Observational study of the effect of beach- chair position on cerebral blood flow in patients undergoing shoulder surgery

A Thesis submitted to the Tamil Nadu Dr. MGR Medical University in partial fulfillment of the degree MD ANAESTHESIA.

By

Dr Jesudoss A

Christian Medical College and Hospital, Vellore,

Tamil Nadu - 632004

India.

CERTIFICATE

This is to certify that the dissertation titled “Observational study of the effect of beach- chair position on cerebral blood flow in patients undergoing shoulder surgery” is a bonafide work of Dr. Jesudoss A in partial fulfillment of the requirements for the MD Anesthesia (final) examination of the Tamil Nadu Dr. MGR medical university to be conducted in April 2017.

Signature:

Dr. Anna Pulimood Principal, Christian Medical College, Vellore	Dr. Sajan Philip George, Guide and Head of The Department, Department of Anesthesiology, Christian Medical College, Vellore.

DECLARATION

I hereby declare that this dissertation titled **"**Observational study of the effect of beach- char position on cerebral blood flow in patients undergoing shoulder surgery**"** was prepared by me in partial fulfillment of the regulations for the award of the degree of MD ANAESTHESIA of the Tamil Nadu Dr.MGR Medical University, Chennai. This has not formed the basis for the award of any degree to me before and I have not submitted this to any other university previously.

Jesudoss A

Vellore

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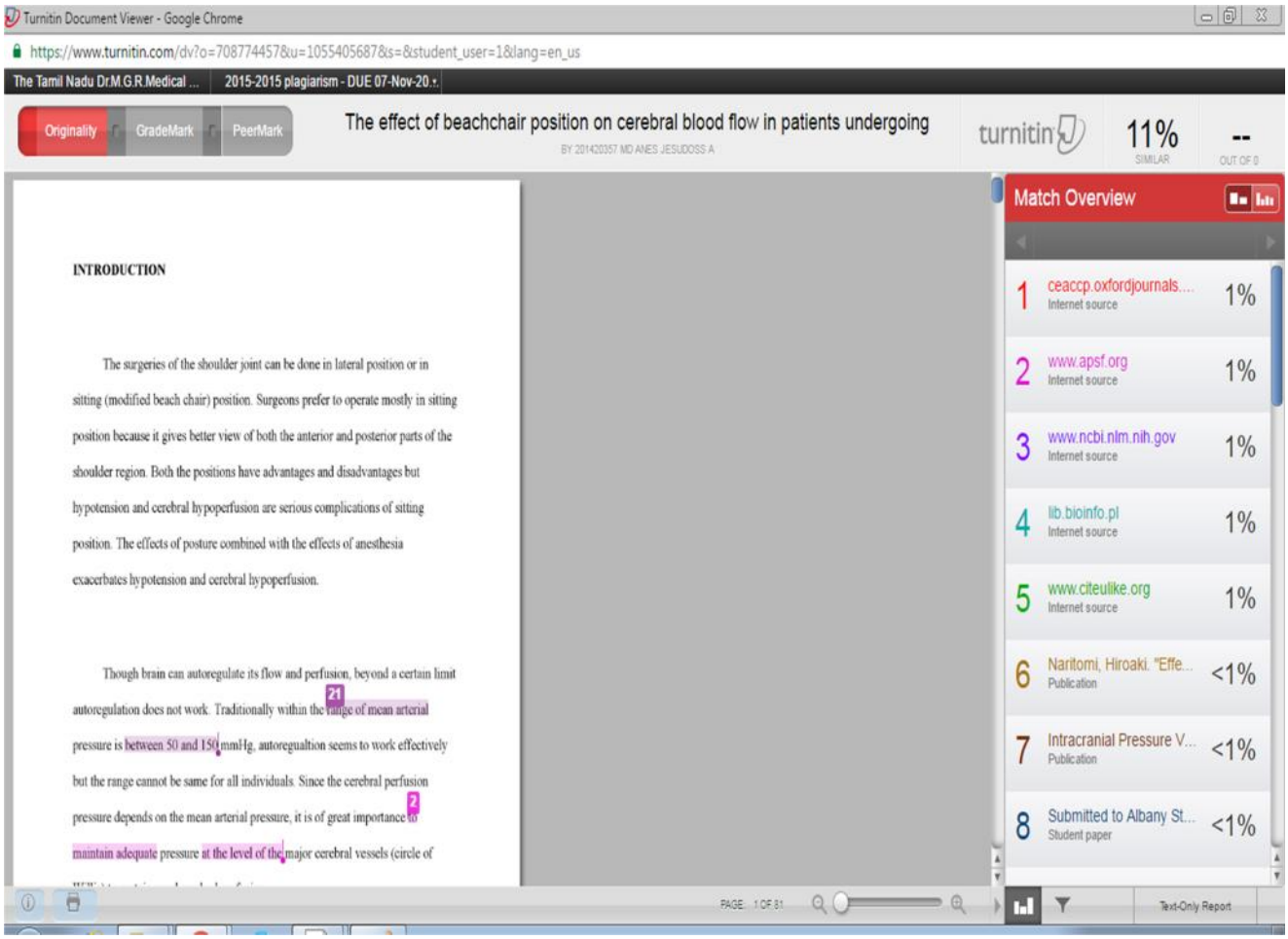
TURNITIN ORIGINALITY REPORT – ANTIPLAGIARISM REPORT



ABSTRACT

TITLE: "Observational study of the effect of beach- chair position on cerebral blood flow in patients undergoing shoulder surgery"

DEPARTMENT: Anesthesiology

NAME OF THE CANDIDATE: Dr. Jesudoss A

DEGREE & SUBJECT: MD Anaesthesia

NAME OF THE GUIDE: Dr. Sajan Philip George

Background:

Shoulder surgeries are done usually in sitting or lateral position depending upon the preference of the operating surgeon. One of the commonly thought positional injury related to the sitting position is decreased cerebral perfusion due to fall in mean arterial blood pressure. Middle cerebral artery blood flow velocity by transcranial doppler is one of the surrogate methods to measure cerebral blood flow. The purpose of the study is to find about the correlation between the range of mean blood pressure fall and the cerebral blood flow (middle cerebral artery blood flow velocity). Indirectly we can derive how much drop in the mean blood pressure can be allowed and still maintain a good cerebral flow for an individual patient.

Methods:

An observational study was conducted in the department of Anesthesia in the Christian Medical College, Vellore from January 2015 to June 2016. All consenting patients who underwent shoulder surgeries in sitting position were included in the study according to the inclusion and exclusion criteria. Standard general anesthesia was given to all patients. In addition to pulse oximetry and electrocardiogram, invasive arterial blood pressure was monitored. Baseline value of middle cerebral artery blood flow velocity is measured by transcranial Doppler before and after induction in supine. After patient is positioned in beach chair position arterial blood pressure was read at the level of the tragus. Doppler was repeated in whenever there was hypotension and after treating it. The percentage reduction in mean arterial blood pressure and the middle cerebral artery blood flow velocity were compared.

Results:

Twenty patients were recruited for the study. The mean age was 38 years. There were 15% ASA II patients and 85% were ASA I. The reduction in the mean arterial pressure was 25% and middle cerebral artery blood flow velocity was 27.9% ($p<0.001$).

Except for the 6 patients who were overzealously treated after hypotension, an average of 73.41mmHg of the mean arterial pressure was required to maintain the middle cerebral artery blood flow velocity, thereby cerebral perfusion.

Conclusion:

There are statistically significant changes in mean arterial pressure and middle cerebral artery blood flow velocity. It was concluded that an average of 73.41mmHg is the minimum mean arterial pressure required to maintain cerebral perfusion.

Keywords: beach – chair, sitting, transcranial Doppler, middle cerebral artery, blood flow velocity

CONTENTS

INTRODUCTION

INTRODUCTION

The surgeries of the shoulder joint can be done in lateral position or in sitting (modified beach chair) position. Surgeons prefer to operate mostly in sitting position because it gives better view of both the anterior and posterior parts of the shoulder region. Both the positions have advantages and disadvantages but hypotension and cerebral hypoperfusion are serious complications of sitting position. The effects of posture combined with the effects of anesthesia exacerbates hypotension and cerebral hypoperfusion.

Though brain can autoregulate its flow and perfusion, beyond a certain limit autoregulation does not work. Traditionally within the range of mean arterial pressure between 50 and 150 mmHg, autoregualtion seems to work effectively but the range cannot be the same for all individuals. Since the cerebral perfusion pressure depends on the mean arterial pressure, it is of great importance to maintain adequate pressure at the level of the major cerebral vessels (circle of Willis) to sustain good cerebral perfusion.

One of the methods of measuring the cerebral blood flow is by transcranial Doppler. The middle cerebral artery blood flow velocity (measured by the Doppler) can be used as a surrogate of cerebral blood flow. This study aims at correlating the mean arterial pressure measured at the level of the major cerebral vessels (level of tragus) and the middle cerebral artery blood flow velocity.

