearly 23.5 % from T1 and was statistically significant (p-0.005). When considering the change from baseline T0 to T2, it was nearly 29.3 % decrease, and was statistically significant (p-0.001)

The baseline ACA MFV on right side was 37.4 ± 10.19 cm/sec, which decreased to 32.47 ± 12.21 cm/sec. The decrease seen was around 13.18 %, and it was not statistically significant. After final positioning, the ACA MFV further decreased to 28.19 ± 14.62 cm/sec. This decrease was 13.18 % from T1 and was also not statistically significant. When compared to baseline T0, the change to T2 was nearly 246% decrease, which was statistically significant (0.038).

3) Correlation of the percentage changes from T0 (baseline) to T1 (after induction) in NIRS with percentage changes in Heart rate & MAP

Table 20 showing the correlation of the percentage changes from T0 (baseline) to T1 (after induction) in NIRS (L) with percentage changes in heart rate & MAP

	Supine		Prone		Lateral	
	r	p	r	p	r	p
Heart Rate	-0.089	0.710	0.109	0.648	0.31	0.184
MAP	-0.3	0.199	0.069	0.772	-0.095	0.690

The correlation data of NIRS (L) with heart rate and MAP for all 3 groups shows there is no correlation between NIRS (L) with heart rate and MAP from T0 to T1.

Table 21 showing the correlation of the percentage changes from T0 (baseline) to T1 (after induction) in NIRS (R) with percentage changes in heart rate & MAP

	Supine		Prone		Lateral	
	r	p	r	p	r	p
Heart Rate	0.023	0.922	0.335	0.149	0.06	0.802
MAP	-0.395	0.085	0.381	0.098	-0.053	0.824

The correlation data of NIRS (L) with heart rate and MAP for all 3 groups shows there is no correlation between NIRS (L) with heart rate and MAP from T0 to T1.

Thus, at T0 to T1, NIRS values on both sides were not found to have any correlation with heart rate and MAP.

4) Correlation of the percentage changes from T1 (after induction) to T2 (after positioning) in NIRS with percentage changes in Heart rate & MAP

Table 22 showing the correlation of the percentage changes from T1 (after induction) to T2 (after positioning) in NIRS (L) with percentage changes in heart rate & MAP

	Supine		Prone		Lateral	
	r	p	r	p	r	p
Heart Rate	-0.12	0.613	0.039	0.870	-0.013	0.957
MAP	0.531*	0.016	0.22	0.352	0.273	0.245

The correlation data of NIRS (L) with heart rate and MAP from T1 to T2 shows that the correlation coefficient of MAP (in Group S) is 0.531 with a significant p-value, which implies moderately positive correlation. The same can be appreciate from the scatter plot of NIRS (L) with MAP in Figure 6. Except this none of the groups demonstrated any significant correlation from T1 to T2 with heart rate and MAP.

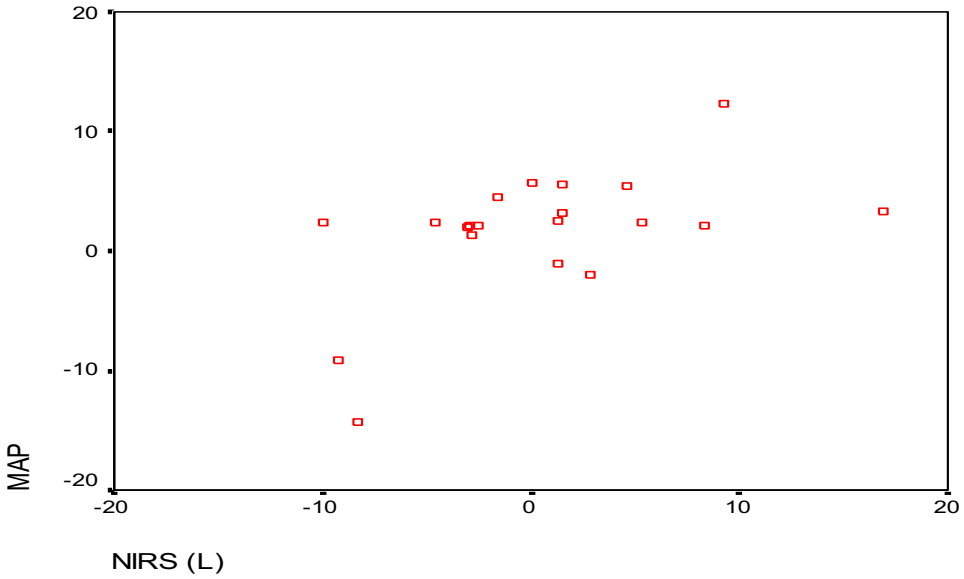


Figure 16: Scatter diagram for correlation of the percentage changes from T1 (after induction) to T2 (after positioning) in NIRS (L) with percentage changes in Heart rate & MAP

Table 23 showing the correlation of the percentage changes from T1 (after induction) to T2 (after positioning) in NIRS (R) with percentage changes in Heart rate & MAP

	Supine		Prone		Lateral	
	r	p	r	p	r	p
Heart Rate	0.055	0.818	0.274	0.243	-0.048	0.841
MAP	0.366	0.113	0.178	0.453	0.021	0.931

The correlation data of NIRS (R) with heart rate and MAP from T1 to T2 for all 3 groups shows no correlation of NIRS (R) with heart rate and MAP from T1 to T2.

Thus, at T1 to T2 time point, NIRS (R) values were not found to have any correlation with heart rate and MAP, though a moderately positive correlation was seen in NIRS (L) with MAP. No correlation was seen with heart rate though.

5) Correlation of the percentage changes from T0 (baseline) to T1 (after induction) in MCA and ACA with percentage changes in Heart rate & MAP

Table 24 showing the correlation of the percentage changes from T0 (baseline) to T1 (after induction) in MCA and ACA with percentage changes in Heart rate.

		Supine		Prone		Lateral	
		r	P	r	p	r	p
MCA(L)	Mean flow velocity	-0.209	0.376	0.027	0.909	0.385	0.094
MCA(R)	Mean flow velocity	-0.009	0.970	0.087	0.714	0.294	0.209
ACA (L)	Mean flow velocity	-0.011	0.963	0.338	0.145	-0.019	0.935
ACA(R)	Mean flow velocity	0.042	0.860	-0.265	0.259	0.247	0.293

The correlation data of MCA and ACA mean flow velocity with heart rate and MAP for all 3 groups shows there is no correlation of MCA and ACA mean flow velocity with heart rate from T0 to T1. Thus, at T0 to T1, MCA and ACA MFV on both sides were not found to have any correlation with heart rate.

Table 25 showing the correlation of the percentage changes from T0 (baseline) to T1 (after induction) in MCA and ACA with percentage changes in MAP.

		Supine		Prone		Lateral	
		r	p	r	p	r	p
MCA(L)	Mean flow velocity	0.268	0.253	0.11	0.643	0.221	0.350
MCA(R)	Mean flow velocity	0.207	0.380	-0.13	0.584	0.079	0.741
ACA (L)	Mean flow velocity	-0.067	0.780	0.526*	0.017	-0.038	0.873
ACA(R)	Mean flow velocity	-0.043	0.857	-0.213	0.368	-0.191	0.420

The correlation data of MCA and ACA MFV with MAP for all 3 groups is shown above. There is a moderately positive correlation of ACA (L) mean flow velocity with MAP (in Group P) from T0 to T1 (r=0.526) (p-0.017). None of the other groups demonstrated any correlation with MAP from T0 to T1.

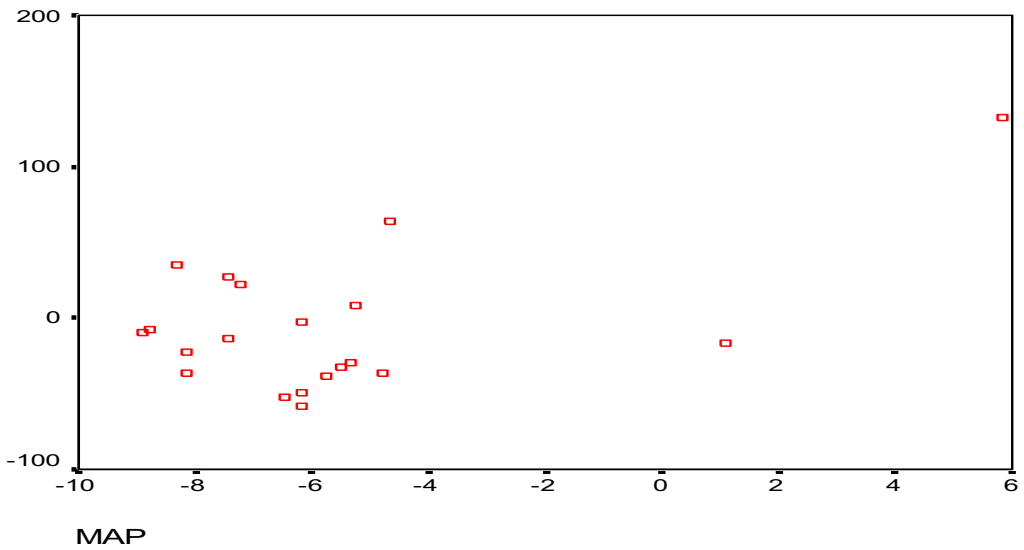


Figure 17: Scatter diagram for correlation of the percentage changes from T0 (baseline) to T1 (after induction) in ACA (L) MFV with percentage changes in MAP.

5) Correlation of the percentage changes from T1 (after induction) to T2 (after positioning) in MCA and ACA with percentage changes in Heart rate & MAP

Table 26 showing the correlation of the percentage changes from T1 (after induction) to T2 (after positioning) in MCA and ACA with percentage changes in Heart rate.

		Supine		Prone		Lateral	
		r	p	r	p	r	p
MCA(L)	Mean flow velocity	-0.116	0.627	0.142	0.550	0.019	0.936
MCA(R)	Mean flow velocity	0.08	0.737	-0.153	0.521	0.029	0.903
ACA (L)	Mean flow velocity	0.093	0.695	-0.043	0.857	0.054	0.822
ACA(R)	Mean flow velocity	0.009	0.969	-0.087	0.714	0.011	0.963

From T1 to T2, there is no correlation of MCA and ACA mean flow velocity with heart rate. Thus, from T1 to T2, the changes occurring in MFV of MCA and ACA were independent of the changes in HR.

Table 27 showing the correlation of the percentage changes from T1 (after induction) to T2 (after positioning) in MCA and ACA with percentage changes in MAP.

		Supine		Prone		Lateral	
		r	p	r	p	r	p
MCA(L)	Mean flow velocity	-0.287	0.219	0.245	0.298	-0.017	0.945
MCA(R)	Mean flow velocity	0.015	0.951	0.065	0.786	0.178	0.452
ACA (L)	Mean flow velocity	-0.292	0.211	0.173	0.466	-0.104	0.663
ACA(R)	Mean flow velocity	0.47*	0.036	-0.074	0.756	-0.094	0.695

The correlation data of MCA and ACA MFV with MAP for all 3 groups is shown above. There is a weakly positive correlation of ACA (R) mean flow velocity with MAP (in Group S) from T1 to T2 (r=0.47, p-0.036). None of the other groups demonstrated any correlation with MAP from T1 to T2.

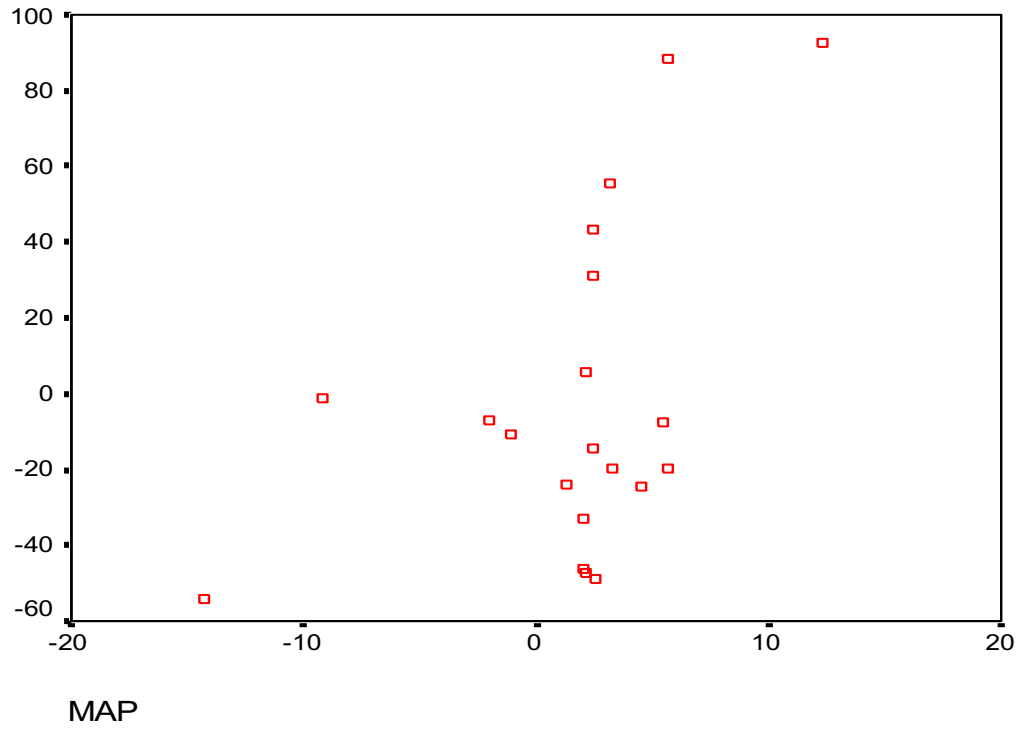


Figure 18: Scatter diagram for correlation of the percentage changes from T1 (after induction) to T2 (post positioning) in ACA (R) MFV with percentage changes in MAP.

4) COMPARISON OF PARAMETERS BETWEEN THE 3 GROUPS

A. Comparison of Heart Rate between the 3 Groups

Table 28 shows the changes in Heart Rate between the three study groups at different time intervals.

HEART RATE (beats/min)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value
T0	71.3 ± 8.91	73.8±9.85	71.95±8.77	0.673
T1 (% change from T0)	76 ± 9.71 (6.59 ± 3.9)	79.1±10.36 (7.18 ± 2.4)	77.35±9.04 (7.51 ± 2.6)	0.602
T2 (% change from T1)	75.6±7.85 (-0.53 ± 5.8)	77.6±8.79 (-1.93 ± 5.3)	76.6±7.89 (-0.98 ± 4.6)	0.743

Group S= Supine, Group P=Prone, Group L= Lateral, P<0.05 is Significant. Test- Repeated measure ANOVA.

In Group S, the mean baseline heart rate was 71.3 ± 8.91 per minute which increased to 76 ± 9.71 per minute after induction and to 75.6±7.85 per minute after the final surgical positioning. In Group P, the mean baseline heart rate was found to be 73.8±9.85/minute which increased to 79.1±10.36 per minute after anesthesia and to 77.6± 8.79/minute after prone positioning. In Group L, the mean baseline heart rate was found to be 72±8.8/minute which increased to 77.6±8.8/minute after anesthesia and to 76.6±7.9/minute after final positioning. The changes in heart rate were not statistically significant between the time periods recorded and were comparable between the three groups.

B. Comparison of Mean Arterial Pressure between the 3 Groups

Table 29 shows the changes in Mean arterial Pressure (MAP) between the three study groups at different time intervals

Mean arterial pressure (mmHg)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value
T0	94.85 ± 8.39	92.05 ± 5.16	90.7 ± 9.89	0.087
T1 (% change from T0)	88.3 ± 7.57 (-6.91 ± 1.6)	86.8 ± 5.55 (-5.7 ± 3.5)	86.55 ± 8.95 (-4.58 ± 5.8)	0.728
T2 (% change from T1)	89.65 ± 7.96 (1.51 ± 5.5)	86.85 ± 4.17 (0.06 ± 4.3)	88.35 ± 8.07 (2.04 ± 6.2)	0.451

Group S= Supine, Group P= Prone, Group L= Lateral, P<0.05 is Significant. Test- Repeated measure ANOVA.

The MAP in group S before induction was 94.85 ± 8.39 mmHg before induction which became 88.3 ± 7.57 mmHg after anesthesia and 89.65 ± 7.96 mmHg after final positioning. The maximum percentage change was only -6.9±1.6 %. The group P had a baseline MAP of 92.05 ± 5.16 mmHg which had become 86.8 ± 5.55 mmHg after induction, but the percentage change being -5.7±3.5 %. The baseline MAP in Group L was 90.7 ± 9.89 mmHg which decreased to 86.55 ± 8.95 mmHg after induction and finally settled to a MAP of 88.35±8.07 mmHg, the maximum percentage change being -4.58±5.8 %, which was within the maximum allowable change of 10% from the baseline values. The MAP characteristics at T0, T1 and T2 time points were comparable between the groups and statistically not significant.