AIMS AND OBJECTIVES

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To observe the effect sitting position on cerebral blood flow in patients undergoing shoulder surgery.

OBJECTIVES

Primary objective:

To compare the cerebral blood flow with invasive blood pressure in supine and beach chair positions in patients undergoing shoulder surgery

Secondary objective:

Correlation between mean arterial pressure measured at the level of tragus and the cerebral blood flow. To determine the mean arterial pressure required to maintain cerebral perfusion

REVIEW OF LITERATURE

Cerebral Blood Flow

Brain is a highly perfused organ, receiving approximately 14% of cardiac output. (54ml/ 100g / minute). The total flow to the whole brain is approximately 750 ml.

Sir Thomas Willis described the arterial circle (*circulus arteriosus cerebri*) formed by the two internal carotid arteries, basilar artery and communicating arteries to form a complete anastomotic ring. This gives rise to three pairs of anterior, middle and posterior cerebral arteries which supplies cerebral cortex. The cerebellum and brain stem are supplied by the vertebral and basilar arteries.(1),(2)

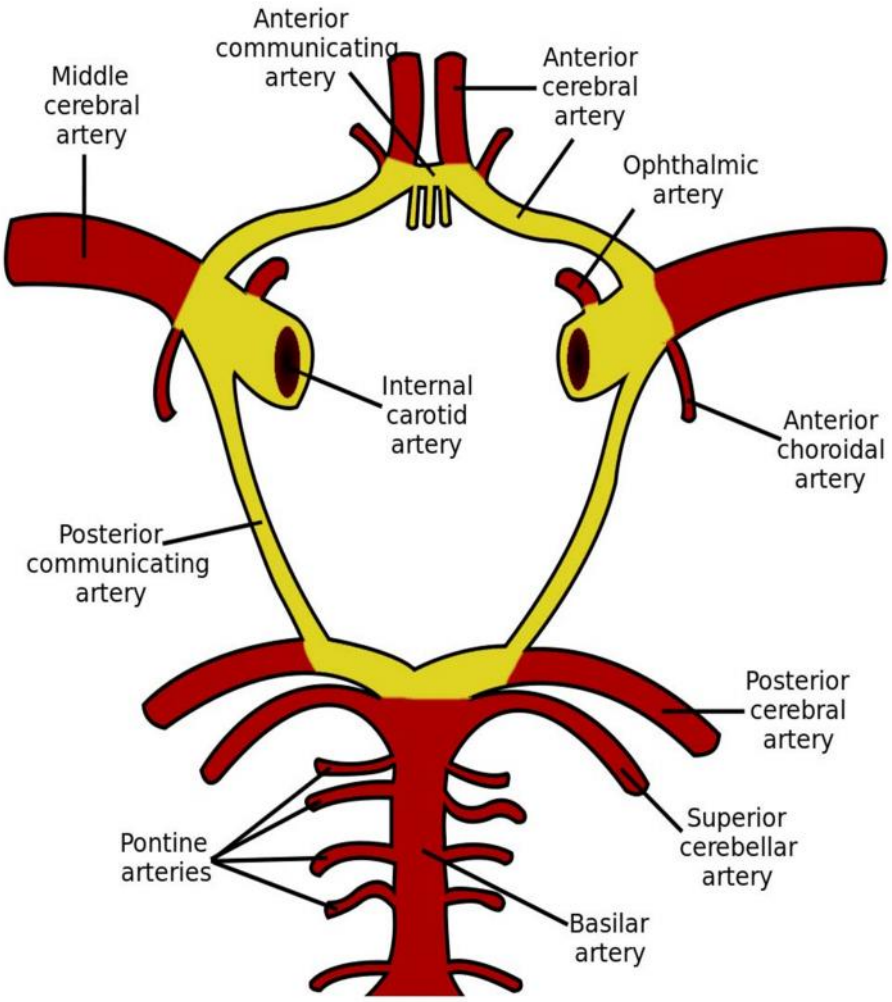


Figure1: ***Circulus arteriosus cerebri***

Autoregulation

Brain utilizes 20% of the oxygen need of the body, even though it is 2% of the total body weight. Brain requires glucose to provide ATP which is the fuel for its (i) basal metabolism i.e. for maintaining the trans-membrane ionic gradient (ii) for maintaining cellular integrity and (iii) synaptic transmission. Since there is no substrate reserve in the central nervous system, it cannot sustain anaerobic metabolism for more than a few minutes.

Auto regulatory responses maintain the internal milieu of the central nervous system. Flow-metabolism coupling and active vasomotion are the two clinically distinct processes involved in auto regulation.(3)

Flow Metabolism Coupling

Flow and metabolism are said to be coupled under physiological conditions and this is present even during sedation and general anesthesia. Roy and Sherrington hypothesized (4)that local metabolic factors are involved. There are studies which showed linear coupling between cerebral blood flow (CBF) and oxidative metabolic rate. The regional blood flow is several times higher than the oxidative metabolic rate.(4, 5)

Bayliss effect (myogenic response)

The smooth muscle cells of large arteries and smaller arterioles constrict in response to increased pressure and dilate in response to decreased pressure. It is a critical component of these vessels and more prominent in the cerebral vasculature.

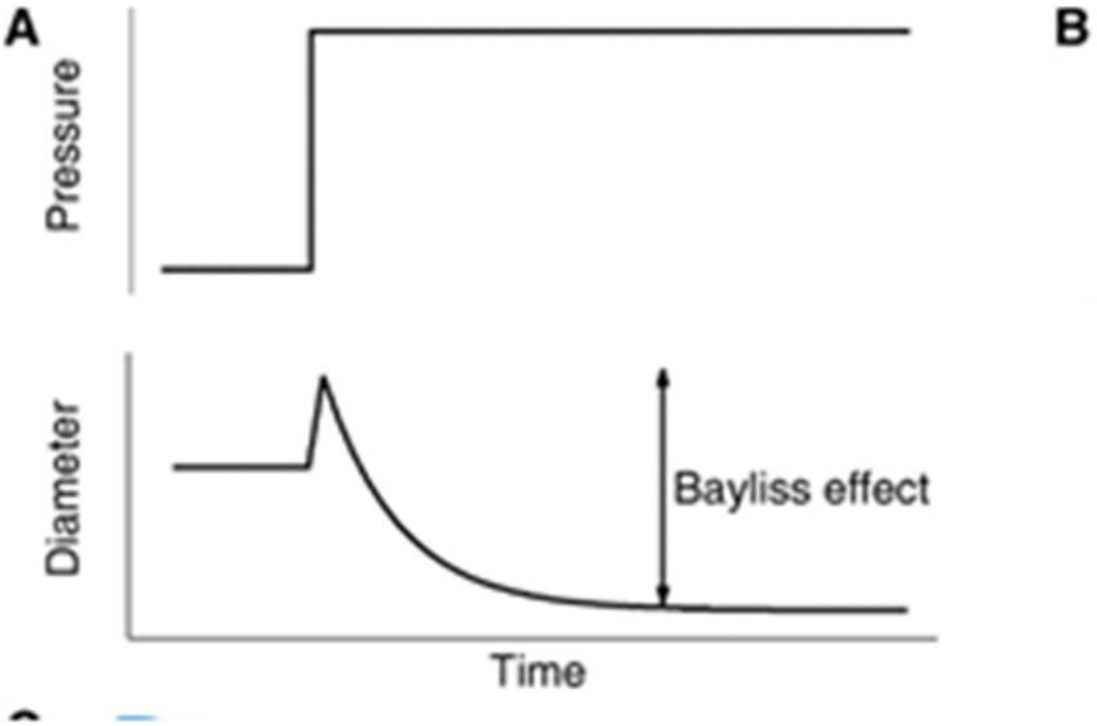


Figure 2: Bayliss effect

Regulation of CBF

1. Cellular mechanisms of cerebral vasomotion

a. Nitric Oxide (NO)

Studies show that Nitric Oxide may be involved in the basal cerebro-vascular tone by activating guanylatecyclase. It may also act through calcitonin gene related peptide and ATP-K Channels and thromboxane A2 which is a vasoconstrictor.

Nitric Oxide also plays a role in the vasodilatory response to the changes in perfusion pressure and hypercapnia.(3)

b. Vasoactive Peptides

Calcitonin gene-related peptide, substance P, and neurokinin A are some VAP present in the perivascular nerves. CGRP acts by increasing C AMP and partly mediates vasodilation in response to hypotension and cerebral ischemia.(3)

Substance P may be responsible for vasodilation in situations of cerebral and meningeal inflammation and edema.

c. Potassium Channels

K-ATP: ATP sensitive potassium channels are responsible for dilation response to hypotension, hypercapnia, acidosis and hypoxia.

K-CA: Calcium activated potassium channels are responsible for basal cerebrovascular tone.(3)

d. **Prostaglandins**

Prostaglandins play a significant role in the neonatal cerebral blood flow than that of the adult. Pg E2 and Pg I2 are the dilators and

Pg F2alpha and thromboxane A2 are the constrictors in the cerebral circulation that mediate hypercapneic vasodilation indirectly.(3)

e. Endothelin

It has been implicated in the vascular spasm after subarachnoid hemorrhage. Endothelin 1 and 3 secreted by the brain activates Endothelin A receptors and causes influx of extracellular calcium.(3)

2. **Cerebral Micro Circulation**

Highly tortuous capillary beds (increased path length) increases the transit time of red blood cells even though blood flow velocity is greater than other tissues. “Functional Reserve”: A small proportion of brain capillaries that have high RBC flow velocity do not participate in the substrate exchange. During cerebral hypo perfusion, the flow in these capillaries reduces, thereby improving the cerebral hemodynamics.(3)

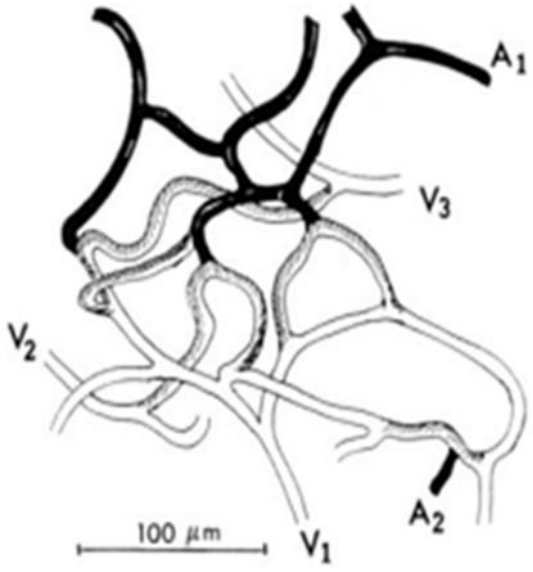


Figure 3: Cerebrocortical capillary network in brain: solid vessels indicate arteriolar capillaries, shaded vessels are true capillaries, empty vessels are venous capillaries. The major input of the network is via A1 with a secondary input by A2. There are four parallel pathways from A1 to the principal venule V1.

(i) Pressure regulation:

Cerebral blood flow is explained by Hagan-Poisseuille - Laminar flow equation. It states that there is a direct relationship between the flow, the caliber of cerebral vessels and the cerebral perfusion pressure (CPP).

$$\mathrm{CBF} = \frac{\pi \Delta P r^4}{8 \mu l}$$

π - Mathematical constant

ΔP - Pressure gradient (CPP)

r – Radius or caliber of blood vessel

μ - Dynamic viscosity of blood

l - Length of the blood vessel.

Hence any increase in CPP or cerebral vasodilation will lead to increase in cerebral blood flow.

$$\mathrm{CPP} = \mathrm{MAP} - \mathrm{ICP}$$

MAP: Mean arterial pressure

ICP: Intra cranial pressure

Because intracranial pressure is not measured in normal subjects cerebral perfusion pressure is not known. Normal ICP in supine person is between 5 and 15 mmHg.(6)

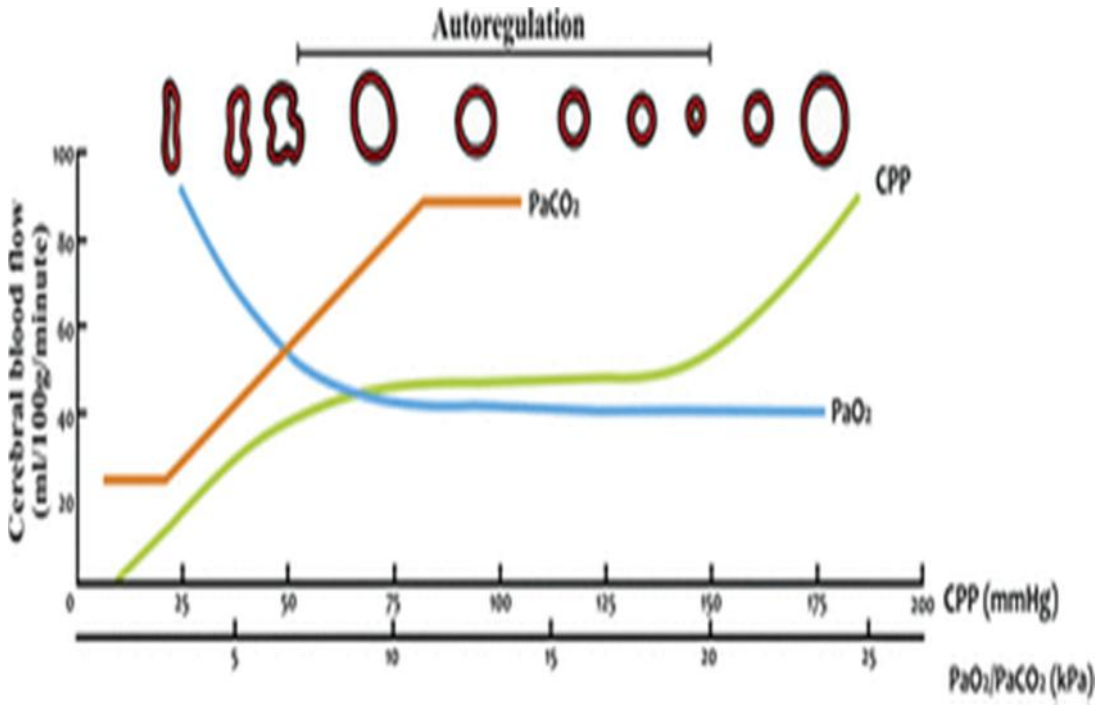


Figure 4: Relationship between cerebral blood flow and cerebral perfusion pressure, PaO_2(partial pressure of oxygen); $PaCO_2$(partial pressure of carbondoxide

Over a large range of mean arterial pressure the cerebral blood flow remains unchanged. The lower range of auto regulation is 50 mm Hg and the upper limit is 150mm Hg. Beyond these limits the flow is pressure dependent or pressure passive. The flow increases with increase and reduces with decrease in mean arterial pressure. It is also to be noted that even within the auto-regulatory range, a rapidly changing mean blood pressure will cause transient change in cerebral blood flow. (6, 7)

(ii) Venous physiology

Since large amount of cerebral blood volume is in the veins, any change in the venous diameter, can cause increase in the intra cranial blood volume and hence the intra cranial pressure. (3,8)

Monroe Kellie doctrine states that “in the setting of a non-distensible cranial vault, the volume of blood, CSF and brain tissue must be in equilibrium”. (8)

Studies showed that the venous system may be regulated by neurogenic factors rather than by myogenic or metabolic factors.

(iii) Rheologic factors

Some animal and human studies show that inverse relationship exists between the CBF and hematocrit. Hematocrit influences the viscosity of blood. Viscosity also determines the circulatory resistance as per the Hagen – Poisseuille law.

It states that,

$R = 8\ l\ \mu\ /\ r^4$, where l is length of conduct, μ is blood viscosity, r is radius of the vessel.

In anemic patients, cerebral vascular resistance will be decreased and hence blood flow is increased. Hematocrit ranging between 33% and 45% could result only modest variations in cerebral blood flow but beyond 45%, changes in blood flow in cerebral vessels will be more substantial. (6)

4. Metabolic & Chemical influences

a. Carbondioxide

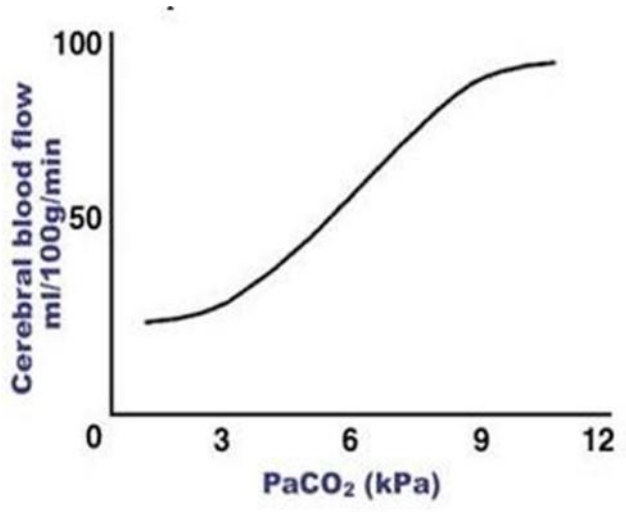


Figure 5: relationship between cerebral blood flow and partial pressure of carbondioxide($PaCO_2$)

Within physiological range, for every 1 mm Hg increase or decrease in partial pressure of carbondioxide in artery, there will be 3% increase or decrease in cerebral blood flow in a linear relationship. At normotension, there is a nearly linear response of cerebral blood flow at a $PaCO_2$ between 20 and 80 mm Hg. As $PaCO_2$ approaches extremes the linear response fades. This response is highly reproducible one.(3)