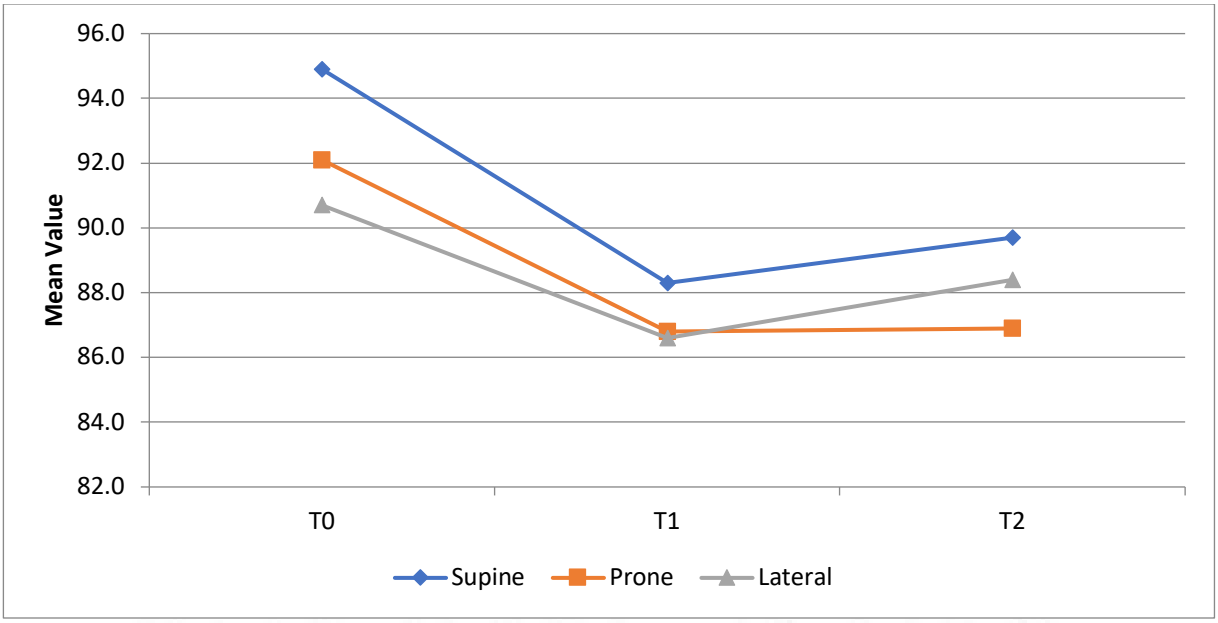


Fig 19. Comparison of MAP in three groups at different time intervals

C. Comparison of EtCO2 and Peak Airway Pressure between the 3 Groups

Table 30 shows the Capnography (EtCO2) between the three study groups at different time intervals.

ETCO2 (mmHg)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value
T1	34.4 ± 0.8	34.4 ± 0.7	34.1 ± 0.8	0.332
T2	34.4 ± 0.8	34.4 ± 0.7	34.1 ± 0.8	0.332

P<0.05 is Significant. Test- Repeated measure ANOVA. Group S= Supine, Group P= Prone, Group L= Lateral.

The baseline EtCO2 was 34.4 ± 0.8 mmHg after induction which was maintained to 34.4 ± 0.8 mmHg after final positioning in Group S. The EtCO2 in group P was 34.4 ± 0.7 mmHg which was maintained even after prone positioning. Similarly, in Group L, the baseline EtCO2 was 34.1 ± 0.8 mmHg which had not changed at T2 time point. The EtCO2 values between the three groups were found to be statistically insignificant.

Table 31 shows the Peak airway pressure between the three study groups at different time intervals.

Peak Airway Pressure (cm H2O)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value#
T1	19.5 ± 1.8	19.3 ± 1.5	19.4 ± 1.7	0.924
T2	21.2 ± 2.2	21.8 ± 2.1	21.4 ± 2.2	0.712

Group S= Supine, Group P= Prone, Group L= Lateral, P<0.05 is Significant. # ANOVA Test

The baseline airway pressure was 19.5 ± 1.8 cmH2O after induction which increased to 21.2 ± 2.2 mmHg after the final surgical position in Group S. In group P, it was 19.3 ± 1.5 cmH2O which increased to 21.8 ± 2.1 cmH2O after prone positioning. In Group L, it was 19.4 ± 1.7 cmH2O which had also increased to 21.4 ± 2.2 cmH2O at time point T2. The airway pressure between the three groups was statistically insignificant.

D. Comparison of NIRS between the 3 Groups

Table 32 shows the comparison of NIRS (L) between the three groups at different time intervals.

NIRS (L)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value*
T0	67.6 ± 7.4	67.8 ± 6.6	66.3 ± 6.4	0.753
T1 (% change from T0)	68.2 ± 5.5 (0.89 ± 5.7)	66.9 ± 5.2 (-1.4 ± 9.4)	66.2 ± 5.6 (0.23 ± 8)	0.487
T2 (% change from T1)	68.4 ± 5.8 (0.22 ± 6.6)	64.7 ± 6 (-3.4 ± 7.2)	66.3 ± 6.3 (0.23 ± 6.6)	0.161

P<0.05 is Significant. Test- Repeated measure ANOVA. Group S= Supine, Group P=Prone, Group L= Lateral,

The baseline NIRS on the left side was 67.6 ± 7.4 in group S before induction of anesthesia which minimally increased to 68.2 ± 5.5 after induction and 68.4 ± 5.8 after the final positioning. The baseline NIRS on the left side was 67.8 ± 6.6 in group P which decreased to 66.9 ± 5.2 after anesthesia and further decreased to 64.7 ± 6 after the final prone positioning. In Group L, the

baseline NIRS was 66.3 ± 6.4 which had decreased to 66.2 ± 5.6 and had remained almost same to 66.3 ± 6.3. The maximum percentage change noted in the NIRS was a decrease of 3.4 % in Group P (within maximum allowable change of 10%). The NIRS (Left) was comparable between the three groups.

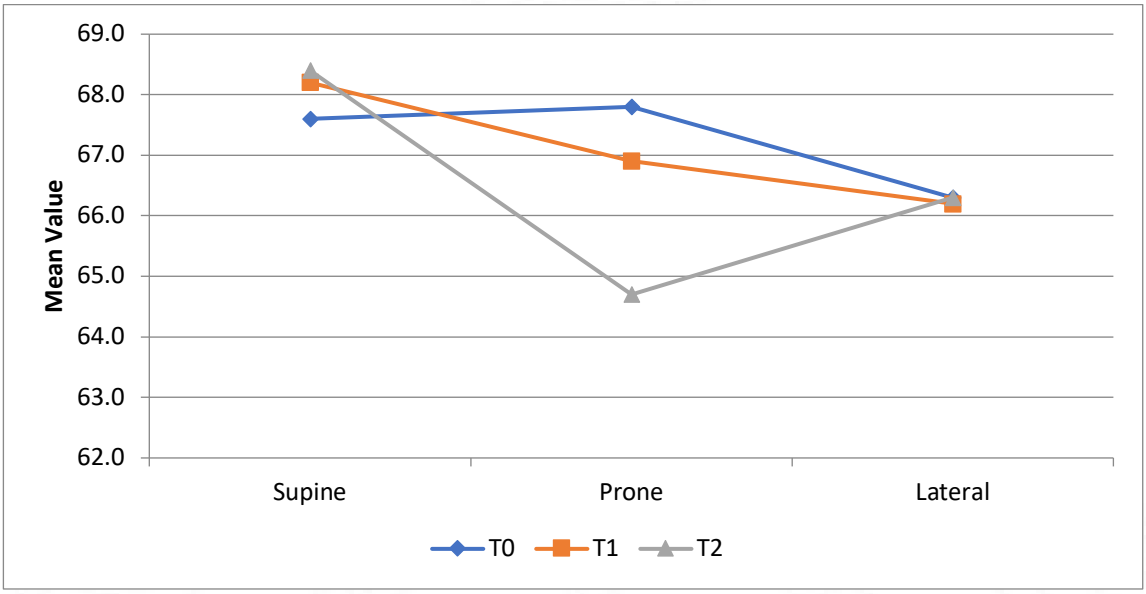


Fig 20: Comparison of NIRS (L) in three groups at different time intervals.

Table 33 shows the comparison of NIRS (R) between the three groups at different time intervals.

NIRS (R)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value#
T0	69.9 ± 5.7	67.9 ± 5.7	66.8 ± 4.6	0.189
T1 (% change from T0)	70.1 ± 4.9 (0.21 ± 6.5)	65.5 ± 5.9 (-3.53 ± 7.9)	65.2 ± 5.9 (-2.4 ± 8.2)	0.012
T2 (% change from T1)	70.7 ± 5.5 (0.92 ± 9)	65.4 ± 5.6 (0.15 ± 7.7)	64.2 ± 6.1 (-1.56 ± 8.6)	0.002

P<0.05 is Significant. Test- Repeated measure ANOVA. Group S= Supine, Group P=Prone, Group L= Lateral,

The baseline NIRS on the right side was 69.9 ± 5.7 in group S before induction of anesthesia which minimally increased to 70.1 ± 4.9 after induction and 70.7 ± 5.5 after the final positioning. The baseline NIRS on the right side was 67.9 ± 5.7 in group P which decreased to 65.5 ± 5.9 after anesthesia and remained nearly stable to 65.4 ± 5.6 after assuming the final prone position. In Group L the baseline NIRS value was 66.8 ± 4.6 which had decreased to 65.2 ± 5.9 and had dropped further to 64.2 ± 6.1. Though The NIRS (R) was statistically significant among groups at T1 and T2 time intervals, the maximum percentage change noted in the NIRS was a decrease of 3.53 % in Group P.

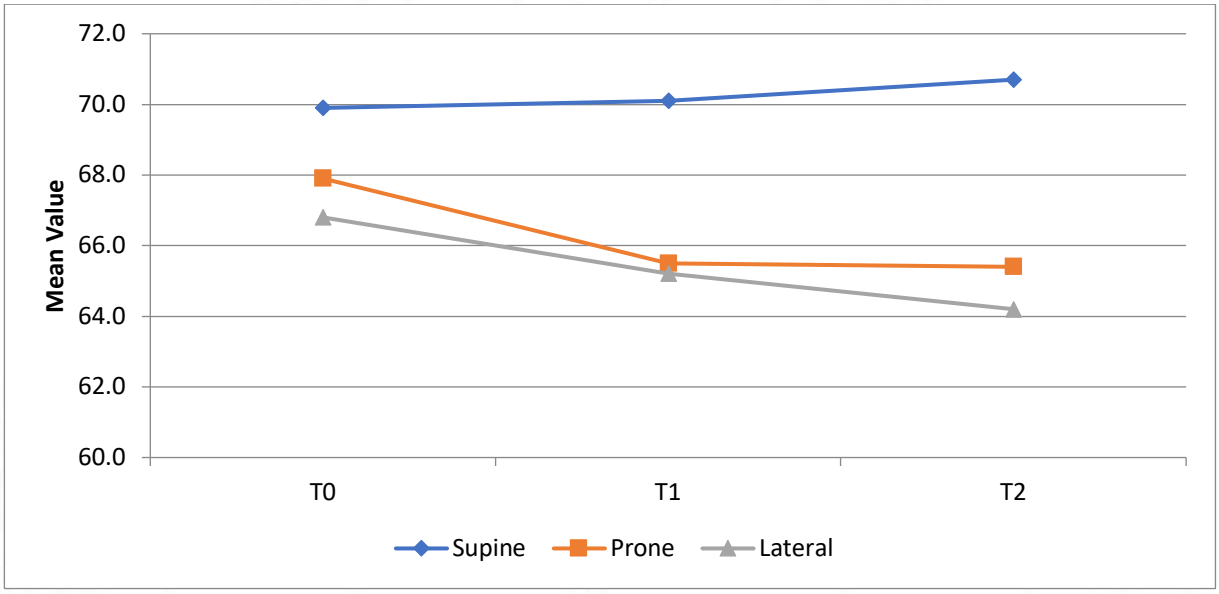


Fig 21: Comparison of NIRS (R) in three groups at different time intervals.

D. Comparison of Mean Flow Velocity between the 3 Groups

Table 34 shows the comparison of (L) MCA Mean flow velocity (MFV) between the three groups at different time intervals.

MCA MFV (L) (cm/sec)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value#
T0	45.4 ± 16.3	40.9 ± 12	44.2 ± 13	0.572
T1 (% change from T0)	35.8 ± 11.8 (-21.01 ±25.3)	32.5 ± 11.4 (-20.38 ± 22.6)	36.2 ± 11.5 (-18.06 ± 28.9)	0.543
T2 (% change from T1)	30.8 ± 10.9 (-16.20 ± 30)	25.7 ± 9.9 (-26.55 ± 34.9)	31.1 ± 12.6 (-16.56 ± 40.3)	0.240

P<0.05 is Significant. Test- Repeated measure ANOVA. Group S= Supine, Group P=Prone, Group L= Lateral,

The MCA MFV (L) was comparable between the three groups and was not statistically significant. In group S, the baseline MCA MFV (L) was 45.4 ±16.3 cm/sec which reduced to 35.8±11.8 cm/sec after induction and further reduced to 30.8±10.9 cm/sec after final positioning. In group P, the baseline MCA MFV (L) was 40.9 ±12 cm/sec which reduced to 32.8±11.4 cm/sec after induction and further reduced to 25.7± 9.9 cm/sec after final positioning. In group L, the baseline MCA MFV (L) was 44.2 ±13 cm/sec which reduced to 36.2±11.5 cm/sec after induction and further reduced to 31.1±12.6 cm/sec after final positioning.

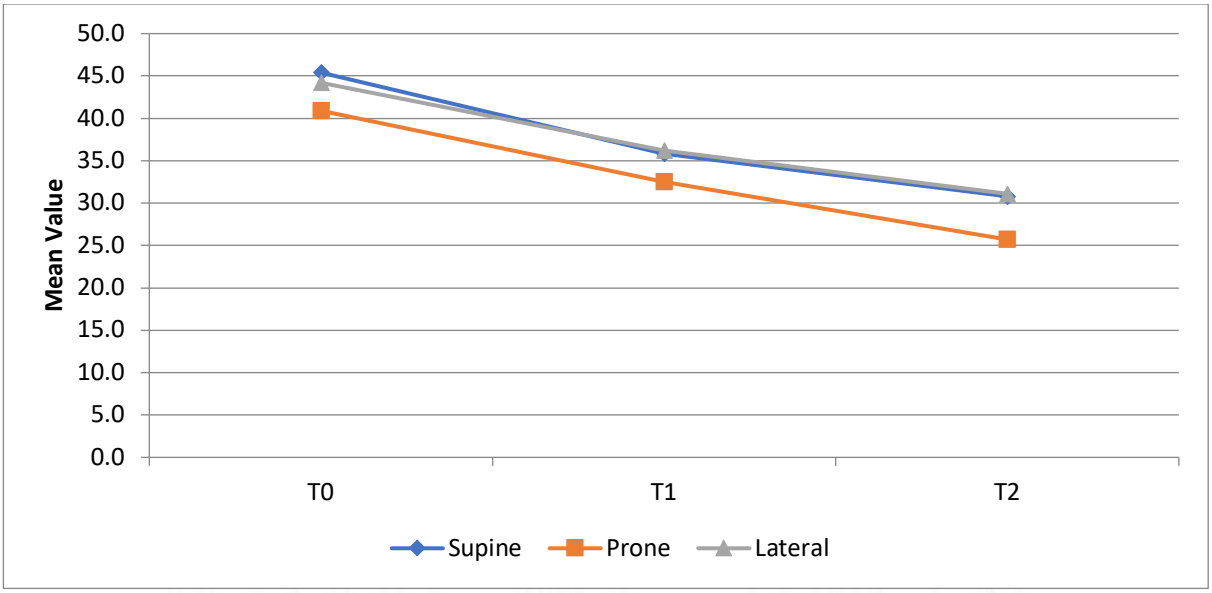


Fig 22: Comparison of MCA(L) Mean flow velocity in three groups at different time intervals

Table 35 shows the comparison of (R) MCA Mean flow velocity (MFV) between the three groups at different time intervals.

MCA MFV (R) (cm/sec)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value#
T0	42.6 ± 11	40.9 ± 11.3	38.6 ± 10.2	0.511
T1 (% change from T0)	37.2 ± 14.2 (-12.67 ± 39.3)	35.3 ± 8.9 (-13.7 ± 25.2)	35.7 ± 11.9 (-7.5 ± 38.6)	0.864
T2 (% change from T1)	30.2 ± 11.5 (-23.21 ±29.9)	24.9 ± 8.4 (-41.65 ± 21.7)	27.3 ± 8.1 (-30.76 ± 22.5)	0.215

P<0.05 is Significant. Test- Repeated measure ANOVA. Group S= Supine, Group P=Prone, Group L= Lateral,

In group S, the baseline MCA MFV (R) was 42.6 ± 11 cm/sec which reduced to 37.2 ± 14.2 cm/sec after induction and further reduced to 30.2±11.5 cm/sec after final positioning. In group P, the baseline MCA MFV (R) was 40.9 ± 11.3 cm/sec which reduced to 35.3 ± 8.9 cm/sec after induction and further reduced to 24.9 ± 8.4 cm/sec after final positioning. In group L, the baseline MCA MFV (R) was 38.6 ± 10.2 cm/sec which dropped to 35.7 ± 11.9 cm/sec after induction and further

reduced to 27.3 ± 8.1 cm/sec after final positioning. The MCA MFV (R) was comparable between the three groups and was not statistically significant.