In a normal brain, cerebral blood volume is a 5 millilitre per 100g of brain and if $PaCO_2$ ranges from 25-70 mm Hg, cerebral blood volume changes about 0.049 millilitre per 100g for every 1-3 mmHg change in $PaCO_2$. For the brain tissue in an adult(which is about 1400g), there can be around 20 ml change in total cerebral blood volume if the $PaCO_2$ values from of 25 to 55 mm Hg.(6)

Arteriolar tone set by the systemic arterial blood pressure, modulates the effect of $PaCO_2$ on cerebral blood flow. It is blunted by moderate hypotension and is abolished by severe hypotension. Conversely, carbondioxide tension modifies pressure auto-regulation by widening the auto-regulatory plateau from hypercapnia to hypocapnia.(3)

b. Oxygen

Within the range of 60-300 mmHg of PaO2 there is no change in cerebral blood flow. PaO2 of less than 60 mm Hg increase cerebral blood flow rapidly. Cerebral blood flow roughly doubles at a PaO2 of 30 mm Hg. Hyperoxia decreases CBF by 10% - 15%. (3)

c. Temperature

Hypothermia reduces the rate of energy utilization by both electro physiological functions as well as the basal metabolism associated with the maintenance of cellular integrity. For every 1^{o} C reduction of temperature, there is 7% reduction in $CMRO_2$ (Cerebral metabolic rate for O_2). Because the cerebral blood flow is closely coupled with metabolism, there is a parallel decrease in cerebral blood flow with hypothermia – induced reduction in $CMRO_2$. Auto-regulation and carbondioxide reactivity are well preserved in moderate hypothermia.(3)

5. Neurogenic influences

There is a relative lack of humoral control as well as autonomic control over the normal tone in cerebral vessels. It is a major difference between systemic and cerebral circulation. Cerebral vasculature is extensively innervated by serotonergic, adrenergic and cholinergic systems. At the lower limits of auto-regulation, sympathetic activity modifies the auto-regulatory response of cerebral blood flow to a decrease in arterial blood pressure. At equivalent blood pressures, cerebral blood flow is lower during hemorrhagic hypotension than during pharmacologically induced hypotension. Parasympathetic fibers around major

cerebral vessels and the cortical pial vessels, mediate vasodilation through substance P, neutrokinin A and CGRP. Ischaemia stimulates these fibres causing vasodilation.

6. Clinical Consideration

a. Hypertensive Patient:

Chronic hypertension leads to increase in both lower and upper limits of the auto-regulation probably as a consequence of arterio-sclerosis.(9)

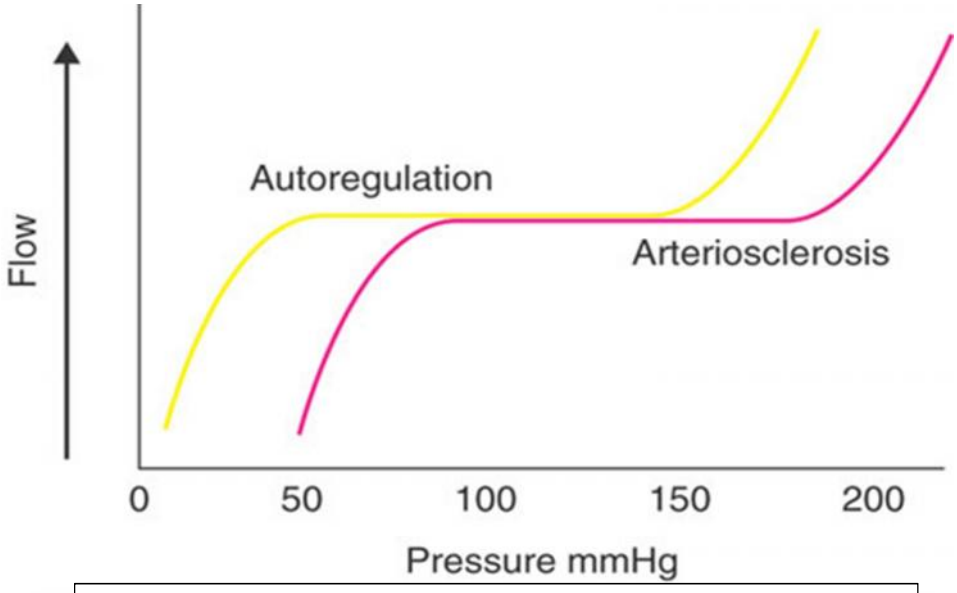


Figure 6: Autoregulation shifted to right due to arteriosclerosis

Although this has some protective effect against “break-through” caused by surpassing the upper limit of auto-regulation, it occurs at the expenses of lower limit. The sclerotic changes in the vessels and the shift in auto-regulatory response caused by hypertension are modified by treatment with antihypertensives for a long time.

CO_2 reactivity in this group is no different from the normotensive population. This finding underscores the probable difference in mechanisms between carbondioxide induced and blood pressure induced cerebral vasomotion.

b. Elderly Patient:

Some elderly patients who had postural hypotension were found to suffer from cerebral ischemia with very small decrease in blood pressure, which would not have otherwise occurred. This shows that ageing affects cerebral auto-regulation.(10)

In a study done in normal subjects and subjects with risk factors for stroke like diabetes mellitus, hypertension, hyperlipidemia etc,. it was shown that in normal volunteers, there was diffuse and progressive reduction of grey matter flow as well

as increases of cerebrovascular resistance with advancing age. Neuronal atrophy and in part cerebral arteriosclerosis play a role in reduction of grey matter flow with advancing age were most evident in the middle cerebral arterial distribution. The association of risk factors on enhances the reduction in flow.(11)

There is also variation of auto-regulation in elderly population. Some can tolerate a greater fall in mean arterial pressure whereas, even a minor fall in mean arterial pressure leads to cerebral ischemia.

In a study conducted in elderly healthy subjects done by quantitative T2 mapping MRI, it was found that there is reduction of cerebral blood flow between 0.5% and 0.7% per year with a high range of individual variation.(12)

7. Pharmacology

a. Intravenous anesthetic agents

The reduction in cerebral blood flow caused by these agents is primarily due to reduction in cerebral metabolic artery. Some agents have a direct effect on the vascular smooth muscle. Ketamine is the only drug which increases cerebral blood flow and cerebral metabolic artery($CMRO_2$).

b. Volatile anesthetics

They have intrinsic vasodilatory property and cause a dose dependent reduction in systemic blood pressure and modify cerebral auto-regulation. They reduce $CMRO_2$ in a dose dependent manner but at a slower rate. Certain studies have shown that at for isoflurane at a minimum alveolar concentration of 1.1 there is 19% increase in cerebral blood flow when the arterial blood pressure is kept within normal limits. Cerebral metabolic rate is reduced by about 45%. Recent studies have shown that in human, sevoflurane as well as desflurane reduce the cerebral blood flow significantly (compared to the cerebral blood flow in non-anesthetized patients). At 1.0 MAC concentrations, sevoflurance and desflurane decreased cerebral blood flow by 38% and 22% and CMR by 39% & 35% respectively.

CO_2 reactivity is well maintained with volatile agents.(6)

8. Pathological changes (Cerebral ischemia)

The brain uses large amount of energy but it can store only very less amount of energy. Therefore it is more susceptible to injury in case of cessation of energy (oxygen, glucose) supply. Neuronal function deteriorates progressively with declining cerebral blood flow rather than in an all-or-none fashion. The EEG

evidence of ischemia begins to appear only when cerebral blood flow has fallen to 20 ml/100g/min. When it drops to 15 ml/100g/min, the cortical EEG becomes isoelectric. Potentially irreversible membrane failure (elevated extra cellular potassium and loss of the direct cortical response) rapidly occurs only when cerebral blood flow is reduced to about 6 ml/ 100g/min.(6)

The ancient idea of ischemic insult and injury was that lysis of the neurons was restricted to the duration of ischemia and during the initial reperfusion stage. Recent data shows that neuronal lysis after an ischemic insult is an active process in which neurons continue to die for a prolonged duration after the trigger of ischemic insult. The severity of the ischemic insult determines the degree of the neuronal lysis.(6)

Most of the neurons undergo rapid lysis during severe ischemia. With more moderate insults, delayed death occurs for the neurons which escapes the initial insult. This dynamic process of neuronal loss contributes to the progressive expansion of cerebral infarction after focal ischemic injury. There are evidences of cerebral inflammation even after 6-8 months of the primary ischemic insult. This can further contribute to cerebral injury.

Cerebral neuroprotection and anesthetics

1. Barbiturates

Barbiturate therapy has been mainly useful in preventing neuronal injury due to hypoxia and ischemia. The neuroprotective effects of barbiturates are mainly by reducing the oxygen demand, increasing the oxygen delivery and by inhibiting the damaging pathological neuronal pathways. The exact mechanism of action is not completely known but the proposed mechanisms are reduction in cerebral metabolism ($CMRO_2$) by improving regional cerebral blood flow;

suppression of seizures; reduction in the intracranial pressure; loss of thermo – regulation; free radical scavenging effect; membrane stabilization. Studies on loss of thermo-regulation by barbiturates have been reviewed and found that there is some evidence that this group of anesthetic drugs protect neurons by inducing hypothermia. (13)

2. Volatile anesthetics:

Isoflurane is a powerful suppressant of cerebral metabolic rate in the cortex of cerebrum. There have been reports of protective effects (EEG evidence) in humans. It has been recently observed that neuroprotective efficacy of isoflurane is not lasting. When evaluated for neuronal lysis 2 days after ischemic insult isoflurane anesthesia is found to reduce the injury vigorously. But it was found on that after 14 days reduction in injury was much evident. This indicates that neuronal injury sustains into the post-ischemic recovery time and that the neuroprotection may not continue for a long period after the insult. (6)

Similarly sevoflurane also reduces ischemic injury but the efficacy of the sevoflurane does vary from halothane. Desflurane' role in neuroprotection is also similar to isoflurane. Therefore adequate depth of anesthesia alone may be protective compared with the unanesthetized state.

SHOULDER SURGERY

Positions for shoulder surgery

For shoulder surgery the selection of an appropriate position of the patient and its maintenance throughout the procedure is of major importance. The surgeon must support the part and work in such a way that he would gain easy access to the structures he wants to expose.(14)

1. **Lateral Position**

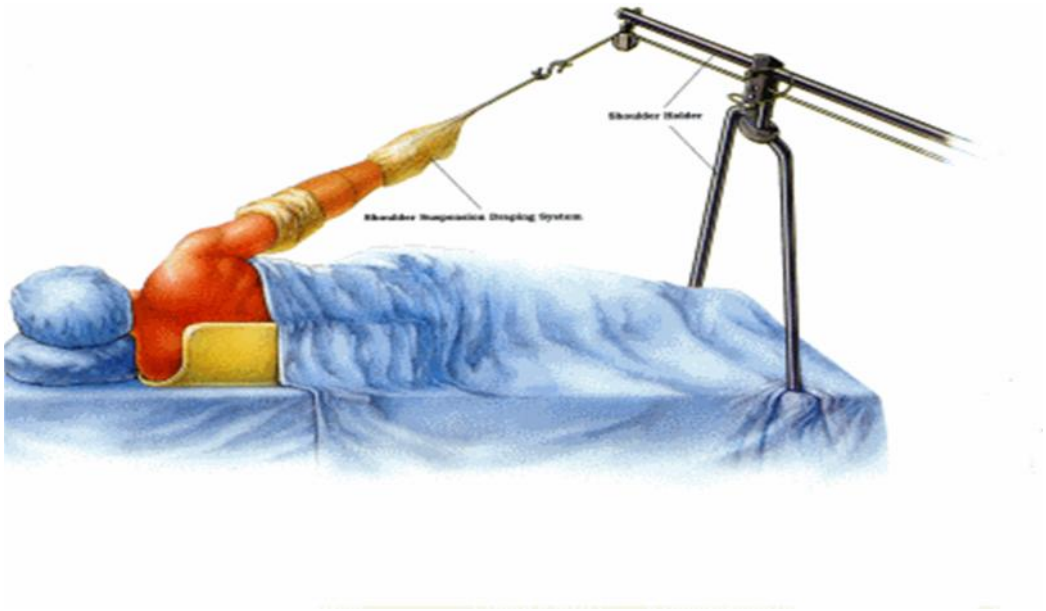


Figure 7: Patient in lateral decubitus position for shoulder surgery

As shown in the figure patient should be rolled into lateral position on to adjacent lateral positioning posts. A vacuum / bean bag can also be used. An axillary roll should be placed and care taken to ensure optimum ventilation and prevent traction injuries to brachial plexus.(15)

Advantages

When compared to sitting position the advantages of lateral position are better visualization and instrument access for some surgeries. There is decreased risk of reduced cerebral perfusion.

Injuries

Specific to the upper extremity traction in shoulder arthroscopy certain injuries have been reported. They are brachial plexus palsy, traction related soft-tissue injury, digital nerve compression, ischemic injury. When establishing antero-inferior portal there are chances of neurovascular injury and internal rotation of arm following gleno-humoral reconstruction in this position. (16)

In the dependent arm (in the lateral position) there can be injury to the brachial plexus. It is due to the stretching of the upper plexus caused by the patient's body weight compressing the arm which is abducted and internally rotated. Brachial plexus could get injured in the non-dependent side also. The non-dependent arm is usually suspended on the ether screen above the head. A modification of this position, in which the arm is held by a suspension toward the ceiling is used during shoulder procedures.(17)

Radial nerve palsy of the dependent arm is another serious injury which can happen in this position. For non-shoulder surgery incorrect positioning, positioning on a hard surface like arm boards without appropriate padding or a combination of both can cause compression of the radial nerve at the bend of the elbow.(18)

A bean bag should be available to stabilize patients while keeping them in lateral position. We should be careful that the soft tissues of the lower limbs are padded well against hard surface there by prevent compartment syndrome.

Peroneal nerve injury can happen when the fibular head is compressed against the operating table directly. Bony prominences such as greater trochanter, fibular head and ankle of the dependent side are to be padded properly. Saphenous nerve injury is prevented by placing pillows between the knees and ankles. Avoid excessive flexion or extension at the hip to prevent lumbar plexus or sciatic nerve injury.

To stabilize the patient, Anterior hip support is used. It is also a potential source of neuropraxia. If not properly padded, this may cause under pressure against public symphysis or anterior superior iliac spine. Improperly positioned hip support can occlude large blood vessels. There are case reports of femoral artery occlusion in obese patients due to the anterior hip support device.

2. Sitting or beach chair position

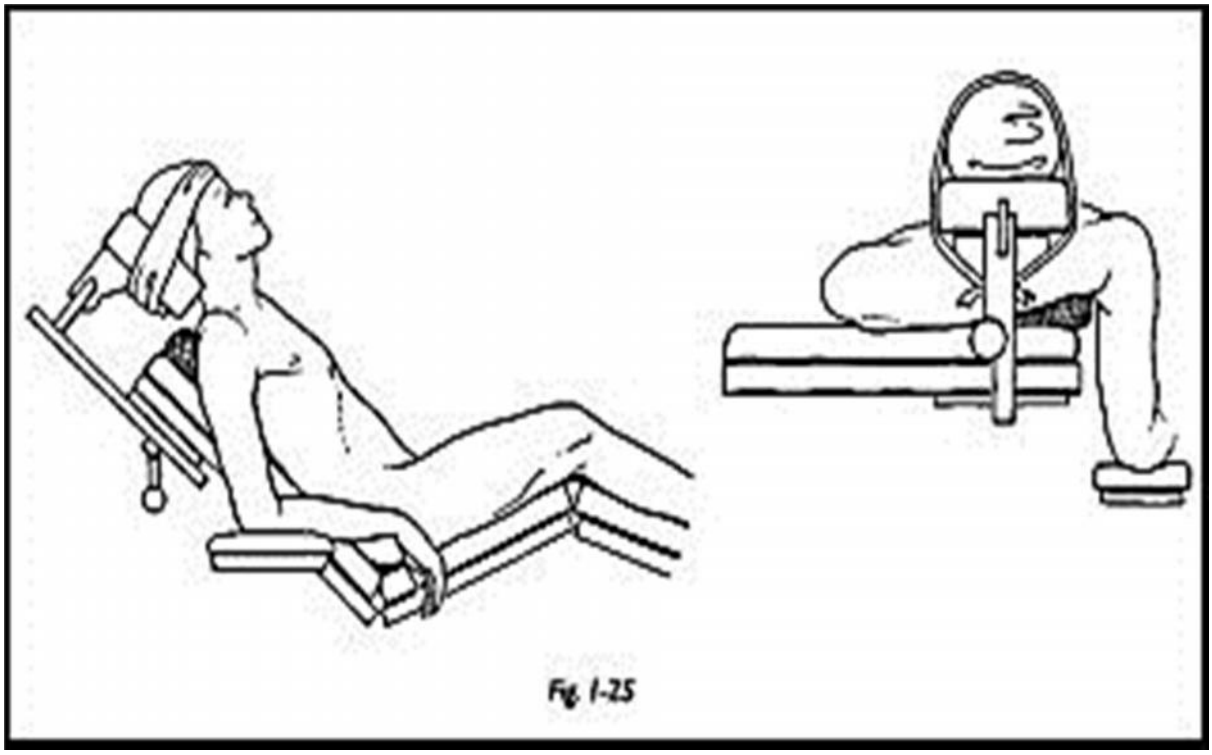


Figure 8: Patient in beach-chair position for shoulder surgery

It has been found that supine position for shoulder surgery was uncomfortable and does not provide adequate access to superior posterior and lateral aspects of shoulder. The uncomfortable posture for surgeons leads to mental & physical fatigue.