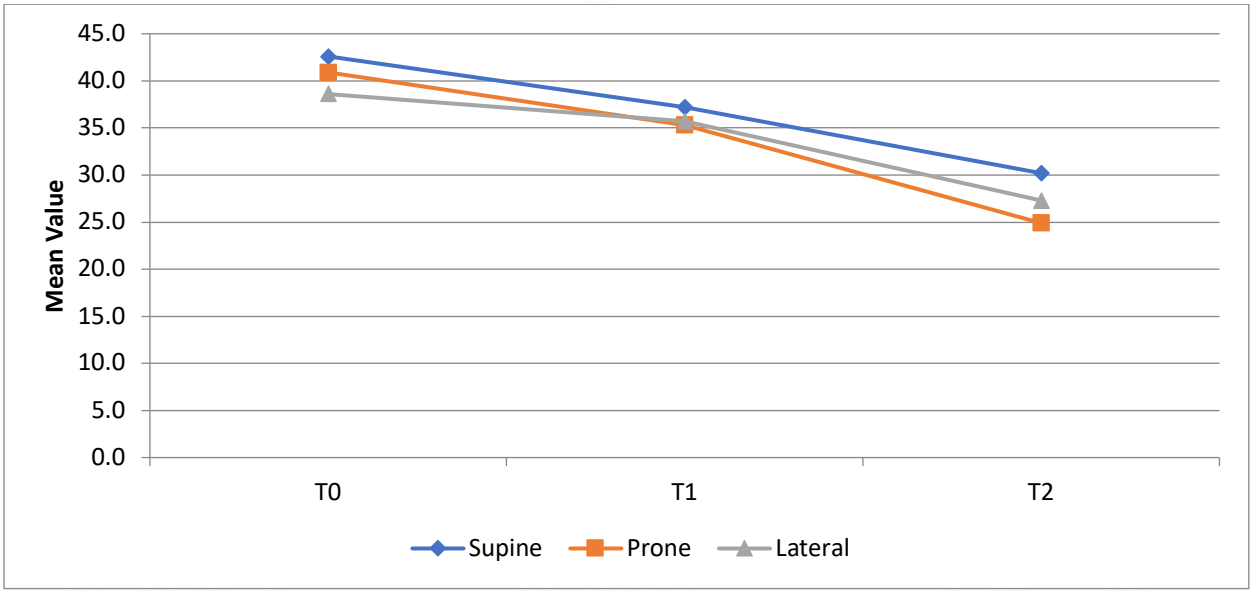


Fig 23: Comparison of MCA(R) Mean flow velocity in three groups at different time intervals.

Table 36 shows the comparison of (L) ACA Mean flow velocity (MFV) between the three groups at different time intervals.

ACA MFV (L) (cm/sec)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value#
T0	35.3 ± 9.8	36.1 ± 10.6	34.7 ± 9.4	0.913
T1 (% change from T0)	31.6 ± 17.5 (-10.24 ± 45.4)	31.9 ± 13.6 (-11.48 ± 45.4)	32.2 ± 10.5 (-7.37 ± 25.2)	0.993
T2 (% change from T1)	28.2 ± 8.7 (12.34 ± 42.6)	28.1 ± 12.5 (-13.5 ± 51.3)	27.2 ± 9.1 (-18.37 ± 28.8)	0.941

P<0.05 is Significant. Test- Repeated measure ANOVA. Group S= Supine, Group P=Prone, Group L= Lateral,

In group S, the baseline ACA MFV (L) was 35.3 ± 9.8 cm/sec which reduced to 31.6 ± 17.5 cm/sec after induction and further reduced to 28.2 ± 8.7 cm/sec after final positioning. In group P, the baseline ACA MFV (L) was 36.1 ± 10.6 cm/sec which reduced to 31.9 ± 13.6 cm/sec after induction and further reduced to 28.1 ± 12.5 cm/sec after final positioning. In group L, the baseline

ACA MFV (L) was 34.7 ± 9.4 cm/sec which dropped to 32.2 ± 10.5 cm/sec after induction and further reduced to 27.2 ± 9.1 cm/sec after final positioning. The ACA MFV (R) was comparable between the three groups and was not statistically significant.

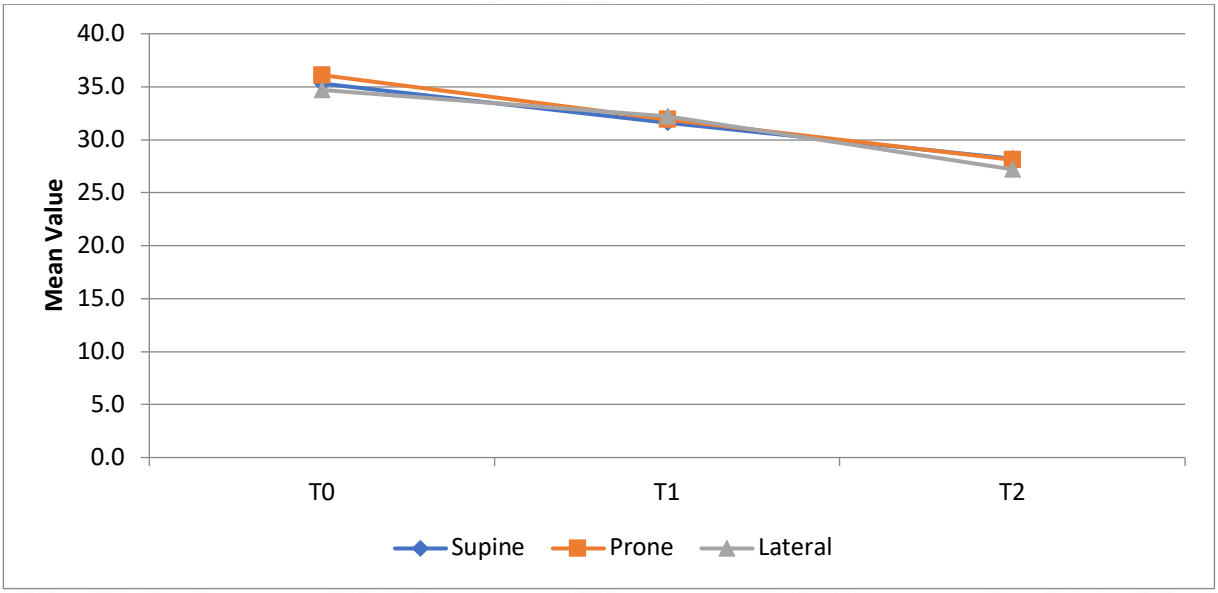


Fig 24: Comparison of ACA (L) Mean flow velocity in three groups at different time intervals.

Table 37 shows the comparison of (R) ACA Mean flow velocity (MFV) between the three groups at different time intervals.

ACA MFV (R) (cm/sec)	Group S (N=20) Mean±SD	Group P (N=20) Mean±SD	Group L (N=20) Mean±SD	p-value#
T0	36.6 ± 12.5	40.1 ± 15.1	37.4 ± 10.2	0.660
T1 (% change from T0)	29.7 ± 11.4 (-18.70 ±33.3)	30 ± 10.4 (-25.19 ± 28.9)	32.5 ± 12.2 (-13.18 ± 33.9)	0.703
T2 (% change from T1)	27.3 ± 14.4 (-8.90 ± 43.2)	24.1 ± 7.1 (-24.29 ± 28.3)	28.2 ± 14.6 (-15.2 ± 31.4)	0.564

P<0.05 is Significant. Test- Repeated measure ANOVA. Group S= Supine, Group P=Prone, Group L= Lateral,

In group S, the baseline ACA MFV (R) was 36.6 ± 12.5 cm/sec which reduced to 29.7 ± 11.4 cm/sec after induction and further reduced to 27.3 ± 14.4 cm/sec after final positioning. In group P, the baseline ACA MFV (R) was 40.1 ± 15.1 cm/sec which reduced to 30 ± 10.4 cm/sec after induction and further reduced to 24.1 ± 7.1 cm/sec after final positioning. In group L, the baseline ACA MFV (R) was 37.4 ± 10.2 cm/sec which dropped to 32.5 ± 12.2 cm/sec after induction and further reduced to 28.2 ± 14.6 cm/sec after final positioning. The ACA MFV (R) was comparable between the three groups and was not statistically significant.

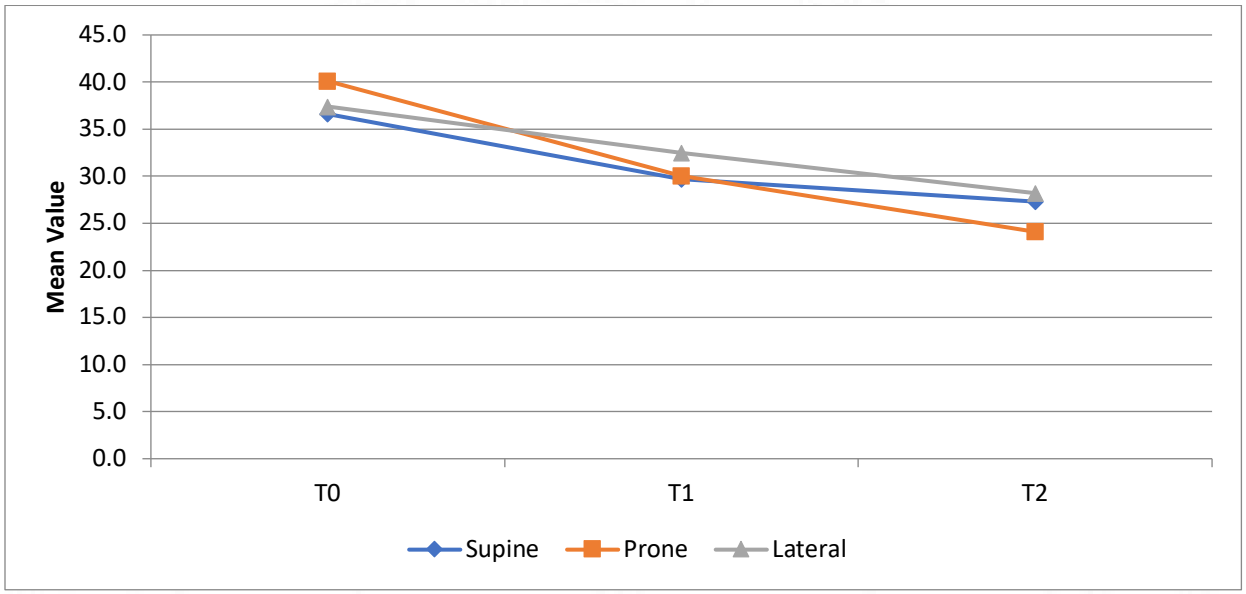


Fig 25. Comparison of ACA (R) Mean flow velocity in three groups at different time intervals

7.DISCUSSION

Discussion

Neurosurgical procedures necessitates intraoperative positioning of patients as per the location of the pathology and the surgical approach. The essential objective of perioperative neurosurgical positioning is to give better working conditions during the surgical procedure, providing adequate surgical exposure and maintaining cerebral hemodynamics and to keep up with typical body arrangement without causing injury. Numerous physical and physiological changes can happen to the patient due to surgical positioning. (1) In supine position venous return may be affected by head-up or head-down tilt. Head-down positioning may cause increase in venous return to the heart but at the expense of causing venous congestion in upper part of the body. Thus, to avoid this venous congestion neurosurgery patients are frequently given head-up tilt. Prone position also has been associated with perturbations in systemic hemodynamics. Prone position causes increase in intraabdominal pressure which leads to reduction in venous return to heart and increases in systemic and pulmonary vascular resistance. (58) Lateral position alters the ventilation-perfusion relationship in the lungs. The non-dependent lungs are better ventilated than the dependent portions of the lungs which are better perfused. This potentiates ventilation-perfusion mismatch in lateral position. In lateral position, marked flexion of the legs can lead to reduction in venous return. There is paucity of data for the changes in cerebral blood flow velocity and cerebral oxygenation during various surgical positioning assumed for neurosurgery under anesthesia. Literature search showed the available data on patient positioning is focused on the healthy volunteers and awake state.

Our study is aimed to understand the effects of patient positioning during elective neurosurgery on cerebral hemodynamics using TCD and NIRS. We initially hypothesized that positions adopted

for various neurosurgical procedures from supine to either lateral or prone positions causes significant changes in cerebral oxygenation and cerebral blood flow velocities compared to neurosurgical procedures performed in supine posture under anesthesia, as evaluated using NIRS and TCD.

Based on our study results, we found significant changes in the cerebral blood flow velocities and cerebral oxygenation in all the 3 neurosurgical positions (supine, prone and lateral), though the changes were of higher magnitude in prone and lateral group when compared to supine position.

Induction of anesthesia can affect the mean CBF. In a study of forty patients who underwent faciomaxillary or spinal surgery, induction of anesthesia with either propofol or sevoflurane caused decrease in mean flow of MCA velocity. Moreover, higher doses of sevoflurane (2-4%) impaired the autoregulation and caused increase in mean flow velocity. (59) We also noted similar decrease in the MFV following induction of anesthesia and the concentration of Sevoflurane was less than 1.8.

In another study of patients with supratentorial tumors, the investigators noted a fall in MAP of 17% for sevoflurane and 23% for propofol group from baseline after tracheal intubation and the mean MCA FV decreased to 21% in sevoflurane and 26% in Propofol group. (60) In our study we noted the MAP to decrease by 4-6% from baseline to T1 in all the groups, but greater fall in the mean FV of MCA of 32-36% from T0 to T1 than reported in their study.

One of the primary goals in neurosurgery is maintenance of CPP. The mean arterial pressure is an important factor in assessment of CPP due to the relationship CPP=MAP-ICP. However the relationship between the blood pressure and cerebral blood flow is not linear. In a study, Holzer et

al studied the effects of Systemic blood flow velocity (SBFv) on Cerebral blood flow velocity (CBFv). (61) This was done by calculating the cerebral/systemic blood flow velocity-index (CsvI). A CsvI value of 100 indicating a 1 : 1 relationship between CBFv and SBFv. The study showed that propofol anaesthesia produced a significantly greater reduction in CsvI than did sevoflurane anaesthesia [propofol: 60 (19); sevoflurane: 83 (16), p = 0.009, t-test]. The authors concluded that a direct reduction in CBFv occurred independent of SBFv during propofol anaesthesia implying lower cerebral metabolic demand with propofol.

B) <u>Changes Observed in Supine Position:</u>

Supine position is viewed as physiological among all the positions utilized in neurosurgery, in light of the fact that the effect of position is least on the cerebral blood flow, cerebral venous drainage and ICP. (62) Usually, neurosurgeons place the patients in supine position with mild HOB elevated to $15\text{-}30^0$ along with the head fixed in Mayfield clamps with the aim to prevent increase in the ICP, facilitate CSF and cerebral venous drainage, to provide optimal relaxation of brain, minimize blood loss and to provide a clear surgical field. CBF is considered to be minimally affected if the above position is employed whereas higher degrees of HOB elevation have shown to reduce the CBF due to higher gradient between the heart and brain for forward flow and venous return due to collapse of the jugular veins by gravity. (63) In contrast, we found alterations in TCD and NIRS values under anesthesia and final position compared to awake state.

In our study in the supine group, the NIRS values increased marginally on the both sides from baseline from awake state to anesthesia, but negligible change in final position. The maximum

percentage change was seen from T1 to T2 time point in both left (1.11%) & right (1.14 %) side NIRS. Similar increase in NIRS was also seen in study by Iwasaki et al. (22) The authors found significant increase in oxyhemoglobin concentration with sevoflurane. Lovell et al., reported a similar increase in both oxygenated hemoglobin and total hemoglobin concentration with propofol. (64)

Effect of positioning on cerebral oxygenation has been studied by Kurihara et al. (65) The authors studied the effects of postural changes on cerebral oxygenation by spatially resolved spectroscopy (SRS)-NIRS. Cerebral oxyhemoglobin (O2Hb), deoxyhemoglobin (HHb), and the TOI were recorded bilaterally from forehead in 5 healthy male volunteers during 90° head-up tilt (HUT) and 6° head-down tilt (HDT). 3 series of data were recorded on separate days. The authors found that during HUT, O2Hb value was found to decrease. TOI was also found to be significantly reduced in HUT when compared with the supine position. In contrast to this, no significant change was noted in TOI with HDT. Though this study was done in awake patients, nonetheless it gives insight into effect of position on cerebral oxygenation.

Arjen Mol et al. performed a study on 15 healthy young adults using NIRS and evaluated cerebral oxygenated hemoglobin (O_2Hb), deoxygenated hemoglobin (HHb) and tissue saturation index (TSI). (39) These parameters were measured bilaterally on the forehead using NIRS. Sitting to standing, and slow and rapid supine to standing movements were assessed in their study. Both oxygenated and deoxygenated hemoglobin dropped in the early phase after standing up, indicating a lower concentration of total hemoglobin, therewith reflecting a decrease of cerebral perfusion. Their study found that both the oxygenated and deoxygenated hemoglobin dropped in the early phase after standing up, indicating a lower concentration of total hemoglobin, therewith reflecting

a decrease of cerebral perfusion. The minimum O2Hb response in their study was found to be most sensitive to postural changes and showed significant differences ($p < 0.001$) between standing up from sitting and supine position.

Our study did not show any significant changes in NIRS on either side in supine position. The maximum percentage change seen in NIRS was 1.1 % on left side, and 1.14 % on right side. Both these maximum changes were seen after positioning (T2) and both were not statistically significant.

Ludovico, et al., studied 16 healthy volunteers to evaluate if transient systemic blood pressure changes are reflected in brain NIRS values. (66) Authors performed two independent manipulations while recording data from the visual cortex. Cortical hemodynamic responses were induced by visual stimulation using reversing checkerboards of varying duration. Simultaneously, transient blood pressure changes were generated by rapid arm raising which was independent of visual stimulation. The authors concluded that transient systemic blood pressure changes were indeed reflected in brain NIRS values, and that the blood pressure effects should always be taken into account whenever reporting NIRS.

In our study, the NIRS (L) values showed no correlation with HR (r=0.089, p-0.71) or MAP (r=0.3, p-0.199) at T0 to T1 time points. The NIRS (R) values showed no correlation with HR (r=0.023, p-0.922) or MAP (r=0.395, p-0.085) at T0 to T1 time points. From time point T1 to T2, the NIRS (L) values showed no correlation with HR (r=0.12, p-0.613), though a moderately positive correlation with MAP was seen (r=0.531, p-0.016). The NIRS (R) values showed no correlation with HR (r=0.055, p-0.818) or MAP (r=0.366, p-0.113).

We believe that the difference in response seen in our study could be due to the study conducted in anesthesia state (where brain activity is suppressed with consequently higher systemic oxygenation) where modest changes in MAP did not affect the NIRS values.

In another study, Ledwith et. al studied the effects of body position on cerebral oxygenation. (44) This study used brain tissue oxygen ($PbtO_2$) for monitoring cerebral oxygenation. They studied the effects of 12 different body positions on neurodynamic and hemodynamic outcomes in 33 patients. The patient population included patients affected by traumatic brain injury (TBI), subarachnoid hemorrhage (SAH), or craniotomy for tumor excision. Cerebral oxygenation changes were evaluated and simultaneously ICP was also calculated. The study evaluated 12 different positions, which were as follows: supine, supine with knee bent, left lateral position, and right lateral position, and in each of these positions, the head of bed was then elevated to 15^0, 30^0, or 45^0. Changes from baseline to the 15-minute postposition assessment mean change scores were calculated which showed a downward trend for PbtO2 for all positions with statistically significant decreases observed for supine head of bed (HOB) elevated 30^0 and 45^0 ($p < .01$) and right and left lateral positioning HOB 30^0 ($p < .05$). ICP decreased with supine HOB 45^0 ($p < .01$) and knee elevation, HOB 30^o and 45^o ($p < .05$), and increased ($p < .05$) with right and left lateral HOB 15^o. Hemodynamic parameters were comparable in the various positions. The lateral position was found to have the most adverse effect on intracranial physiology. This study though utilized $PbtO_2$ (and not NIRS) for measuring cerebral oxygenation, nonetheless gives important insight into the effect of positioning on cerebral oxygenation. Also of note is the fact that the patient population in this study included in addition to tumor, TBI & SAH, whereas our study included elective patients for tumor excision.

We noted significant changes in MFV of MCA and ACA. There was significant decrease noted in MFV post anesthetic induction (T1) which was highest in MCA (L), ~21% decrease. This decline could possibly be due to effect of anesthetic drugs, though the changes in hemodynamic parameters were less than 10% (decrease in MAP 6.91 %, and increase in HR by 6.6%) in the same period. This suggests that the changes in MFV occurred independent of the changes in hemodynamics, which was also confirmed by the correlation studies. From T0 to T1 no correlation of MFV of either MCA/ACA was seen with HR or MAP. After final positioning further fall in MFV was noted, maximum decrease noted in MCA (R) MFV (~19%). This additional decline in MFV could be due to added effect of head elevation after the final positioning in addition to the effect of anesthetic drugs. From T1 to T2 time point, a weak correlation was seen between ACA (R) MFV (r=0.47, p-0.036) and MAP, though percentage change seen in ACA (R) MFV was 8.17%.

Gulsah & Sevgi, have studied the effects of head & neck positions on the cerebral blood flow velocity by transcranial Doppler ultrasound in awake patients who underwent cranial surgery before surgery and postoperatively within 72 hours after surgery. (43) The MCA mean flow velocity was measured in supine position with 0° and 30° head elevations, as well as in right and left lateral positions, right and left lateral positions with head flexion and extension. The study found that the MCA mean flow velocity was increased in supine position with head elevations from 0° to 30°. In. contrast to their study, we noted a decrease in mean flow velocity bilaterally. This difference could be due to the fact that our study being done under anesthesia and with proper surgical position assumed. Similar changes were noted in right and lateral positions with 30° head elevations.

C) Changes Observed in Prone Position:

In the prone group, we found that the baseline NIRS on the left side was 67.8 ± 6.58 before induction of anesthesia which minimally decreased to 66.85 ± 5.55 after induction and further decreased to 64.65 ± 6.03 after the final positioning. The changes from T0 to T1 and to T2 were not statistically significant. The baseline NIRS on the right side was found to be 67.9 ± 5.69 before induction of anesthesia which minimally decreased to 65.5 ± 5.86 after induction and remained nearly the same at 65.4 ± 5.61 after the final positioning. The changes were also found to be statistically insignificant. The maximum percentage change was seen from T0 to T2 time point in both left (4.65%) & right (3.68 %) side NIRS.

The NIRS (L) values showed no correlation with HR (r=0.109, p-0.648) or MAP (r=0.069, p-0.772) at T0 to T1 time points. The NIRS (R) values as well showed no correlation with HR (r=0.0335, p-0.149) or MAP (r=0.381, p-0.098) at T0 to T1 time points.

From time point T1 to T2, the NIRS (L) values showed no correlation with HR (r=0.039, p-0.870) or MAP (r=0.22, p-0.352) and the NIRS (R) values also showed no correlation with HR (r=0.274, p-0.243) or MAP (r=0.178, p-0.453).

We noted significant changes in MFV of MCA and ACA. There was significant decrease noted in MFV post anesthetic induction (T1) which was highest in ACA (R), ~25% decrease. This decline could possibly be due to effect of anesthetic drugs, though the changes in hemodynamic parameters during the same period were less than 10% (decrease in MAP 5.7 %, and increase in HR by 7.18%) in the same period. This suggests that the changes in MFV occurred independent of the changes in hemodynamics, which was also confirmed by the correlation studies. From T0 to T1 no correlation

of MFV of either MCA/ACA was seen with HR. However, ACA (L) MFV showed a moderately positive correlation with MAP (r=0.526, p-0.017).

After final positioning further fall in MFV was noted, maximum decrease noted in MCA (R) MFV (~29.5%). This additional decline in MFV could be due to added effect of head elevation after the final positioning in addition to the effect of anesthetic drugs. From T1 to T2 time point, no correlation was seen between MFV of either MCA or ACA. Airway pressure and $EtCO_2$ were kept constant when turning from supine to prone position. The changes were recorded 10 minutes after the final positioning.

Our results were similar to the study by Jakob et al., though their study was done in awake healthy individuals (22 individuals). (42) The authors measured MAP, stroke volume (SV), and CO and MCA MFV, jugular vein diameters bilaterally in 3 positions with and without supplementation of 10 cmH2O CPAP: (1) supine, (2) prone with the head centered, (3) prone with the head rotated nearly∼80 degrees to the right. Their study found that during positive pressure breathing, the prone position with head rotated sideways lead to reduction in mean flow velocity of MCA by nearly 10% even when MAP is elevated. Even in our study MAP changed by maximum -5.70 % (From T0 to T1) and by 0.06% (from T1 to T2), but the changes observed in MFV were to the tune of 39 % from baseline. Prone positioning with rotated head likely leads affects both CBF and venous drainage (as seen by reduction in bilateral internal jugular vein diameters) implying that head should be well centered for optimal brain perfusion. These results may have implications for the anesthetized and ventilated patient. In all our cases head was well centered.

Thus, this study is probably the first one to evaluate the changes observed in cerebral oxygenation and flow velocity in prone position under anesthesia.

D) Changes Observed in Lateral/Park-Bench Position:

The lateral position is utilized as the surgical approach of choice for patients requiring skull base, posterior fossa and temporal lobe procedures.

In lateral/Park-bench position, we found that the baseline NIRS on the left side was 66.3 ± 6.41 before induction of anesthesia which minimally increased to 66.15 ± 5.63 after induction and was nearly the same after the final positioning at 66.3 ± 6.25. The changes from T0 to T1 and to T2 were not statistically significant. The baseline NIRS on the right side was found to be 66.8 ± 4.62 before induction of anesthesia which minimally decreased to 65.2 ± 5.85 after induction and marginally decreased to 64.2 ± 6.09 after the final positioning. The changes were also found to be statistically insignificant. The maximum percentage change was seen from T0 to T2 time point in right (-3.89 %) NIRS.

The NIRS (L) values showed no correlation with HR (r=0.31, p-0.184) or MAP (r=0.095, p-0.690) at T0 to T1 time points. The NIRS (R) values as well showed no correlation with HR (r=0.06, p-0.802) or MAP (r=0.053, p-0.824) at T0 to T1 time points. From time point T1 to T2, the NIRS (L) values showed no correlation with HR (r=0.013, p-0.957) or MAP (r=0.273, p-0.245) and the NIRS (R) values also showed no correlation with HR (r=0.248, p-0.841) or MAP (r=0.021, p-0.931). Thus, no correlation was found between NIRS at any time points during the study.

We noted significant changes in MFV of MCA and ACA. There was significant decrease noted in MFV post anesthetic induction (T1) which was highest in MCA (L), ~18% decrease. This decline could possibly be due to effect of anesthetic drugs, though the changes in hemodynamic parameters were less than 10% (decrease in MAP 4.58 %, and increase in HR by 7.5%) in the same

period. This suggests that the changes in MFV occurred independent of the changes in hemodynamics, which was also confirmed by the correlation studies. From T0 to T1 no correlation of MFV of either MCA/ACA was seen with HR or MAP. After final positioning further fall in MFV was noted, maximum decrease noted in MCA (R) MFV (~23.5%). This additional decline in MFV is possibly due to added effect of head elevation after the final positioning in addition to the effect of anesthetic drugs. From T1 to T2 time point also no correlation was seen between the MFV of either ACA or MCA.

As noted above in the study by Gulsah and Sevgi, wherein they studied cerebral blood flow velocity TCD in patients who underwent cranial surgery. (43) The MCA mean flow velocity of the patients was measured in supine position with 0° and 30° head elevations, as well as in right and left lateral positions, right and left lateral positions with head flexion and extension. The measurements were taken before surgery and postoperatively within 72 hours after surgery. The authors found that MCA MFV increased in the both right and left lateral positions with 30° head elevation, and it was found to reduce when flexion and extension positions were applied, though the changes in the MFV were not statistically significant. The same findings were seen in the postoperative period as well. This is in contrast to our study where we found a uniform reduction in MFV after induction and further reduction with the final lateral position. This difference could be explained by our study being done under anesthesia, whereas the authors conducted the study on awake patient preoperatively and 72 hours after surgery. So intraoperative effects of anesthesia and position as utilized for the surgery in head clamp per se were not done in the author's study.

Only one study has been done previously which simultaneously evaluated cerebral oxygenation and CBF with TCD, though done in awake patients. (67) This study was conducted in 35 subjects

(with 15 having history of orthostatic syncopes, 20 healthy controls). Tilt table was used for simulating orthostatic stress. The parameters recorded were NIRS, CBF velocity by TCD and hemodynamic data (Blood Pressure (BP), Heart Rate (HR)). In healthy subjects: the HR increased, the MAP remained unchanged, and the CBFV decreased. The NIRS demonstrated an increase in deoxyhemoglobin (HHb) and a decrease in oxyhemoglobin (O2Hb) as well as regional oxygen saturation. Patients had a significant and prominent decrease in arterial blood pressure ($p < 0.001$), CBFV ($p < 0.001$} and regional oxygen saturation ($p = 0.04$). This is in contrast to our study results wherein we found that changes in NIRS oxygenation were more or less insignificant, however, severe decrease in MFV was found in both ACA and MCA, even with less than 10 % change in MAP and HR.

In our study, a simultaneous assessment of CBF velocity and cerebral oxygenation during anesthesia and mechanical ventilation was carried out for the first time in neurosurgical patients. NIRS and TCD both non-invasive modalities are simple to use, easily repeatable, and can also be used for continuous real time monitoring. Another advantage of combining both the modalities is that both are complementary tools and monitor different anatomical levels. TCD gives information on CBF velocity of intracranial arteries, whereas NIRS provides information regarding cerebral oxygenation. (68, 69)

Although NIRS is considered as a sensitive monitor for detecting alterations in cerebral oxygenation and indirectly cerebral hemodynamics caused by positional changes, the results of our study indicate that this cannot be generalized especially in surgeries away from the frontal lobe. This is due to the fact that measurements of NIRS from forehead reflects only local oxygenation changes in a small volume of the frontal lobe only, which may not necessarily be

representative of the entire brain. This is where the role of TCD comes into play. TCD gives valuable information regarding the flow velocity in major intracranial blood vessels, which are representative of a more global change.

8. LIMITATIONS

Limitations of the Study

Our study has few following limitations;

1. Being operator dependent, TCD technique is prone for technical errors and inter observer variability. So, to avoid this limitation, a well-trained Neuroanesthesiologist for TCD, had determined all parameters. The TCD examinations were done by the PI and Co-PI in all cases.
2. Use of TCD is difficult in 10 -15% of patients due to poor acoustic window. We have excluded those who had poor TCD window in the preoperative examination.
3. We have not evaluated the autoregulation status of the patients. However, the changes in CBF in our study were higher than the changes in MAP. Moreover, we have not evaluated the clinical outcome measures.
4. We have not measured the actual CBF, but only the CBF velocity as a surrogate of CBF. As no methods are available for intraoperative use. Hence, we consider TCD is the best method considering its non-invasiveness and reproducibility.
5. NIRS measures only frontal oxygenation changes, so provides information regarding regional cerebral oxygenation changes only.
6. The NIRS & TCD data was recorded 10 minutes after the final position. Though this time has been established to be sufficient for hemodynamic equilibrium to occur, it would have been more useful to repeat the measurement at a later stage as well. This was not practical in our study as the neurosurgical procedures for craniotomy and excision warrants the sterile draping of the head and neck area leading to its inaccessibility at a later stage.

7. We have not studied the effect of sitting position on NIRS & TCD flow as the number of cases done in sitting posture in our center is of limited number.

However, in spite of these limitations, we found a significant reduction in cerebral blood flow velocity even with less than 10% change in heart rate and MAP, more so in the prone position than lateral and supine positions.

9. SUMMARY

SUMMARY

Various positions used in neurosurgical procedures can compromise the cerebral oxygenation and cerebral blood flow velocity. This can cause perturbations in intracranial physiology.

One of the essential objectives of neuroanaesthesia in the perioperative period is to give ideal position during neurosurgical procedures which would keep up with cerebral blood flow, cerebral oxygenation and provide with satisfactory cerebral venous drainage while forestalling any increment in ICP. This study was conducted to evaluate the effects of different positions used in neurosurgical patients and its effects on cerebral oxygenation and cerebral blood flow velocity under anesthesia and mechanical ventilation using NIRS and TCD. This prospective observational study included 20 patients in each of the three groups (total 60) with GCS 15, aged 18-60 years belonging to ASA I, II who underwent elective neurosurgery.

We found that changing the position of the patients from baseline to the final surgical position results in significant variations in cerebral blood flow velocity in all three positions routinely utilized in neurosurgery: supine, prone and lateral groups. These significant drops in MFVs were over and above the changes caused under anesthesia. Cerebral oxygenation did not change significantly whereas MCA/ACA MFV decreased significantly with maximum fall seen in prone position (41.6%), and almost equal changes in lateral (30.7%) supine (32%) positions. The decrease in MFV was not associated with concomitant decrease in HR or MAP.

We found that mean flow velocity as assessed by TCD is a useful and feasible technique to diagnose and quantify the position related variations in neurosurgical patients, which can pick up changes even when regional cerebral oxygenation by NIRS may fail to do so.

10. CONCLUSION

CONCLUSION

We undertook the present study to evaluate the hypothesis that patient positions adopted for various neurosurgical procedures from supine to either lateral or prone positions causes significant changes in cerebral oxygenation and cerebral blood flow velocities compared to neurosurgical procedures performed in supine posture under anesthesia, as evaluated using NIRS and TCD. We found that NIRS oxygenation values did not change much from the baseline in all three positions, however, significant decrease in mean flow velocity of MCA & ACA was seen in all three positions (Prone>Lateral=Supine).

Our study is probably the first one in neurosurgical patients to evaluate the effects of position on cerebral oxygenation & flow velocities under anesthesia using NIRS & TCD. Based on our study we found that TCD flow velocities showed a severe decrease despite the MAP maintained within the normal limits, thus making neurosurgical patients vulnerable to ischemia when only CPP thresholds and accordingly MAP is used for improving cerebral blood flow intraoperatively. Hence, we believe TCD monitoring may be used when the CBF needs to be maintained. Future studies are warranted using multimodal neuromonitoring to assess the well-being of neurosurgical patients intraoperatively.