In 1925, surgeons used sitting position for doing shoulder arthrodesis in children affected by anterior poliomyelitis (for the first time in St. Louis unit of Shriners' Hospital for crippled children) They found that only in this position they could palpate bony landmarks and secure optimum position for function.(14)

As shown in the picture, the beach chair (sitting) position is used for most of the anterior approaches of the shoulder surgeries. The torso and head are elevated to an angle of about 20-30^{o} to horizontal. The legs are kept flexed at knee joints using a pillow to avoid over -stretching of the hamstrings. The legs are maintained little raised than the horizontal to increase the venous return. Sequential compression devices are placed on legs to prevent deep venous thrombosis.

Pressure points like elbows and heels are carefully padded. The ulnar nerve injury can happen due to undue pressure at the elbow. The un-operated arm is comfortably placed onto a padded arm holder. It must be ensured that there is slight flexion and abduction of the shoulder. This is to avoid stretching of brachial plexus. The head must be positioned on a head rest and the neck should be in neutral position. Care must be taken to avoid excessive stretching of the brachial plexus due to surgical traction.(19,20)

Disadvantages

Postural hypotension, reduced cerebral blood flow, macroglossia, quadriplegia, injury to brachial plexus and sciatic nerve are the possibilities. Greater auricular nerve neuropraxia and incidents of ischemic optic neuropathy are also recorded. (21,22)

Advantages

- Easy exposure of the surgical field.
- Though associated with incidents of cerebrovascular events, it is not an independent risk factor for stroke.(23)

Surgical Procedures

1. Open Surgery

Repairs for instability, subacromial decompression, acromioplasty, fracture fixation and arthroplasty are the most commonly performed open surgical procedures.

Approaches

(i) Anterior approach: where the incision starts just lateral to the tip of the coracoid upto the axillary crease.

(ii) Extended approach, (in shoulder arthroplasty)

(iii) Posterior approach is rarely used.

2. Arthroscopic surgery

Both diagnostic and therapeutic procedures are done through arthroscopy. The main post sites are posterior and lateral. The posterior site is not covered when only interscelene block is given. Hence additional local anesthetic infiltration is required.

Effect of beach-chair position on cerebral blood flow during arthroscopic shoulder surgeries

Arthroscopic shoulder surgeries in sitting position requires the trunk of the patient to be positioned at various degrees to the horizontal depending upon whether open or arthroscopic procedure. A minimum of 20 to 30^{o} elevation is used usually(19). Normal physiological changes which occur during sitting position are due to gravity and are increased during anesthesia. In sitting position venous return reduces, which is worsened by the vasodilating and myocardial depressant effects of anesthesia. This causes reduction in stroke volume and cardiac output by approximately 20%. In case of awake patients, there should be an increase in systemic vascular resistance by upto 50% to 80%. This is caused by autonomic reflex. The anesthetics block the sympathetic system and cause more vasodilatation

and reduction in cardiac output. Venous return from the cerebral circulation is increased by inspiratory sub-atmospheric pressure during spontaneous ventilation, but this mechanism is nullified by positive-pressure ventilation. Obstruction of the internal jugular veins in the sitting position may also impede cerebral venous drainage, especially with unfavorable positions of the head and neck such as flexion of the head.

In a case series where the head elevation was 90^0 to the horizontal the reduction in mean arterial pressure was around 30%. At the end of the procedure there was significant neurological dysfunction like hemispheric infarct, posterior circulation infarct, and even brain death in two patients.(22)

Generally cerebral perfusion pressure is auto-regulated adequately if the mean arterial pressure is between 50-150 mm Hg. It was Drummond who argued that the actual values must be more and the lower limit of auto-regulation should be calculated on a patient to patient basis derived from the individual's resting mean arterial pressure. The lower limit of auto regulation in an awake patient is considered to be a mean arterial pressure of about 25% less than the baseline value. If the blood pressure is reduced beyond the range of 40% to 50% symptoms of cerebral hypoperfusion begin to appear.(22)

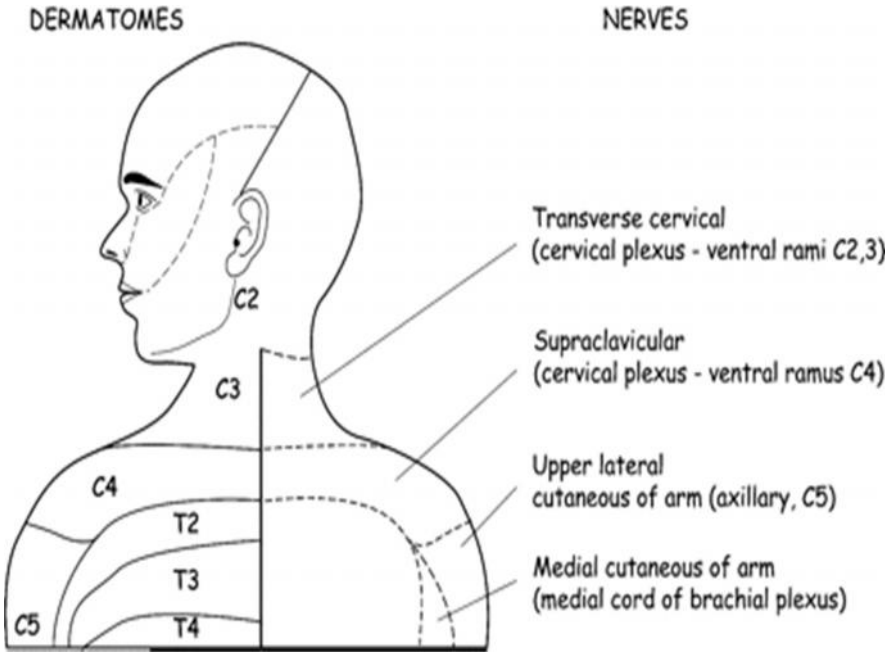
Anesthesia for Shoulder Surgeries

There are numerous approaches of anesthesia for shoulder surgery and each technique should be tailored to the surgical and patient requirement. For short procedures nerve block can be sufficient if the patient is willing. Longer procedure usually requires general anesthesia. In case of regional anesthesia, sedating the patient might lead to loss of airway. Hence regional with light sedation or formal general anesthesia can be used.(20)

Even in case of general anesthesia, regional blocks can be used to reduce severe postoperative pain which limits early ambulation and discharge. It reduces the usage of opioids as analgesic, thereby reducing the adverse effects of opioids. It also increases the patient satisfaction. Continuous infusion via interscalene catheter is better than most of the regional techniques.

REGIONAL ANESTHESIA

Innervation of shoulder



The shoulder region is supplied by nerves of cervical and brachial plexus. The cervical plexus supplies the skin over the clavicle, the shoulder tip, and first two intercostal spaces anteriorly via the superficial cervical plexus and supraclavicular nerves. Brachial plexus via the upper lateral cutaneous branch of axillary nerve supplies skin over deltoid muscle. The medial cutaneous nerve of the arm, intercosto-brachial nerve supply the medial side of the arm and axillary region.

The acromioclavicular joint and capsule of the glenohumeral joint are supplied by suprascapular nerve. The inferior aspect of the capsule and glenohumeral joint.is supplied by the axillary nerve (19)

Techniques of regional anesthesia for shoulder surgery

Interscalene block

This is the best regional intervention for providing analgesia for shoulder surgeries along with general anesthesia. The modified-Winnie approach is the commonly used method. Utrasound can be used for localization and local anesthetic infiltration. This block is associated with complications like phrenic nerve block,

stellate ganglion block, pneumothorax and inadvertent injection into epidural space or vertebral artery.(19)

Meier modification approach is used to insert perineural catheter along the interscalene groove. This is used to infuse local anesthetics using elastomeric ballon pumps or syringe pumps.

The advantages are we can totally avoid opioids and patient can be fully ambulant. We can give patient controlled analgesia through the perineural catheters. Some centers discharge the patient with catheter in situ and simple elastomeric pump.

The cervical paravertebral block and suprascapular nerve blocks are other options of regional anesthesia. These are used in patients, with contra indications for interscalene block.(19)

General Anesthesia

Patients undergoing arthroscopic shoulder surgery may be awake, undergo conscious sedation (with regional blocks) or general anesthesia with either a supraglottic airway or tracheal tube. The anesthesia practice varies widely from country to country as well as between institutions.

Extravasation of irrigation fluid in should arthroscopy is common during subacromial procedures. The subacromial space is not enclosed within a capsule. There are multiple case reports in the literature of airway obstruction due to tracheal compression and laryngeal edema from extravasated fluid.

Methods of monitoring cerebral blood flow

Hemispheric methods

(i) It means global measurements of cerebral blood flow. It is otherwise called as one-dimensional method. This was first described by Kety-Schmidt. They used nitrous oxide as the substrate to measure global cerebral blood flow, based on Fick's principle. This method has the disadvantage of being cumbersome and invasive because it requires retrograde catheterization of the jugular bulb and arterial blood sampling.