11. BIBLIOGRAPHY

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12. APPENDICES

1. TAC FORM
2. IEC FORM
3. PATIENT INFORMATION SHEET ENGLISH
4. PATIENT INFORMATION SHEET MALAYALAM
5. CONSENT FORM ENGLISH
6. CONSENT FORM MALAYALAM
7. PROFORMA
8. MASTER CHART
9. PLAGIARISM REPORT

TAC FORM

Technical Advisory Committee (Clinical Studies)

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES & TECHNOLOGY
THIRUVANANTHAPURAM – 695011, INDIA

TAC Registration No: SCT-/S/2019/933 **Date: 17.06.2019**

Project title: AN EVALUATION OF CHANGES IN CEREBRAL OXYGENATION USING NEAR INFRARED SPECTROSCOPY DURING VARIOUS POSITIONS (SUPINE, PRONE & LATERAL) IN PATIENTS UNDERGOING ELECTIVE NEUROSURGERY – AN OBSERVATIONAL STUDY

Principal Investigator:
Dr. Ashutosh Kumar Degree: MBBS, MD (Anaesthesiology and Critical Care), PDCC, DM (Neuroanesthesia) Senior Resident, Division of Neuro-anaesthesiology, Department of Anaesthesiology, SCTIMST
Co-Principal Investigator(s)
Dr. Manikandan S Degree: MBBS, MD (Anaesthesiology and Critical Care), PDCC (Neuro-anesthesiology) Professor, In-charge of Division of Neuro-anaesthesiology, Department of Anaesthesiology, SCTIMST
Dr. Ranganatha Praveen C S, Degree: MBBS, MD (Anaesthesiology and Critical Care), DM (Neuroanesthesia) Assistant professor, Division of Neuro-Anaesthesiology, Department of Anaesthesiology, SCTIMST
Dr. Easwer H.V Degree: MBBS, MS (General surgery), MCh (Neurosurgery) Professor, Department of Neurosurgery, SCTIMST

Members who participated in the TAC meeting on 01/06/2019

Dr. Rupa Sreedhar (Chairperson)
Dr. Sankara Sarma P
Dr. Prasantakumar Dash
Dr. Sylaja. P.N
Dr. Ashalatha
Dr. Krishna Kumar K
Dr. Sanjay G
Dr. Bijulal S
Dr. Syam K
Dr. Jayadevan ER
Dr. K. Shivakumar (Member Secretary)

Dr. Jayadevan ER, Dr. Sylaja. P.N, Dr. Bijulal S, Dr. Ashalatha, Dr. Rupa Sreedhar, Dr. Prasantakumar Dash and Dr. Sanjay G stayed away from the proceedings when the projects in which they are involved as investigator were discussed (#921, 925, 929, 934, 937, 938, 942, 943, 945, 948).

Risk Classification of the project (Minimum/ Moderate/ High): Minimum
Requirement of DSMB: No
Recommended members of DSMB: Not applicable

Recommendations of TAC:
Recommended for consideration of IEC in the light of the responses received from the investigator

The PI may note that there can be no additions / alterations in the documents approved by TAC when they are submitted to the IEC.

Signature of the Member Secretary, TAC (Clinical Studies)

Note for IEC
Copy of the investigator's responses to questions/suggestions from TAC is attached (Appendix-1).

Received
17/6/19

IEC FORM

श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)
Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

Institutional Ethics Committee

(IEC Regn No. ECR/189/Inst/KL/2013/RR-16)

SCT/IEC/1401/JULY-2019 12.08.2019

Dr. Ashutosh Kumar
Senior Resident, Department of Ananesthesiology
SCTIMST, Thiruvananthapuram

Dear Dr. Ashutosh Kumar,

The Institutional Ethics Committee reviewed and discussed your application to conduct the study entitled "AN EVALUATION OF CHANGES IN CEREBRAL OXYGENATION USING NEAR INFRARED SPECTROSCOPY DURING VARIOUS POSITIONS (SUPINE, PRONE & LATERAL) IN PATIENTS UNDERGOING ELECTIVE NEUROSURGERY – AN OBSERVATIONAL STUDY (IEC/1401)" on 26th July, 2019.

The following documents were reviewed:

Original submission

1. *Covering Letter addressed to the Chairperson, IEC, SCTIMST dated 27.06.2019 with checklist*
2. *TAC Approval Letter*
3. *IEC Application Form*
4. *Project Proposal*
5. *Proforma*
6. *Patient Information Sheet and Informed Consent Form in English and Malayalam*
7. *Forwarding Letter from the HOD*
8. *CV of Principal Investigator and Co-Principal Investigators*

Revised submission

1. *Covering Letter addressed to the Chairperson, IEC, SCTIMST dated 09.08.2019 with checklist*
2. *TAC Approval Letter*
3. *IEC Application Form*
4. *Project Proposal*
5. *Proforma*
6. *Patient Information Sheet and Informed Consent Form in English and Malayalam*
7. *Forwarding Letter from the Guide*
8. *CV of Principal Investigator and Co-Principal Investigators*

The following members of the Ethics Committee were present at the meeting held on 26th July, 2019 at Noshir H Wadia Conference Hall, AMCHSS, SCTIMST

SL. No.	Member Name	Highest Degree	Gender	Scientific /Non Scientific	Affiliation with Institution(s)
1.	Dr. Harikrishnan S	MD, DM (Cardiology) DNB (Cardiology)	Male	Clinician	Yes
2.	Dr. Kala Kesavan. P	MBBS, MD	Female	Basic Medical Scientist	No
3.	Smt. Sathi Nair	MA (English Literature)	Female	Lay Person	No
4.	Dr. Christina George	MD Psychiatry	Female	Clinician	No
5.	Dr. Mala Ramanathan	PhD	Female	Social Scientist (Member Secretary)	Yes

IEC Decision

The IEC approved the conduct of the study in the present form.

Remarks:

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

There was no member of the study team who participated in voting / decision making process. The ethics committee is organized and operated according to the requirements of Good Clinical Practice and the requirements of the Indian Council of Medical Research (ICMR).

Sincerely,

Mala Ramanathan
Member Secretary, IEC

PATIENT INFORMATION SHEET ENGLISH

Title of the study:

An Evaluation Of Postural Variation on Cerebral Oxygenation and Cerebral Blood Flow Velocity Due To Various Neurosurgical Positions (Supine, Prone & Lateral) in Elective Neurosurgery – An Observational Study

Name of the Investigators:
1.Principal Investigator: Dr. Ashutosh Kumar, MD, PDCC, DM (Neuroanesthesia), Senior Resident, Division of Neuro-anaesthesiology, Department of Anaesthesiology, SCTIMST
2.Co-principal Investigator and Guide: Dr. Manikandan S, MD, PDCC, Professor, Incharge of Division of Neuro-anaesthesiology, Department of Anaesthesiology, SCTIMST
3.Co-principal Investigator and Co-Guide: Dr. Ranganatha Praveen C S, DM (Neuroanesthesia) Assistant professor, Division of Neuro-Anaesthesiology, Department of Anaesthesiology, SCTIMST.
4.Co-principal Investigator : Dr.Easwer H.V. MCh (Neurosurgery), Professor, Department of Neurosurgery, SCTIMST

You are being requested to participate in the above titled study which is undertaken to know the changes in cerebral oxygenation using NIRS (Near Infrared Spectroscopy) during various positions (supine, prone & lateral) in patients undergoing elective neurosurgery in our institute. We have planned to include about 60 patients (20 in each group) undergoing neurosurgical procedure from this hospital to the study.

1. What is NIRS?

Near-infrared spectroscopy (NIRS) a non-invasive technology has emerged as a monitor of brain oxygen tension. It is recorded by placing electrodes on both sides of forehead. In the presence of a stable metabolic rate, regional cerebral oxygen saturation, as monitored with NIRS, is therefore an indirect measure of blood flow to brain and provides an ‘‘index’’ of organ ischemia.

2. What is TCD?

Transcranial doppler (TCD) is a non-invasive technology which is emerging as a monitor of brain blood flow. It is recorded by placing an ultrasound probe just above the ears on either side. TCD gives information regarding the blood flow velocity which can be used as a surrogate to measure the cerebral blood flow.

2. If you take part what will you have to do?
On the day of surgery, you will be taken inside the Operation Theatre. Monitors to check your heart beat, blood pressure and oxygen saturation level will be attached. A small venous cannula needle will be inserted under local anesthesia in the hand for fluid and drug administration. Protocol of induction of anesthesia will be according to standard protocol followed in the hospital.
Before induction NIRS & TCD baseline reading will be taken in supine position. Then general anesthesia will be administered for surgery. Ten minutes after securing the airway again the NIRS and TCD values will be recorded. You will be placed in the final surgical position as decided by the neurosurgical team. After your final position is achieved, NIRS and TCD values will be recorded again. The study will be completed after 10 minutes, however monitoring with NIRS will be continued throughout the operation as routinely done in neurosurgical patients in our institute.

3. Does NIRS/TCD have any side effects?
As a non-invasive procedure, it doesn’t carry any risk to patient.

4. Can you withdraw from this study after it starts?
Your participation in this study is entirely voluntary and you are also free to decide to withdraw permission to participate in this study. If you do so, this will not affect your usual treatment at this hospital in any way. In addition, if you experience any side effects, the study will be stopped and you will be given treatment for the side effects.

5. What will happen if you develop any study related injury?
We do not expect any injury to happen to you since the anaesthesia technique and monitoring tools would be same even if you were not part of the study. But if you do develop any side effects or problems due to the study, the side effects will be treated at no cost to you. We are unable to provide any monetary compensation, however.

6. Will you have to pay for the cost of using the devices?
NIRS/TCD monitoring is routinely used as a part of anesthesia monitoring in major neurosurgical patients in our institute. No additional charges will be incurred in participating in the study.

7. Will your personal details be kept confidential?
The results of this study will be used for thesis submission as a part of academic research and will be submitted to a medical journal for publication, but you will not be identified by name in any publication or presentation of results. However, your medical notes may be reviewed by people associated with the study, without your additional permission, should you decide to participate in this study.

If you have any further questions, please ask Dr. Ashutosh Kumar (Principal investigator) mobile number: 9188769371. Email: ashukumar.158@gmail.com.
For technical advisory committee contact, please ask Dr. Mala Ramanathan, telephone number: 0471-2524234. Email: iec.mem.sec@sctimst.ac.in

PATIENT INFORMATION SHEET MALAYALAM

രോഗിക്കുള്ള കാര്യവിവരണപത്രം

പഠനശീർഷകം

മുൻകൂട്ടിനിശ്ചയിച്ചപ്രകാരമുള്ള ന്യൂറോ ശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന രോഗികളിൽ നീയർ ഇൻഫ്രാറെഡ് സ്പെക്ട്രോസ്കോപ്പി ഉപയോഗിച്ച് വ്യത്യസ്ഥ നിലകളിൽ (മലർന്ന്, കമിഴ്ന്ന് ചരിഞ്ഞ്) ഉള്ള മസ്തിഷ്ക്കത്തിലെ പ്രാണവായുലഭ്യതയുടെ മാറ്റങ്ങൾ വിലയിരുത്തൽ - ഒരു നിരീക്ഷണപഠനം.

ഗവേഷകരുടെ പേര്

1. **പ്രധാന ഗവേഷകൻ. ഡോ. അശുതോഷ്കുമാർ, എംഡി, പിഡിസിസി, ഡിഎം (ന്യൂറോ അസ്തീഷ്യ), സീനിയർ റെസിഡന്റ്, ഡിവിഷൻ ഓഫ് ന്യൂറോ അനസ്തീഷ്യോളജി, ഡിപ്പാർട്ട്മെന്റ് ഓഫ് അനസ്തീഷ്യോളജി,** SCTIMST
2. **സഹ പ്രധാന ഗവേഷകനും ഗൈഡും. ഡോ. മണികണ്ഠൻ എസ്, എംഡി, പിഡിസിസി, പ്രൊഫസർ, ഇൻചാർജ്- ഡിവിഷൻ ഓഫ് ന്യൂറോ അനസ്തീഷ്യോളജി, ഡിപ്പാർട്ട്മെന്റ് ഓഫ് അനസ്തീഷ്യോളജി,** SCTIMST
3. **സഹ പ്രധാന ഗവേഷകനും സഹഗൈഡും. ഡോ. രംഗനാഥ പ്രവീൺ സി എസ്, ഡിഎം (ന്യൂറോ അനസ്തീഷ്യ), അസിസ്റ്റന്റ് പ്രൊഫസർ, ഡിവിഷൻ ഓഫ് ന്യൂറോ അനസ്തീഷ്യോളജി, ഡിപ്പാർട്ട്മെന്റ് ഓഫ് അനസ്തീഷ്യോളജി,** SCTIMST
4. **സഹ പ്രധാന ഗവേഷകൻ. ഡോ. ഈശ്വർ എച്ച് വി. എംസിഎച്ച് (ന്യൂറോ സർജറി) പ്രൊഫസർ , ഡിപ്പാർട്ട്മെന്റ് ഓഫ് ന്യൂറോസർജറി,** SCTIMST

നമ്മുടെ സ്ഥാപനത്തിൽ മുൻകൂട്ടി നിശ്ചയിച്ച പ്രകാരമുള്ള ന്യൂറോ ശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന രോഗികളിൽ എൻഐആർഎസ് (നീയർ ഇൻഫ്രാറെഡ് സ്പെക്ട്രോസ്കോപ്പി) ഉപയോഗിച്ച് വ്യത്യസ്ഥ നിലകളിൽ (മലർന്ന്, കമിഴ്ന്ന് ചരിഞ്ഞ്) ഉള്ള മസ്തിഷ്ക്കത്തിലെ പ്രാണവായുവിന്റെ മാറ്റങ്ങളുടെ വിലയിരുത്തൽ നടത്തുന്ന പഠനത്തിൽ പങ്കെടുക്കാൻ താങ്കോട് അഭ്യർത്ഥിക്കുന്നു. ന്യൂറോ ശസ്ത്രക്രിയയ്ക്ക് ഈ ആശുപത്രിയിൽ വിധേയരാകുന്ന ഏകദേശം 60 രോഗികളെ (ഒരു സംഘത്തിൽ 20 പേർ വീതം) ഈ പഠനത്തിൽ പങ്കെടുപ്പിക്കുവാൻ ഞങ്ങൾ ആസൂത്രണം ചെയ്യുന്നു.

എൻഐആർഎസ് എന്നാലെന്ത്

നീയർ ഇൻഫ്രാറെഡ് സ്പെക്ട്രോസ്കോപ്പി (എൻഐആർഎസ്) തലച്ചോറിലെ പ്രാണവായുവിന്റെ സാന്ദ്രത നിരീക്ഷിക്കുന്ന ശരീരത്തിൽ പ്രവേശിക്കാതെയുള്ള സങ്കേതമായി ആവിർഭവിച്ചിരിക്കുന്നു. നെറ്റിയുടെ രണ്ടുഭാഗത്തുമായി ഘടിപ്പിക്കുന്ന ഇലക്ട്രോഡുകൾ വഴി അത് രേഖപ്പെടുത്തുന്നു. സ്ഥിരമായ ചയാപചയ തോതിന്റെ സാന്നിദ്ധ്യത്തിൽ, മസ്തിഷ്ക്കത്തിലെ പ്രാണവായുവിന്റെ സാന്ദ്രത എൻഐആർഎസ് വഴി നിരീക്ഷിക്കുന്നതിലൂടെ തലച്ചോറിലേയ്ക്കുള്ള രക്തപ്രവാഹം പരോക്ഷമായി അളക്കുന്നതിലൂടെ അവയവത്തിലെ രക്തപ്രവാഹത്തിന്റെ കുറവിന്റെ സൂചകമാവുകയും ചെയ്യും.

താങ്കൾ പങ്കെടുക്കുകയാണെങ്കിൽ എന്തു ചെയ്യണം

ശസ്ത്രക്രിയാദിവസം താങ്കളെ ശസ്ത്രക്രിയാമുറിയിലേയ്ക്ക് കൊണ്ടുപോകും. താങ്കളുടെ പൃദയമിടിപ്പ്, രക്തസമ്മർദ്ദം പ്രാണവായുവിന്റെ സാന്ദ്രത എന്നിവ ശരീരത്തിൽ പ്രവേശിക്കാതെ പരിശോധിക്കുന്ന ഉപകരണങ്ങൾ ഘടിപ്പിക്കും. ദ്രാവകങ്ങളും മരുന്നും നൽകാൻ പ്രാദേശികമായ മരവിപ്പിക്കലിന് വിധേയമാക്കി ഒരു ചെറിയകുഴൽ കൈയിൽ കടത്തും. അടിസ്ഥാന വിഇപി പെരുമാറ്റച്ചട്ടപ്രകാരം രേഖപ്പെടുത്തും. ആശുപത്രിയിലെ അംഗീകൃത പെരുമാറ്റച്ചട്ടപ്രകാരമുള്ള മയക്കൽ നൽകും,

മയക്കുന്നതിനുമുൻപ് താങ്കളെ മലർത്തികിടത്തി എൻഐആർഎസ് ഇലക്ട്രോഡുകൾ നെറ്റിയിൽ വച്ച് അടിസ്ഥാന അളവുകൾ രേഖപ്പെടുത്തും. അതിനുശേഷം താങ്കൾ പൂർണ്ണമായും മയങ്ങുകയും നിശ്ചലമാവുകയും താങ്കളെ കൃത്രിമ ശ്വസനസഹായിയുമായി ബന്ധിപ്പിക്കുകയും ചെയ്യും. ന്യൂറോ ശസ്ത്രക്രിയാ സംഘത്തിന്റെ തീരുമാനപ്രകാരമുള്ള നിലയിൽ താങ്കളെ കിടത്തും. താങ്കളുടെ കൃത്യമായ നില കൈവരിച്ചശേഷം എൻഐആർഎസ് അളവുകൾ 1 മിനിട്ട് ഇടവേളയിൽ ആദ്യത്തെ 10 മിനിട്ട് രേഖപ്പെടുത്തും അതിനുശേഷം 5 മിനിട്ട് ഇടവിട്ട് ശസ്ത്രക്രിയാനില മാറ്റി കഴിഞ്ഞ് 30 മിനിട്ടുവരെയും രേഖപ്പെടുത്തും. പഠനം 30 മിനിട്ടിനകം പൂർത്തികരിക്കും, എന്നാലും എൻഐആർഎസ് ഉപയോഗിച്ചുള്ള നിരീക്ഷണം നമ്മുടെ ഇൻസ്റ്റിറ്റ്യൂട്ടിൽ പതിവായി ചെയ്യുന്നതുപോലെ ശസ്ത്രക്രിയാ സമയം മുഴുവൻ തുടരും.

എൻഐആർഎസിന് പാർശ്വഫലങ്ങളുണ്ടോ?

ശരീരത്തിൽ പ്രവേശിക്കാതുള്ള ഒരു നടപടി, രോഗിക്ക് ഒരു അപായവുമുണ്ടാകില്ല.

താങ്കൾക്ക് പഠനമാരംഭിച്ചശേഷം പിന്മാറാമോ?
പഠനത്തിലുള്ള താങ്കളുടെ പങ്കാളിത്തം പൂർണ്ണമായും സ്വമേധയായുള്ളതും പഠനത്തിനുള്ള സമ്മതം പിൻവലിക്കാൻ താങ്കൾക്ക് സ്വാതന്ത്ര്യമുള്ളതുമാണ്. താങ്കളങ്ങിനെ ചെയ്യുന്നതുകൊണ്ട് താങ്കളുടെ ഈ ആശുപത്രിയിലെ ചികിത്സയെ ഒരുവിധത്തിലും ബാധിക്കില്ല. കൂടാതെ താങ്കൾക്കെന്തെങ്കിലും പാർശ്വഫലങ്ങളനുഭവിക്കേണ്ടിവന്നാൽ പഠനം നിർത്തുകയും പാർശ്വഫലങ്ങൾക്ക് ചികിത്സ നൽകുകയും ചെയ്യും.

പഠനവുമായി ബന്ധപ്പെട്ട് താങ്കൾക്ക് പരുക്കുണ്ടായാലെന്തുചെയ്യും?

താങ്കൾ പങ്കെടുത്താലുമില്ലെങ്കിലും മയക്കൽ സങ്കേതവും നിരീക്ഷണ ഉപകരണങ്ങളും ഒന്നുതന്നെയാകയാൽ താങ്കൾക്ക് പരുക്കൊന്നുമുണ്ടാകുമെന്ന് ഞങ്ങൾ പ്രതീക്ഷിക്കുന്നില്ല.പക്ഷേ പഠനവുമായി ബന്ധപ്പെട്ട് എന്തെങ്കിലും പാർശ്വഫലങ്ങളോ പ്രശ്നങ്ങളോ ഉണ്ടായാൽ അവ താങ്കൾക്ക് അധികച്ചിലവില്ലാതെ ചികിത്സിക്കും. എന്തായാലും, സാമ്പത്തികമായ നഷ്ടപരിഹാരം നൽകാൻ ഞങ്ങൾക്ക് ആവില്ല.

ഈ ഉപകരണം ഉപയോഗിക്കുന്നതിന് താങ്കൾ പണം മുടക്കണോ?

നമ്മുടെ സ്ഥാപനത്തിൽ എൻഐആർഎസ് നിരീക്ഷണം പ്രധാന ന്യൂറോശസ്ത്രക്രിയയ്ക്ക് വേണ്ടിയുള്ള മയക്കലിന്റെ ഭാഗമാണ്. ആയതിനാൽ പഠനത്തിൽ പങ്കെടുക്കുന്നതുകൊണ്ട് അധികമായി പണം നൽകേണ്ടതില്ല.

താങ്കളുടെ വ്യക്തി വിവരങ്ങൾ രഹസ്യമായി സൂക്ഷിക്കുമോ?

പഠനഫലങ്ങൾ പഠനഗവേഷണത്തിന്റെ ഭാഗമായ പ്രബന്ധത്തിലും വൈദ്യശാസ്ത്ര ജേർണലിലും പ്രസിദ്ധീകരിച്ചേക്കാമെങ്കിലും താങ്കളെ പേരുകൊണ്ടോ മറ്റുവിധത്തിലോ തിരിച്ചറിയാനിടയാക്കുന്നതൊന്നും പ്രസിദ്ധീകരണത്തിലോ പ്രദർശനങ്ങളിലോ ഒരിടത്തും ഉണ്ടാകില്ല. താങ്കൾ പഠനത്തിൽ പങ്കെടുക്കാൻ സമ്മതിച്ചാൽ, താങ്കളുടെ ചികിത്സാവിവരങ്ങൾ താങ്കളുടെ പ്രത്യേക അനുമതി കൂടാതെതന്നെ പഠനവുമായി ബന്ധപ്പെട്ടവർ പരിശോധിച്ചേക്കാം

താങ്കൾക്ക് കൂടുതൽ ചോദ്യങ്ങളുണ്ടെങ്കിൽ, ദയവായി ചോദിക്കുക ഡോ. അശുതോഷ് കുമാർ (പ്രധാന ഗവേഷകൻ) ഫോൺ. 9188769371. ഇമെയിൽ ashukumar.158@gmail.com.

ഇൻസ്റ്റിറ്റ്യൂഷണൽ എത്തിക്സ് കമ്മിറ്റി യുമായി ബന്ധപ്പെടാൻ മെമ്പർ സെക്രട്ടറി, (ഫോൺ 0471-2524234) ഓഫീസ് എക്സ്റ്റൻഷൻ നമ്പർ 234 ഇമെയിൽ mala@sctimst.ac.in

CONSENT FORM ENGLISH

Participant's name: Date of Birth / Age (in years):

I_____, son/daughter of _____ Declare that (Please tick boxes)

• I have read the above information provided to me regarding the study: An Evaluation Of Changes In Cerebral Oxygenation Using Near Infrared Spectroscopy and Cerebral Blood Flow Velocity Using Transcranial Doppler During Various Positions (Supine, Prone & Lateral) In Patients Undergoing Elective Neurosurgery – An Observational Study**.** [ ]

- I have clarified any doubts that I had. [ ]
- I also understand that my participation in this study is entirely voluntary and that I am free to withdraw permission to continue to participate at any time without affecting my usual treatment or my legal rights [ ]

• I understand that the study staff and institutional ethics committee members will not need my permission to look at my health records even if I withdraw from the trial. I agree to this access []

• I understand that my identity will not be revealed in any information released to third parties or published [ ]

- I voluntarily agree to take part in this study [ ]
- I have been provided with the contact numbers of the principle investigator, in case I want to know more about the study and participants rights [ ].

• I received a copy of this signed consent form [ ]

Name: Signature: Date:

Name of witness: Relation to participant:

Signature:

Person Obtaining Consent

I attest that the requirements for informed consent for the medical research project described in this form have been satisfied. I have discussed the research project with the participant and explained to him or her in nontechnical terms all of the information contained in this informed consent form, including any risks and adverse reactions that may reasonably be expected to occur. I further certify that I encouraged the participant to ask questions and that all questions asked were answered.

Name of witness:

Relation to participant:

Signature:

Person Obtaining Consent

I attest that the requirements for informed consent for the medical research project described in this form have been satisfied. I have discussed the research project with the participant and explained to him or her in nontechnical terms all of the information contained in this informed consent form, including any risks and adverse reactions that may reasonably be expected to occur. I further certify that I encouraged the participant to ask questions and that all questions asked were answered.

Name:

Signature: Date:

CONSENT FORM MALAYALAM

സമ്മതപത്രം

മുൻകൂട്ടിനിശ്ചയിച്ചപ്രകാരമുള്ള ന്യൂറോ ശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന രോഗികളിൽ നീയർ ഇൻഫ്രാറെഡ് സ്പെക്ട്രോസ്കോപ്പി ഉപയോഗിച്ച് വ്യത്യസ്ഥ നിലകളിൽ (മലർന്ന്, കമിഴ്ന്ന് ചരിഞ്ഞ്) ഉള്ള മസ്തിഷ്ക്കത്തിലെ പ്രാണവായുലഭ്യതയുടെ മാറ്റങ്ങൾ വിലയിരുത്തൽ - ഒരു നിരീക്ഷണപഠനം.

പഠനനമ്പർ:

പങ്കാളിയുടെ പേര്:
ജനനതീയതി/വയസ്സ് (വർഷത്തിൽ)

ഞാൻ.....പുത്രൻ/പുത്രി..... (ദയവായി കോളങ്ങളിൽ ശരിയടയാളപ്പെടുത്തുക)

- മുകളിൽ പറഞ്ഞ മുൻകൂട്ടിനിശ്ചയിച്ചപ്രകാരമുള്ള ന്യൂറോ ശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന രോഗികളിൽ നീയർ ഇൻഫ്രാറെഡ് സ്പെക്ട്രോസ്കോപ്പി ഉപയോഗിച്ച് വ്യത്യസ്ഥ നിലകളിൽ (മലർന്ന്, കമിഴ്ന്ന് ചരിഞ്ഞ്) ഉള്ള മസ്തിഷ്ക്കത്തിലെ പ്രാണവായുലഭ്യതയുടെ മാറ്റങ്ങൾ വിലയിരുത്തൽ - ഒരു നിരീക്ഷണപഠനം. എന്ന പഠന സംബന്ധിയായി എനിക്കു നൽകിയ വിവരങ്ങൾ വായിച്ചു എന്നു പ്രസ്താവിക്കുന്നു.[ ]

- എന്റെ എല്ലാ സംശയങ്ങളും പരിഹരിച്ചു.[ ]

- എന്റെ ഈ പഠനത്തിലുള്ള പങ്കാളിത്തം പൂർണ്ണമായും സ്വമേധയാ ആണെന്നും അനുവാദം എനിക്ക് ഏതുസമയത്തും എന്റെ ചികിത്സയെയോ നിയമപരമായ അവകാശങ്ങളെയോ ബാധിക്കാതെ പിൻവലിക്കാൻ അവകാശമുണ്ടെന്നും ഞാൻ മനസ്സിലാക്കുന്നു. [ ]

- ഞാൻ ഈ പഠനത്തിൽ നിന്നും പിൻമാറിയാലും പഠനം നടത്തുന്നവർക്കും സ്ഥാപനത്തിലെ നൈതിക കമ്മിറ്റി അംഗങ്ങൾക്കും എന്റെ ആരോഗ്യരേഖകൾ പരിശോധിക്കുന്നതിന് എന്റെ അനുവാദം ആവശ്യമില്ലെന്ന് ഞാൻ മനസ്സിലാക്കുന്നു. അതിനോട് ഞാൻ യോജിക്കുന്നു. [ ]

- എന്നെ തിരിച്ചറിയാനുതകുന്ന വിവരങ്ങൾ ഒന്നും മൂന്നാം കക്ഷികൾക്കു നൽകുകയോ പ്രസിദ്ധീകരിക്കുകയോ ചെയ്യില്ലെന്ന് ഞാൻ മനസ്സിലാക്കുന്നു. [ ]

- ഞാൻ സ്വമേധയാ പഠനത്തിൽ പങ്കെടുക്കാൻ സമ്മതിക്കുന്നു. [ ]

- സമ്മതപത്രത്തിന്റെ ഒപ്പിട്ട ഒരു കോപ്പി എനിക്കു കിട്ടി [ ]

പങ്കെടുക്കുന്നയാളുടെ പേര്

ഒപ്പ്

തീയതി

സാക്ഷിയുടെ പേര്

ഒപ്പ്

പങ്കെടുക്കുന്ന ആളുമായുള്ള ബന്ധം

തീയതി

(സമ്മതം വാങ്ങുന്നയാൾ)

മെഡിക്കൽ റിസർച്ച് പ്രോജക്ടിനാവശ്യമായ സമ്മതപത്രത്തിനു വേണ്ടുന്ന എല്ലാ ഘടകങ്ങളും തൃപ്തികരമായി നിർവഹിച്ചിരിക്കുന്നുവെന്ന് ഞാൻ ബോധ്യപ്പെടുത്തുന്നു. പഠനപങ്കാളിയുമായി ഗവേഷണപദ്ധതിയെപ്പറ്റി സാങ്കേതികേതര പദങ്ങളുപയോഗിച്ച് എല്ലാ വിവരങ്ങളെപ്പറ്റിയും ചർച്ച നടത്തുകയും പ്രതീക്ഷിക്കാവുന്ന അപകടസാധ്യതകളും പാർശ്വഫലങ്ങളും വിശദീകരിക്കുകയും ചെയ്തു. പങ്കാളിയെ ചോദ്യങ്ങൾ ചോദിക്കാൻ പ്രേരിപ്പിക്കുകയും എല്ലാ ചോദ്യങ്ങൾക്കും ഉത്തരം നൽകുകയും ചെയ്തു എന്നും ഞാൻ സാക്ഷ്യപ്പെടുത്തുന്നു.

സമ്മതപത്രം വാങ്ങുന്ന ആളുടെ പേര്

ഒപ്പ്

പ്രധാന ഗവേഷകൻ

PROFORMA

Preop data

GROUP	I	II	III
Age (Years)			
Sex			
Weight (Kg)			
Height (cm)			
BSA (Kg/m^2)			
Diagnosis			
Surgery			
ASA Grading			

Hemodynamic data:

	HR			SBP			DBP			MAP		
	T0	T1	T2	T0	T1	T2	T0	T1	T2	T0	T1	T2
Group S												
Group P												
Group L												

Ventilatory data

	PIP		ETCO2		TV		RR	
	T1	T2	T1	T2	T1	T2	T1	T2
Group S								
Group P								
Group L								

NIRS DATA

	NIRS (L)			NIRS (R)		
	T0	T1	T2	T0	T1	T2
Group S						
Group P						
Group L						

TCD data

	MCA (L) MFV			ACA (L) MFV			MCA (R) MFV			ACA (L) MFV		
	T0	T1	T2	T0	T1	T2	T0	T1	T2	T0	T1	T2
Group S												
Group P												
Group L												

MASTER CHART

GROUP SUPINE

SL.No	GROUP NO.	GROUP	Age(Years)	Sex(M/F)	Weight (Kg)	Height	BMI
1	1	SUPINE	45	M	78	176	25.2
2	1	SUPINE	39	F	55	160	21.5
3	1	SUPINE	30	F	56	162	21.3
4	1	SUPINE	35	F	47	158	18.8
5	1	SUPINE	36	F	55	160	21.5
6	1	SUPINE	47	F	61	154	25.7
7	1	SUPINE	37	M	80	175	26.1
8	1	SUPINE	28	M	80	180	24.7
9	1	SUPINE	50	F	48	147	22.2
10	1	SUPINE	52	F	58	175	18.9
11	1	SUPINE	40	M	80	178	25.2
12	1	SUPINE	55	F	47	158	18.8
13	1	SUPINE	50	M	84	164	31.2
14	1	SUPINE	51	M	79	170	27.3
15	1	SUPINE	48	M	75	164	27.9
16	1	SUPINE	37	F	80	161.5	30.4
17	1	SUPINE	27	M	115	170	42
18	1	SUPINE	28	M	70	162	26.7
19	1	SUPINE	45	F	55	160	21.5
20	1	SUPINE	58	F	82	150	36.4

Diagnosis	Surgery	Co morbidity	ASA Grading
LEFT SPHENOID WING MENINGIOMA	LEFT PTERIONAL Craniotomy & excision		I
Right frontal lobe glioma	Right PTERIONAL Craniotomy & excision	DM	2
Left temporal glioma	LEFT PTERIONAL Craniotomy & excision		I
RIGHT FCD	Right frontal Craniotomy & Excision		I
right parietal cavernoma	Right parieto occipital craniotomy & excision		I
Left temporo insular mets	Left temporal craniotomy & excision	Hypothyroidism	2
Left callosal Glioma	Left FTP craniotomy & decompression		1
Right basifrontal cavernoma	Right pterional craniotomy & excision		1
Left gangliocapsular GBM	LEFT PTERIONAL Craniotomy & excision		1
Right frontal gbm	RighT PTERIONAL Craniotomy & excision		1
Right parafalcine Meningioma	Craniotomy & excision		1
Right sphenoid wing Meningioma	Right pterional craniotomy & excision		I
Right trigeminal Neuralgia	Right mcirovascular decompression		1
Right frontal convexity meningioma	Right pterional craniotomy & excision		1
Left cpa lesion	MCF approach Craniotomy & decompression		1
Tubercullum sella meningioma	Right pterional craniotomy & excision		1
hypothalamic Glioma	ht pterional craniotomy & subtotal decompress	Bronchial Asthama	2
Colloid cyst	Right pterional craniotomy & excision		1
Right frontal cavernous malformation	Right FP craniotomy & decompression		1
Right Sphenoid wing meningioma	Right FT Craniotomy & Excision	Htn	2