(ii) $AVDO_2$ the arterio-venous difference in oxygen content:

This method is based on the principle that if cerebral metabolic activity remains constant, the relative changes in the arterio-venous difference in oxygen content on unit reflect global cerebral blood flow. This is also an invasive procedure because an oximetric – catheter is to be inserted into the jugular bulb.(3)

Two Dimensional Method

(i) Xenon Clearance

On the basis of Kety's work, Lassen and Ingvar developed methods to determine cortical regional cerebral blood flow. The radioactive tracer 133 xenon is injected into cerebral arterial supply and cerebral washout was followed using external scintillation counters placed over skull. This requires carotid artery puncture.

This has been modified into intravenous and inhalational xenon which are less invasive.(3)

(i) Thermal Clearance

This method of measuring cerebral blood flow is to be done on the exposed cortex. It has a high resolution of less than 1cm brain area. It has a possibility of repeated measurement.(3)

(ii) Cold Xenon

This method is non-invasive and has high resolution. The disadvantages are its high cost and longer time taken to measure the blood flow.

(iii) Tomography

Tomography methods are relatively high cost, take several minutes to measure the cerebral blood flow and the scope of repeated measurement is limited.

(a) Xenon-enhanced computed tomography

Nonradioactive or stable xenon is used in conjunction with computed tomography to quantity CBF. Regional blood flow is measured with the help of anatomic delineation of structures by CT scan.

Disadvantage:

(i) unfavourable signal / noise ratio

(ii) anesthetic effect of xenon due to high dose usage

(iii) Some studies show that xenon increases intracranial pressure when used beyond certain concentration.(3)

(b) Perfusion Tomography

(c) Position Emission Tomography

Measurement of cerebral blood flow with the use of several tracers and techniques has been described. The earliest used tracer was ^{15}O-labelled CO_2 which was given through inhalation. The ^{15}O is rapidly transferred to $H_2{}^{15}O$ by carbonic anhydrase in RBC. After 10 minute, tracer entry into brain is in equilibrium with venous flow and radioactivity decay. The arterial input function is assessed from peripheral blood. Partition coefficient and flow limitation of $H_2{}^{15}O$ as a tracer are some of the drawbacks of PET CT studies.(3)

(d) Single Photon Emission

(iv) Perfusion Weighted Magnetic Resonance Imaging

This method provides a higher resolution (<1cm) three dimensional picture of cerebral blood flow taking several minutes to measure with limited possibility of repeated measurements. MRI resolution and the ability to correlate cerebral blood flow information with structural information could potentially make this “the gold standard”.

Doppler Methods

(i) Laser Doppler Flowmentry

(ii) Transcranial Doppler Ultrasonography (also hemispheric method)

TRANSCRANIAL DOPPLER (TCD)

TCD is a non-invasive technique used to measure blood flow, velocity in basal cerebral arteries. It was introduced in 1982 by Aaslid et al.(24). Basically a wave formed produced by the moving red blood cells is visualized by TCD.(25) Then the velocity of those cells is calculated by means of Doppler principle.

Advantages

Of the various mentioned methods of measurement of cerebral blood flow, the choice depends upon the local availability of equipment and expertise, cost, subject, derived anatomic resolution (one or two dimensional). A particularly important consideration is the ability to perform repeated measures in a given subject.(3)

TCD is easy and quick to do repeatable and low cost. It is also non-invasive. Hence it has become a widely utilized method to evaluate the basal cerebral arteries for various disease processes.(25)

It has been shown in various studies that the changes in the middle cerebral artery blood flow velocity correlate with changes in the cerebral blood flow. The absolute velocity cannot be used as an indicator of absolute cerebral blood flow. Hence TCD can be used as a surrogate method of measurement of changes in cerebral blood flow. (26, 27)

Equipment

- **Pulsed wave Doppler**

A transducer generates pulses of ultrasound which are sent into the patient and echoes are produced. These echoes return to the transducer and are detected and displayed as Doppler waveforms. TCD uses pulsed waved Doppler which allows the technologist to change the depth (in millimeters) and follow a vessel along its course. Each vessel is identified at certain depth. The middle cerebral artery and basilar artery are long vessels that are insonated at multiple depths along their course. Pulsed wave Doppler allows for direction of flow, either toward or away from the probe.

- **Frequency** is the number of cycles a sound wave goes through in one second. The frequency of the probe is inversely proportional to the depth of insonation. For insonating shallow vessels high frequency probe is used and for insonating deeper vessels low frequency probe is used. TCD uses low frequency probe of about 1.5 to 2 MHZ, which can insonate the intracranial vessels which lie deep in the brain.

- **Sample volume** is the length of the vessel in millimeters from where the Doppler wave forms are obtained. It is usually from 6 to 8 mm.(25)

- **Intensity / Power** is the amount of energy dissipated into the tissue which is converted into heat. While doing transorbital window it is important to reduce the power and keep it between 10% and 25%. (according to the FDA limits) (24)

- **Positioning**

All windows except transforamenal (suboccipital) window can be done in supine. For insonating basilar artery through suboccipital window patient will be either in prone or in lateral. If the patient needs to be in supine, thin neck is exposed by keeping a rolled towel under head and shoulder.(28)

Probe positioning varies for different window. The vessels in the circle of Willis lie in different angle which requires various angulations of the probe to insonate them. The general condition is that the probe should be kept parallel to the vessel and the blood flow. It will be difficult to insonate a tortuous vessel.(25)

- **Windows**

The areas in the head through which the ultrasound beam can travel easily are called acoustic windows.

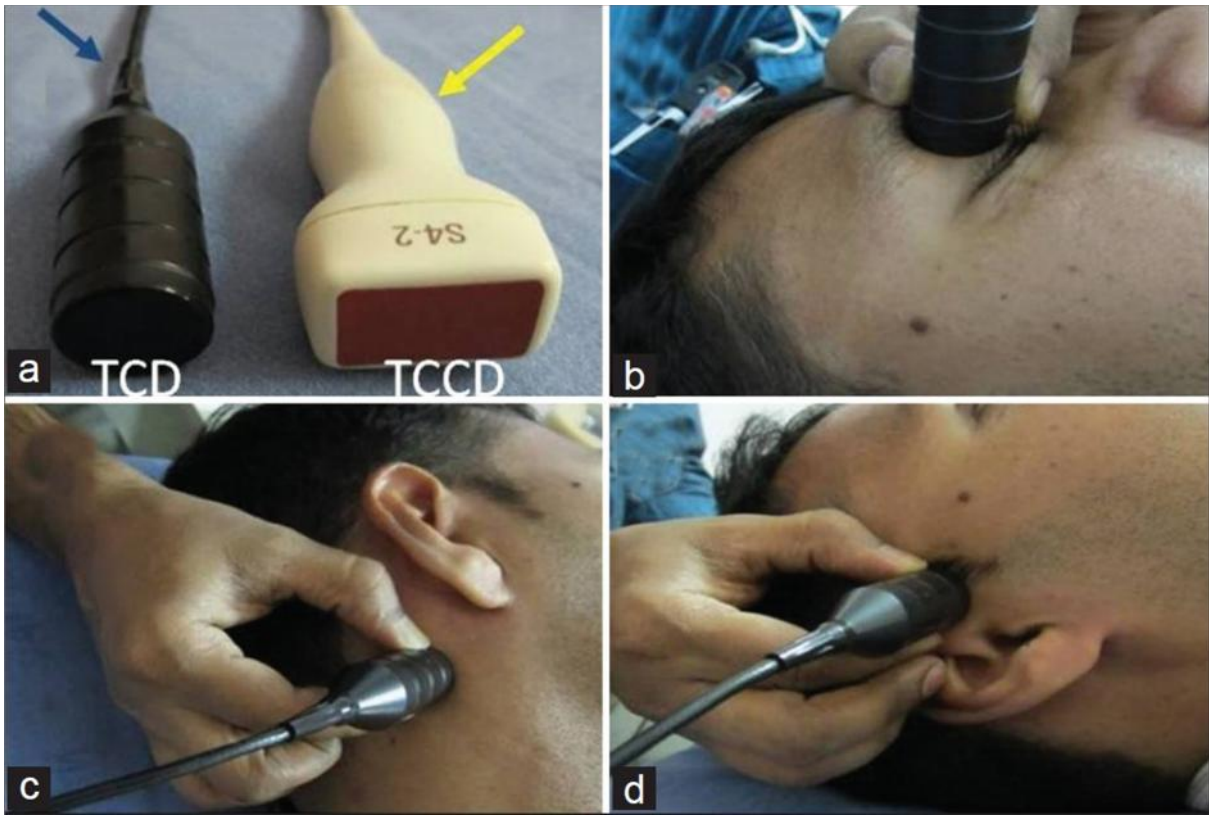


Figure 9: a- TCD transcranial doppler probes

b- Transorbital window

c- Retromandibular window

d- Transtemporal window

(i) The Retro mandibular window

Otherwise called as submandibular window, it is situated below the angle of mandible. The probe is to be pointed superiorly and slightly medial to insonate extracranial part of the internal carotid artery.