Heart Rate			SBP			DBP			MAP		
T0	T1	T2	T0	T1	T2	T0	T1	T2	T0	T1	T2
74	82	85	124	118	120	70	66	68	88	83	85
65	74	78	114	108	106	72	69	73	86	82	84
71	75	80	106	101	110	66	60	62	79	74	78
77	82	79	130	120	128	80	72	75	97	88	93
73	80	82	136	122	130	90	81	81	105	95	97
70	74	75	130	122	120	86	80	79	100	94	93
66	69	72	114	105	108	76	72	73	89	83	85
72	77	76	115	109	111	68	62	65	84	78	80
85	90	92	136	125	130	77	71	75	97	89	93
58	62	61	122	118	128	67	62	72	85	81	91
57	60	61	131	118	128	80	74	77	97	89	94
80	84	79	131	124	128	80	74	77	97	91	94
79	83	80	138	130	132	80	74	77	99	93	95
65	70	71	135	128	130	88	81	83	104	97	99
66	62	69	136	129	132	88	84	85	104	99	101
74	79	75	114	106	109	70	63	62	85	77	78
80	84	79	112	103	110	82	78	63	92	87	79
59	66	64	126	113	109	85	80	62	99	91	78
90	97	82	150	140	137	90	82	80	110	101	99
65	70	72	127	119	124	87	82	84	100	94	97

PEAK Airway Pressure		ETCO2		Tidal Volume		Respiratory Rate	
T1	T2	T1	T2	T1	T2	T1	T2
18	19	35	35	480	480	12	12
20	21	33	33	400	400	12	12
21	21	34	34	410	410	12	12
19	20	33	33	410	410	12	12
19	19	34	34	400	400	12	12
22	24	35	35	450	450	12	12
21	24	35	35	500	500	12	12
18	19	35	35	500	500	12	12
17	19	33	33	380	380	12	12
20	22	34	34	420	420	12	12
20	23	35	35	500	500	12	12
19	21	33	33	370	370	12	12
22	25	35	35	525	525	12	12
20	22	35	35	490	490	12	12
18	19	35	35	480	480	12	12
17	19	35	35	500	500	12	12
23	25	35	35	600	600	16	16
18	21	35	35	480	480	12	12
17	18	34	34	400	400	12	12
20	23	35	35	500	500	12	12

NIRS (L)			NIRS (R)		
T0	T1	T2	T0	T1	T2
63	65	62	66	69	63
69	70	63	70	72	68
67	65	68	73	69	68
69	70	70	70	71	70
54	60	65	64	70	68
82	77	78	77	74	72
75	75	79	73	70	78
82	75	76	72	70	78
60	62	61	62	65	63
68	65	71	70	62	75
69	66	67	72	68	62
56	59	69	62	60	70
75	79	77	80	82	79
65	67	65	69	69	77
61	65	63	64	68	72
64	71	69	66	75	68
62	65	59	63	67	62
71	72	66	68	71	70
69	71	73	76	77	75
71	65	66	81	72	76

MCA (L)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
66.4	41.8	44.3	48.5	28.5	29.6	0.64	0.87	1.15
94.4	82.5	75.4	58.3	54.1	57.2	1.01	0.88	0.62
57	56.3	34.6	35.4	37	22.9	1.25	1.09	0.86
70.2	47.7	48.6	50.6	37.1	29.6	0.72	0.6	1.03
79.4	85	70	53.4	58.1	43	0.74	0.75	1.07
80.8	66.6	53	56.5	45.2	31.7	0.85	0.86	1.13
91.4	44	39.2	64.1	23.8	21.8	0.74	1.18	1.2
42.3	58.1	66.4	23.6	29.9	32.5	1.33	1.31	1.41
129	96	41.2	81	54.2	22.9	0.94	1.18	1.34
70.1	66.7	68.7	45.5	46.6	49.4	0.92	0.8	0.95
73.7	73.6	81.6	43.2	40.2	43.1	1.21	1.22	1.31
94.6	63.6	50.5	63.4	42.6	26.2	0.81	0.84	1.53
42.4	30.7	41.2	23.1	17.4	22.9	1.25	1.17	1.34
46	41	63.7	31.1	32	36.9	0.84	0.55	1.1
47.9	56.5	47.2	31.1	39.6	31.2	0.86	0.76	0.75
83.4	44.1	46.7	59.7	27.2	30.2	0.67	0.99	0.91
58.4	42.1	30.5	33.2	22.7	14.1	1.02	1.22	1.77
41.9	39	59.7	25.8	18.9	31.2	1	1.41	1.26
78.9	55.3	44.2	54.5	34.1	26.8	0.81	1.07	1.06
36.4	44.7	22.5	25	27.2	13.3	0.84	1.15	1.17

MCA (R)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
69.7	54.6	27.5	48.7	37.8	19.32	0.74	0.76	0.75
84.1	71.1	53	55	55	67.7	0.94	0.64	0.84
88	54	50.6	69.4	43.3	33.4	0.51	0.64	0.82
73.7	44.9	46.1	41.4	20.7	28	1.4	1.62	1.06
61.1	41.3	63.5	33.9	31.1	41	1.16	0.71	0.9
56.2	42.6	49.6	42.3	25.7	32.1	0.62	1.16	0.92
69.9	66.1	44	49.4	41.5	26.2	0.75	0.91	0.99
38	77.5	44.2	22.7	48.1	24.5	1.25	0.91	1.17
63.8	70	64.6	37	38.8	34.7	1.17	1.26	1.34
78.8	115	66.8	51.8	81.3	48.8	0.99	0.74	0.78
84.1	47.2	41.8	54.9	26	23.6	0.9	1.24	1.22
59.1	57.5	47.4	42.1	40.3	33.1	0.69	0.77	0.74
53.6	54.6	46.6	29.7	37.8	26.2	1.22	0.76	1.22
57.3	56.2	38.9	39.1	36.1	24.23	0.78	0.84	0.91
45.3	47.1	33	30.1	31.4	23.2	0.84	0.83	0.76
48.3	40.2	40.2	34.6	24.2	25.8	0.68	1.03	0.91
57.9	37.1	58.3	35.2	19.5	24.7	0.92	1.28	1.91
49.8	64.9	30.6	38.2	34.8	17.2	0.58	1.22	1.1
78.5	74	41.7	53.6	47.4	25.1	0.79	0.96	1.09
65	42.4	35.4	42.8	23.2	25	0.9	1.21	0.77

ACA (L)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
62.4	38.2	33.8	46.2	89.7	18.4	0.62	0.22	1.2
38.4	50.3	51.4	27	29.3	42.9	0.82	1.06	0.74
44.5	31.9	28.2	54.8	24.8	24.9	0.72	0.61	0.26
54	39.1	32.3	39.4	21.5	21.5	0.68	1.23	0.91
50.2	62.9	59.7	35.1	44.7	44.5	0.62	0.71	0.64
48.8	41.2	25	33.2	29.1	15.8	0.89	0.86	1.04
74.8	35.3	46	41.7	20.8	28.9	1.17	1.03	0.98
53.1	29.5	40.2	38.7	19.9	35.5	0.64	0.75	0.25
57.5	46.6	39.4	30.7	26.2	21.4	1.36	1.22	1.31
62.8	42.9	62.3	48.1	25.5	34.4	0.65	1.1	0.98
34.5	65.1	50.2	19.8	38.4	35.1	1.13	1.11	0.62
74.8	80.8	45	41.7	56.5	28.2	1.17	0.85	1.02
48.2	36.4	36.1	33.1	19.7	22.6	0.77	1.24	0.98
57	45.3	41.7	35.3	26.1	29.2	0.96	1.1	0.78
32.9	35.5	49.2	22.9	28.5	35.8	0.74	0.67	0.74
54.1	50.3	47.5	38.5	47	30.4	0.82	0.76	0.88
24.1	19.4	32.1	14.1	10.5	13.6	1.04	1.34	2.04
62.7	39.1	68.8	43.2	18.9	36.3	0.77	1.47	1.24
51.7	52	40.3	31.8	33.2	25.7	1.01	0.99	1.02
49.8	36.8	30.4	29.7	22.5	18.2	1.08	1.07	1.12

ACA(R)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
39	34	34.8	32.4	23.8	20.4	0.33	0.78	1.06
71.9	70.7	51.1	52	51.5	73.8	0.67	0.66	0.43
60.1	52.2	48	42.4	34	31.5	0.73	0.88	0.9
51.5	23.4	40.3	39.8	13	24.5	0.61	1.62	1.01
47.4	43.4	39.9	30.2	27.8	29.4	1.07	0.92	0.65
57.1	32.6	31.8	42.2	21.1	18.8	0.71	0.98	1.11
45.4	29.5	31.2	33.5	16.9	22.2	0.28	1.12	0.73
34.6	78.6	40.3	21.8	42.2	21.5	0.98	1.17	1.2
60.4	48.4	35.7	39.6	28.3	21.4	0.95	1.21	1.09
49.5	37.5	69.1	36.1	27.4	52.7	0.7	0.67	0.82
34.2	49.2	37.5	22.9	27.4	22	0.85	1.19	1.14
84.3	75	65.6	74.7	58.2	46.7	0.24	0.64	0.71
66.3	61.8	33.8	47.5	32.7	17.3	0.73	1.3	1.34
65	47.7	35.1	44.8	34.7	23.2	0.75	0.69	0.82
51.6	48.9	27.3	34.8	33.9	18.2	0.87	0.75	0.755
37.1	39.4	29.2	27.3	25.1	19.1	0.67	0.93	0.87
47.1	39.9	39.7	23.4	23.4	23.1	1.48	1.01	1.16
46.4	56.5	32.4	34.7	35.7	16.4	0.73	0.9	1.34
39.7	38	31.1	24.5	23.2	21.6	0.97	1.04	0.8
40.3	23	26.9	26.6	14.2	22.1	0.93	1.15	0.7

GROUP PRONE

SL.No	GROUP NO.	GROUP	Age(Years)	Sex(M/F)	Weight (Kg)	Height	BMI
21	1	PRONE	25	M	80	176	25.8
22	2	PRONE	48	F	70	162	26.7
23	2	PRONE	46	F	65	160	25.4
24	2	PRONE	41	F	70	162	26.7
25	2	PRONE	59	F	55	160	21.5
26	2	PRONE	60	F	48	147	22.2
27	2	PRONE	25	M	80	170	27.7
28	2	PRONE	32	M	72	165	26.5
29	2	PRONE	22	M	55	175	18
30	2	PRONE	47	F	80	175	26.1
31	2	PRONE	47	F	55	160	21.5
32	2	PRONE	20	M	58	175	18.9
33	2	PRONE	48	F	60	169	21
34	2	PRONE	30	M	80	170	27.7
35	2	PRONE	40	F	76	157	30.8
36	2	PRONE	60	F	80	175	26.1
37	2	PRONE	46	F	48	147	22.2
38	2	PRONE	36	F	50	162	19
39	2	PRONE	44	F	58	152	25.1
40	2	PRONE	34	M	55	160	21.5

Diagnosis	Surgery	Co morbidity	ASA Grading
C2 EXTRADURAL SCHWANNOMA	Excision & OC fusion		I
L3-L4 PIVD	DECOMPRESSIVE LAMINECTOMY		I
PONTINE CAVERNOMA	MLSO craniotomy and excision		I
LEFT CP ANGLE SCHWANNOMA	LEF RSSO craniotomy and excision		I
LEFT PARIETO OCCIPITAL MENINGIOMA	Craniotomy and excision	DM	2
LEFT PETRO TENTORIAL MENINGIOMA	LEFT RSSO craniotomy and excision	Hypothyroidism	2
C1-C2 lesion	Excision & o-C fusion		1
Left petroclival plasmacytoma	Craniotomy & excision		1
pineal region tumor	Modified poppens approach		1
Chiari with syrinynx	Foramen magnum decompression		1
S2 Schwannoma	Laminectomy & excision		1
C2 vertebral body lesion	Excision of lesion		1
Left Cerebellar Convexity meningioma	Left rsso craniotomy and excision		1
L5-S1 PIVD	Laminectomy + Discectomy		1
chiari1	Foramen magnum decompression	HTN	2
L3-L5 IVDP	Decompressive laminectomy, Discectomy & fusion		1
Right petroclival meningioma	Craniotomy & excision	Bronchial Asthama	2
chiari1	Foramen magnum decompression		1
Cerebellar Hemangioblastoma	MLSO Craniotomy & excision		1
Cerebellar Hemangioblastoma	MLSO Craniotomy & excision		1

Heart Rate			SBP			DBP			MAP		
T0	T1	T2	T0	T1	T2	T0	T1	T2	T0	T1	T2
75	83	80	128	118	115	72	66	62	91	83	80
65	74	78	114	108	106	72	69	73	86	82	84
74	80	82	110	101	110	70	65	62	84	77	78
79	83	80	133	127	120	79	72	69	97	90	86
73	80	82	142	134	132	86	81	72	104	99	92
69	74	76	114	109	107	74	69	72	87	82	84
66	69	72	130	122	119	75	70	71	93	87	87
73	77	80	117	110	112	70	63	65	86	79	81
85	90	92	126	121	124	78	70	71	94	87	89
58	62	61	120	115	119	85	79	81	97	91	94
57	60	61	112	105	110	80	76	77	91	86	88
80	84	79	131	123	121	76	72	74	94	89	90
79	83	80	140	132	134	76	70	71	97	91	92
65	70	71	125	132	134	66	70	71	86	91	92
88	95	84	125	128	118	76	75	68	92	93	85
75	80	76	136	129	125	78	72	70	97	91	88
81	85	79	122	111	119	74	68	70	90	82	87
86	91	93	125	120	123	78	70	71	94	87	88
59	65	62	122	113	119	68	62	70	86	79	86
89	97	84	115	108	119	85	81	70	95	90	86

PEAK Airway Pressure		ETCO2		Tidal Volume		Respiratory Rate	
T1	T2	T1	T2	T1	T2	T1	T2
19	23	35	35	500	500	12	12
19	21	34	34	450	450	12	12
21	21	34	34	420	420	12	12
19	20	35	35	450	450	12	12
19	19	34	34	400	400	12	12
22	24	33	33	380	380	12	12
21	25	35	35	500	500	12	12
18	20	35	35	460	460	12	12
18	19	34	34	400	400	12	12
20	24	35	35	500	500	12	12
19	22	34	34	430	430	12	12
19	23	34	34	400	400	12	12
21	25	35	35	420	420	12	12
18	20	35	35	490	490	12	12
18	22	35	35	470	470	12	12
17	19	35	35	500	500	12	12
22	25	33	33	380	380	12	12
18	19	33	33	400	400	12	12
17	21	35	35	420	420	12	12
20	23	34	34	400	400	12	12

NIRS (L)			NIRS (R)		
T0	T1	T2	T0	T1	T2
76	70	60	72	67	54
69	62	65	67	66	65
58	70	58	56	62	69
54	58	54	62	61	59
65	71	62	64	56	60
79	72	75	73	68	71
71	69	72	76	70	68
70	67	58	72	69	65
74	65	67	67	60	62
62	68	72	64	61	68
65	68	70	63	63	66
80	78	75	78	75	80
69	66	65	73	62	63
66	68	62	63	70	67
62	62	59	67	66	63
64	61	62	69	67	70
68	61	60	68	59	62
73	63	65	66	59	61
66	76	70	76	80	72
65	62	62	62	69	63

MCA (L)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
52.1	61.1	26.3	36.8	30	16.4	0.82	1.69	0.85
56.9	40.7	71.7	49.4	28.4	50.3	0.75	0.79	0.81
94	91.8	63.2	68.4	70.5	42.4	0.76	0.64	0.84
48.1	47.2	22.1	33.3	30.5	15.9	0.8	0.86	0.69
45.4	47.4	41	28	37.1	15.6	1.04	0.6	2.34
56.3	29.9	44.9	27.5	18.8	28.6	1.89	0.92	1.18
52.1	61.1	54	36.8	30	29.3	0.82	1.69	1.24
74.5	53.8	40.7	55.4	43.6	28.4	0.86	0.48	0.79
70.8	49.9	25.7	54.3	28.6	14.4	0.54	1.05	1.13
58.9	44.7	33.3	37.9	25.8	19.3	0.91	1.26	1.12
78.5	51.5	44.8	51.9	32.9	26.2	0.9	1.06	1.21
58	65.5	33.9	37.3	40.6	25.1	1.43	0.93	0.77
82.6	42.1	39.7	60.6	24.6	28	0.68	1.11	0.75
52.9	52.7	34.3	40.1	36.7	22.7	0.59	0.72	0.81
53	48.6	34.2	33.8	33.2	23.2	0.89	0.77	0.81
61.8	38	28.3	40.9	26	19.4	0.9	0.82	0.78
34.6	29.1	26.3	21.8	19.9	16.4	0.91	0.83	0.85
68.2	63.4	59.7	37.4	41.8	44.5	1.23	0.87	0.64
59.7	58.2	42.1	35.1	31.6	24.6	1.15	1.35	1.11
58.3	52.7	42.7	30.6	20.1	23.5	1.34	2.1	1.04

MCA (R)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
53.8	56.9	33.5	36.1	29.6	21.5	0.96	1.27	0.81
52.3	53.8	40.7	39.2	40.7	25.9	0.66	0.61	0.94
64.5	68.5	46.9	44.3	51.1	29.8	0.78	0.68	0.94
52.5	46.8	39.1	34.7	30.4	26.4	0.84	0.85	0.78
46.2	37.6	59.1	27.7	22.2	14.9	1.11	1.13	3.92
53.1	61.6	26.8	36.3	39.1	18.3	0.83	1.11	0.79
53.8	56.9	33.5	36.1	29.6	21.5	0.96	1.27	0.81
81.2	40.4	41.5	56.6	26.7	28.3	0.78	0.91	0.75
58.5	55.5	35.7	28	26.3	19.4	1.53	1.49	1.22
54.6	43.8	41.6	34.8	30.5	23.5	1.01	0.73	1.16
76.6	54.8	46.2	52.3	37.6	27.7	0.87	0.85	1.11
77.7	80.2	26.3	47.9	48.7	13.8	1.03	0.99	1.2
70.1	85.4	83.5	49.4	42.9	49.3	0.72	1.41	1.32
99.3	82.3	46.5	70	51.5	29.3	0.73	0.86	1.03
57.9	44.8	35.8	40.1	29.4	23.5	0.82	0.93	0.79
40.8	59.6	21.7	22.5	39.6	13.8	1.24	0.81	0.85
67	61.2	50.2	46	37.9	38	0.7	0.91	0.57
69.7	54.6	44.3	48.7	37.8	29.6	0.74	0.76	1.15
58	56.9	40.6	38.1	32.1	23.6	0.91	1.21	1.39
49.1	50.3	45.4	28.5	21.6	19.8	1.04	1.74	1.63

ACA (L)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
61.1	49.9	33.8	30	27.6	18.4	1.69	1.16	1.2
52.3	77.6	69.4	37.8	62.1	28.5	0.75	0.68	1.35
48.1	64.7	60.2	35.2	47.6	38.9	0.79	0.76	0.98
35.5	42.5	33.1	20.7	25.2	24.5	1.05	1	0.62
54	45.6	40	39.4	25.2	20.7	0.68	1.36	1.34
50.3	32.1	24.2	37.2	23	16.6	0.72	0.74	0.75
31	35.2	49.9	43	20.6	27.6	0.76	1.03	1.16
57.7	46.9	37.5	41.7	32.2	24.7	0.69	0.77	0.84
25.7	26.4	33	16	13.9	16.6	0.9	1.23	1.53
51.5	29.3	45.4	37.9	19.2	23	0.65	0.86	1.39
56.5	43.2	65.9	39.6	26.6	39.5	0.88	1.06	1.21
72.7	59.2	54	46.5	32.6	33.5	0.9	1.15	0.85
85.5	42.8	86.3	59.6	25.1	68.5	0.75	1.14	0.49
40.6	73	44.4	27.2	63.1	28.9	0.86	0.63	0.81
76.7	62.2	23.3	53	44.4	16.8	0.8	0.77	0.66
55.1	53.3	28	34.5	33.5	19.4	0.88	0.94	0.76
43.3	35.4	33.8	26.5	24	18.4	0.97	0.85	1.2
50.2	62.9	59.7	35.1	44.7	44.5	0.62	0.71	0.64
50.7	42.9	35.4	39	24.7	24	0.85	1.18	0.85
34.9	41.8	32.3	21.1	22.9	29.3	1.01	1.15	0.2

ACA(R)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
54	48.4	32.8	29.3	30.6	18.6	1.24	0.93	1.06
52.9	37.7	36.8	35.4	22.6	20.3	0.9	1.27	1.35
63.4	44.8	44.5	41.8	32.4	28.4	0.87	0.71	0.91
51.4	39.7	32.1	34.4	29	21.7	0.83	0.67	0.84
42.5	38.2	34.7	26.6	20.3	20.4	0.98	1.32	1.03
50.6	38.4	23.8	35.3	24.8	16.9	0.77	0.89	0.72
52.9	48.4	32.8	35.4	30.6	18.6	0.9	0.93	1.06
78.6	45.2	39.9	60.6	30.6	27.5	0.59	0.93	0.74
53.4	33.3	42.4	35.2	16.4	23.5	0.79	1.39	1.26
56.2	40	33.6	40.6	29.7	19.7	0.68	0.6	1.07
50.3	39.6	38.6	28	26.7	22	1.26	0.83	1.32
44.3	62.9	36.4	29.9	37.6	22.2	0.82	0.98	0.97
96.7	69.5	70.1	69.5	33.7	49.4	0.73	1.54	0.72
96.5	84.1	37.9	74.6	62	25.1	0.54	0.65	0.96
88	38.1	31.1	64.6	23.1	24.7	0.65	0.97	0.49
40.3	52.6	25.2	23.9	27	16.6	1.06	1.29	0.82
49.5	56.8	44.3	32.5	50.1	29.9	0.87	0.3	0.82
47.4	43.4	39.9	30.2	27.8	29.4	1.07	0.92	0.65
63.7	38.9	40.3	45.6	24.1	24.2	0.87	1.05	1.05
46.5	42.8	42.7	28.4	20.7	23.5	0.97	1.49	1.04

GROUP LATERAL

SL.No	GROUP NO.	GROUP	Age(Years)	Sex(M/F)	Weight (Kg)	Height	BMI	Diagnosis	Surgery	Co morbidity	ASA Grading
41	3	LATERAL	38	M	80	176.5	25.7	Left CP Angle Schwanomma	Left rsso craniotomy and excision		I
42	3	LATERAL	28	F	48	147	22.2	Right Thalamic glioma	Left rsso craniotomy and excision		I
43	3	LATERAL	44	F	47	158	18.8	Right CP Angle Schwanomma	Right rsso craniotomy and excision	DM	2
44	3	LATERAL	38	M	60	169	21	Left CP Angle Schwanomma	Left rsso craniotomy and excision		I
45	3	LATERAL	48	F	55	160	21.5	Left CP Angle Meningioma	Left rsso craniotomy and excision		I
46	3	LATERAL	59	M	70	162	26.7	Right CP Angle Schwanomma	Right rsso craniotomy and excision		1
47	3	LATERAL	45	F	48	147	22.2	Right CP Angle Schwanomma	Right rsso craniotomy and excision		1
48	3	LATERAL	48	M	80	170	27.7	Right CP Angle Schwanomma	Right rsso craniotomy and excision		1
49	3	LATERAL	44	F	80	175	26.1	Right CPA lesion	Right rsso craniotomy and excision		1
50	3	LATERAL	19	F	55	160	21.5	B/L Vestibular Schwannoma	Left rsso craniotomy and excision		1
51	3	LATERAL	53	F	65	160	25.4	Left CP Angle Schwanomma	Left rsso craniotomy and excision	HTN	2
52	3	LATERAL	55	F	48	147	22.2	Left CP Angle Schwanomma	Left rsso craniotomy and excision		1
53	3	LATERAL	48	F	57	160	22.2	Right tentorial meningioma	Right rsso craniotomy and excision		1
54	3	LATERAL	60	F	60	160	23.4	Lower cranial nerve Schwanomma	Left rsso craniotomy and excision		1
55	3	LATERAL	47	F	61	154	25.7	Left CPA Schwanomma	Left rsso craniotomy and excision		1
56	3	LATERAL	37	M	80	175	26.1	Right CPA lesion	Right rsso craniotomy and excision		1
57	3	LATERAL	58	F	60	163	22.5	Left CP Angle Schwanomma	Left rsso craniotomy and excision		1
58	3	LATERAL	55	F	60	169	21	Left CP Angle Schwanomma	Left rsso craniotomy and excision		1
59	3	LATERAL	35	F	45	150	20	Left CP Angle Epidermoid	Left rsso craniotomy and excision		1
60	3	LATERAL	24	F	40	151	18.5	Left CPA Schwanomma	Left rsso craniotomy and excision		1

Heart Rate			SBP			DBP			MAP		
T0	T1	T2	T0	T1	T2	T0	T1	T2	T0	T1	T2
75	83	80	130	118	115	70	66	62	90	83	80
65	74	78	114	108	106	72	69	73	86	82	84
74	80	82	116	110	108	66	60	62	83	77	77
79	83	80	136	128	120	78	72	69	97	91	86
73	80	82	140	129	132	84	79	72	103	96	92
69	74	76	135	129	128	82	79	72	100	96	91
66	69	72	130	119	115	70	65	63	90	83	80
73	77	80	146	134	140	92	87	90	110	103	107
85	90	92	119	112	118	77	75	76	91	87	90
58	62	61	112	102	118	67	60	76	82	74	90
57	60	61	108	118	118	60	66	76	76	84	90
80	84	79	141	138	135	92	90	88	108	106	104
79	83	80	136	122	130	90	81	81	105	94	97
65	70	71	124	112	118	58	75	76	80	87	90
66	73	76	117	112	109	72	69	73	87	83	85
72	80	81	116	110	108	66	60	62	83	77	77
88	95	84	124	104	109	58	61	69	80	75	82
75	80	76	124	114	116	70	65	69	88	81	85
81	85	79	124	114	116	70	65	69	88	81	85
59	65	62	120	121	125	71	76	80	87	91	95

PEAK Airway Pressure		ETCO2		Tidal Volume		Respiratory Rate	
T1	T2	T1	T2	T1	T2	T1	T2
18	19	35	35	480	480	12	12
20	21	33	33	400	400	12	12
19	21	35	35	390	380	12	12
19	20	34	34	425	425	12	12
19	22	33	33	400	400	12	12
22	24	35	35	450	450	12	12
21	24	34	34	410	410	12	12
18	19	35	35	500	500	12	12
18	19	35	35	500	500	12	12
20	22	33	33	420	420	12	12
20	23	34	34	450	450	12	12
19	21	34	34	375	375	12	12
22	25	34	34	475	475	12	12
20	22	35	35	450	450	12	12
17	18	33	33	450	450	12	12
20	23	35	35	500	500	12	12
18	19	34	34	430	430	12	12
17	19	34	34	440	440	12	12
23	25	33	33	380	380	12	12
18	21	33	33	350	350	12	12

NIRS (L)			NIRS (R)		
T0	T1	T2	T0	T1	T2
69	70	60	67	67	54
54	68	68	64	69	69
76	70	60	72	67	54
65	62	66	68	64	69
55	58	54	62	61	59
64	61	61	62	60	59
67	70	66	69	65	61
73	72	71	74	80	77
71	70	76	61	64	69
72	71	73	69	72	68
61	60	64	65	56	58
64	62	65	58	62	69
68	66	67	63	58	60
60	57	62	63	62	65
67	61	65	69	63	66
79	75	77	72	68	70
66	62	60	73	61	62
70	73	72	73	62	66
65	73	76	68	76	70
60	62	63	64	67	59

MCA (L)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
68.2	63.4	24.2	37.4	41	17.5	1.23	0.87	0.71
79.4	85	70	53.4	58.1	43	0.74	0.75	1.07
52.1	61.1	26.3	36.8	30	16.4	0.82	1.69	0.85
68.2	63.4	59.7	37.4	41.8	44.5	1.23	0.87	0.64
62.7	48.1	47.2	45.7	33.3	30.5	0.66	0.8	0.86
95.1	61.6	59	65	42.3	40.5	0.84	0.78	0.81
65.9	73.4	23.8	44.8	48.6	15.1	0.89	0.92	1.02
55.2	23.1	41.7	44.4	16.1	33.1	0.67	0.75	1.23
53.3	41.1	27.4	36.1	23.6	17.5	0.83	1.17	0.99
63.7	91	49.5	41.9	53.6	32	0.85	1.02	0.86
65.9	60.9	41.5	41	40.7	28.2	1.24	0.9	0.88
78.2	45.2	42.4	69.9	30.7	26.8	0.37	0.81	1.25
86.4	50.8	41.5	56.9	30.6	28.2	0.91	1.15	0.88
45.7	41.7	34.4	24.9	33.1	19.4	1.42	1.23	1.22
68.2	61.1	53.3	37.4	30	36.1	1.23	1.69	0.83
42.3	58.1	66.4	23.6	29.9	48.5	1.33	1.31	0.64
55.4	49.6	38.1	37.8	25.1	23.7	0.85	1.73	1.03
48.1	44.6	36.9	29.3	26.3	19.8	1.13	1.22	1.37
84	45.2	54.9	59.7	32.5	37.8	0.66	0.77	0.78
98.7	89	86.1	61.2	57.5	63.2	0.94	0.84	0.69

MCA (R)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
63.5	61.1	33.5	41	33.9	21.5	0.9	1.16	0.81
61.1	41.3	63.5	33.9	31.1	41	1.16	0.71	0.9
53.8	56.9	33.5	36.1	29.6	21.5	0.96	1.27	0.81
69.7	54.6	44.3	48.7	37.8	29.6	0.74	0.76	1.15
52.5	46.8	39.1	34.7	30.4	26.4	0.84	0.85	0.78
64.1	50.4	37.9	43.3	43.5	25.8	0.89	0.64	0.82
70.4	95.1	47.8	48.9	67.5	33.4	0.87	0.72	0.79
57.6	32.9	36.1	42.1	22.6	24.8	0.67	0.74	0.78
53.2	47.2	34.4	37	24	19.4	0.75	1.39	1.22
80.1	48.3	35.2	57	31	21.2	0.75	0.99	1.23
89.4	75.9	49.4	56.4	50.9	31.5	1.02	0.78	0.93
37.6	48.5	32.4	24.5	35.6	22.2	0.91	0.68	0.82
52.9	45.9	38.3	33.1	27	23.4	1.01	1.16	1.1
44.6	29	32.4	27.3	18.2	22.2	1.17	1.02	0.82
57	56.3	44	35.4	37	23.8	1.25	1.09	1.18
38	77.5	44.2	22.7	48.1	24.5	1.25	0.91	1.17
56.1	50	39.3	33.7	29.1	24.8	1.15	1.13	0.96
43.7	47.9	40.5	27.2	27.2	21.7	1.14	1.24	1.34
80.2	55.1	55.6	54.5	36.5	34.3	0.79	0.85	0.96
50.9	72.7	81.3	34.6	52.8	52.9	0.87	0.84	0.85

ACA (L)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
42.5	34	33.8	49.9	21.2	18.4	0.51	0.91	1.2
50.2	62.9	59.7	35.1	44.7	44.5	0.62	0.71	0.64
61.1	49.9	33.8	30	27.6	18.4	1.69	1.16	1.2
50.2	62.9	59.7	35.1	44.7	44.5	0.62	0.71	0.64
42.5	35.5	32.8	25.2	20.7	24.9	1	1.05	0.6
75.2	48.4	40.7	51.8	34.1	29.7	0.86	0.7	0.66
56.7	53.2	30.4	36.8	39	20.5	0.96	0.77	0.9
24.6	23.6	29.7	18.5	16.7	21.7	0.61	0.73	0.66
40.4	46.5	36.4	25.5	27.8	20.6	1.13	1.03	1.07
54.7	55	40.8	32.8	32.8	27	1.05	1.05	0.84
52.7	40.3	38.1	35.2	26.8	25.6	0.83	0.84	0.88
37	35.6	31.1	22.2	24.2	21.2	1.16	0.79	0.85
45.8	53.2	40.4	32.2	39	25.5	0.95	0.77	1.13
35.7	35.2	33.8	22.1	21.2	18.4	1.03	1.23	1.2
53.2	44.5	31.9	39	54.8	24.8	0.77	0.72	0.61
53.1	40.4	40.2	38.7	25.5	35.5	0.64	1.13	0.25
49.9	56	28.4	42.3	42.5	18.9	0.65	0.66	0.82
52.4	52.7	39.5	30.8	30.2	22.6	1.19	1.22	1.2
61.2	37.3	49.6	43.1	23.6	34.8	0.67	0.92	0.73
63.8	57.6	66.3	48	46	45.8	0.56	0.83	0.76

ACA(R)								
Peak flow velocity			Mean flow velocity			Pulsatility Index		
T0	T1	T2	T0	T1	T2	T0	T1	T2
42.3	39.5	34.8	23.7	23.7	20.4	1.22	1.05	1.06
47.4	43.4	39.9	30.2	27.8	29.4	1.07	0.92	0.65
54	48.4	32.8	29.3	30.6	18.6	1.24	0.93	1.06
47.4	43.4	39.9	30.2	27.8	29.4	1.07	0.92	0.65
51.4	39.7	32.1	34.4	29	21.7	0.83	0.67	0.84
61.5	50.4	49	41.6	43.5	82.5	0.83	0.64	0.34
66.6	59.3	63.2	47.1	74.7	44.8	0.75	0.48	0.8
49.2	30.1	30.2	33.9	22	21.3	0.74	0.64	0.75
56.9	40.2	34	38.9	25.5	19.5	0.8	0.95	1.07
71.5	58.9	48.5	47.2	35.4	28.4	0.84	1	1.21
75.8	47	53.4	49.3	34.6	35.9	0.87	0.66	0.87
65.7	49.9	40.2	43.6	41.9	25.5	0.88	0.65	0.95
46.7	33.7	36.1	26.9	21.3	21.9	1.16	1.06	1.05
43.9	33.3	34	29.6	20.2	19.5	0.96	1.08	1.07
48.8	41.2	37	33.2	29.1	22.2	0.89	0.86	1.16
34.6	78.6	40.3	21.8	42.2	21.5	0.98	1.17	1.2
71.3	30.3	33.7	49	21.4	23.5	0.87	0.77	0.79
50	53.5	26.3	45.7	34.7	14.2	0.71	0.97	1.25
78	52.5	44	60.6	35.4	34.1	0.57	0.82	0.58
44.1	37.8	40.9	31.7	28.5	29.4	0.76	0.75	0.76

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