(ii) The Transtemporal window

The probe is placed over the temporal area above the zygomatic arch and in front of the tragus. The entire window is divided into anterior, pre auricular and posterior regions which are to be scanned for the strongest possible signal. Here we can insonate middle, anterior and posterior cerebral artery.

Pre auricular region is the commonly used window. The probe is placed just in front of the ear and pointed straight or slightly superior and anterior to insonate middle and anterior cerebral arteries. It will be pointed posteriorly for insonating posterior cerebral artery.

(iii) The Tranforamenal window

The soft past below the bony cranium in the middle of the neck is palpated and the probe is placed in this region. This window is useful in insonating basilar and vertebral artery through foramen magnum. (25)

(iv) The Transorbital window

This is used to evaluate ophthalmic artery and carotid siphon. The probe is placed over the eyelid and pointed slightly medial and upward.(28)

Waveform and variables

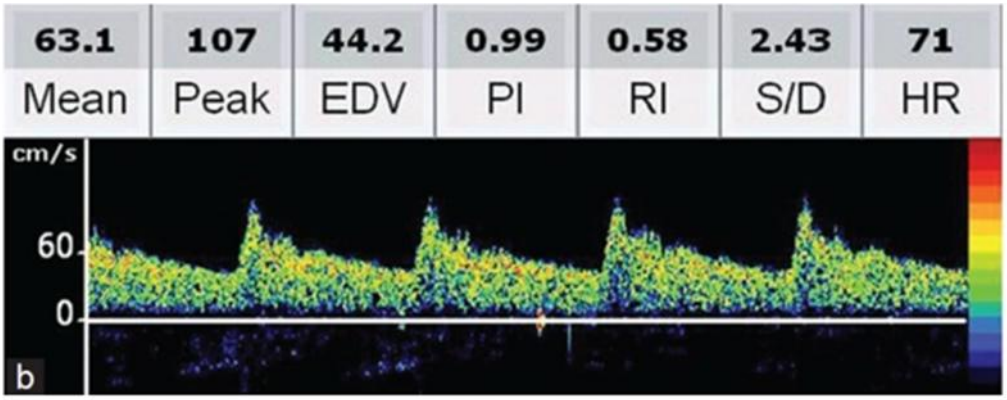


Figure 10: Transcranial Doppler waveform

PI: pulsatality index, RI: resistivity index, HR: heart rate

- **Peak Systolic Velocity (cm/s)**

It is the first peak on every TCD waveform for each pulse. Rapid upstroke rules out any stenotic lesion.

- **End-Diastolic Velocity (cm/s)**

It is seen in all major intracranial arteries which will be between 20 and 50% of peak systolic velocity.

- **Mean Flow Velocity**

It is calculated as end-diastolic velocity plus one third of the difference between peak systolic velocity and end diastolic velocity. It is the highest in middle cerebral arteries.

- **Pulsatility index**

It is the difference between systolic and diastolic flow velocities divided by the mean flow velocity. Pulsatility index value ranges from 0 to infinity. 0 means systolic and diastolic velocity are the same. In case of infinity mean velocity is 0. It is generally applied to vascular beds in skin and muscles where resistance and impedance to flow are high.

- **Resistivity index**

It is the difference between peak systolic and diastolic flow velocity divided by systolic velocity. This index is commonly applicable in vascular beds with low oscillatory and low impedance. Example: cerebral and renal vessels.

The normal values of velocity in different arteries are given in the table below.

ARTERY	WINDOW	DEPTH mm	MEAN VELOCITY cm/ sec	DIRECTION OF FLOW
ECICA	Retromandibular	45 – 50	30 +/- 9	Away
MCA	Transtemporal	30 – 65	50 +- 12	Toward
ACA	Transtemporal	60 – 75	50 +/- 11	Away
PCA – P1	Transtemporal	60 – 70	39 +/- 10	Toward
PCA – P2	Transtemporal	60 – 70	40 +/- 10	Away
OA	Transorbital	45 – 55	21 +/- 5	Toward
Supracliniod ICA	Transorbital	65 – 80	41 +/- 11	Away
Parasellar ICA	Transorbital	65 – 80	47 +/- 14	Toward
VA	Transforaminal	60 – 75	38 +/- 10	Away
BA	Transforaminal	80 – 120	41 +/- 10	Away

Table1: ECICA extracranial internal carotid artery, MCA middle cerebral artery, ACA anterior cerebral artery, PCA posterior cerebral artery, OA ophthalmic artery, ICA internal carotid artery, VA verterbral artery, BA basilar artery

METHODS

- Patients admitted for shoulder surgery under Orthopaedics department will be chosen according to the inclusion and exclusion criteria. Informed consent will be taken
- Inside the operation room, with patient in supine position intravenous line will be secured. Before induction his/her radial artery will be cannulated under local anesthesia for invasive blood pressure measurement (MAP).
- Transcranial doppler will be used to monitor middle cerebral artery blood velocity – Vmca (as surrogate for cerebral blood flow monitoring) which will be done in the side opposite to the operating side.
- Data will be collected pre-induction in supine (NIBP & IBP- (MAP), HR, SPO2, Vmca)
- After preoxygenation patient will be induced with Fentanyl 2mcg/kg, Propofol 2-3mg/kg, will be relaxed and intubated, mechanically ventilated and maintained with isoflurane (MAC 0.9 - 1). ET_{CO2} will be maintained between 30 and 35mmHg.
- Data will also be collected after induction in supine, immediately after positioning the patient in beach chair position followed by every

fifteen minutes till the end of the surgery and post extubation in supine. Transcranial doppler will be done whenever there is a fall in blood pressure and after it has been corrected.

- Blood pressure will be maintained MAP>60 mmHg in normotensive patients and MAP>70 mmHg in hypertensive patients.
- Data will be analyzed.

RESULTS

DEMOGRAPHIC VARIABLES

AGE DISTRIBUTION

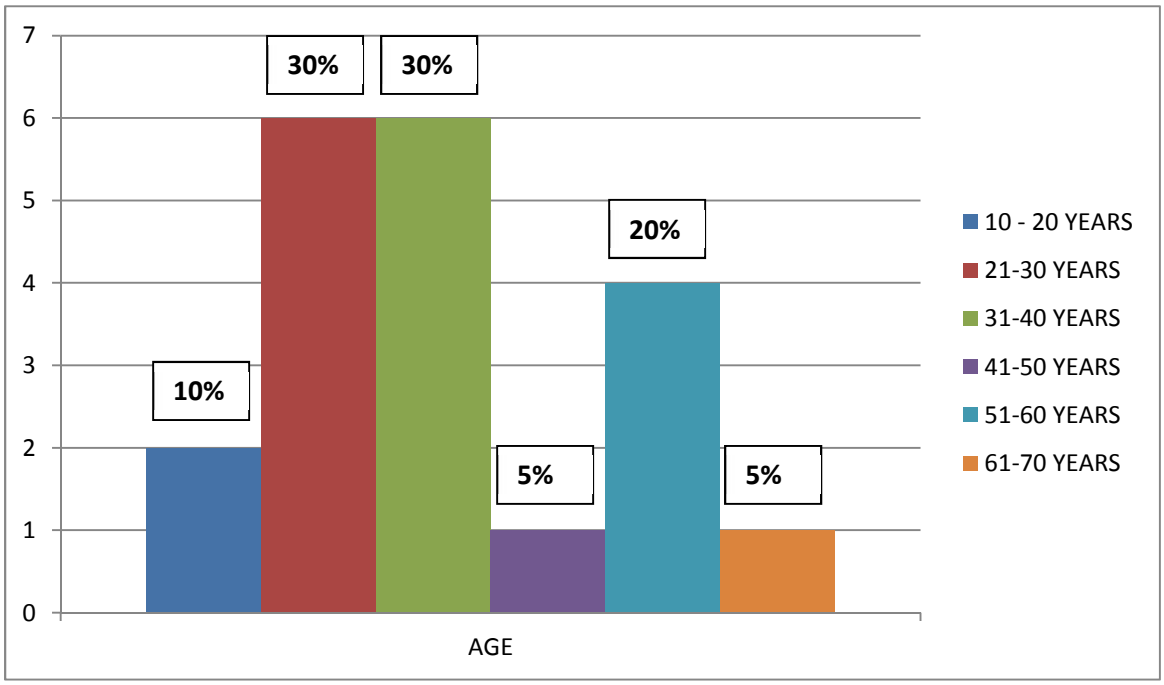


Figure 11: Age distribution of the patients (n=20).

There were totally 20 subjects. The age of the patients ranged from 18 to 65 years with mean (standard deviation) as 38 (14) years. 60% patients are between 21 to 40 years of age. 10% patients are from the age of 18 to 20years.20% of the patients are from 51 to 60 years.

GENDER DISTRIBUTION

Out of the 20 patients recruited for the study only one was a female patient which is only 5% of the total.

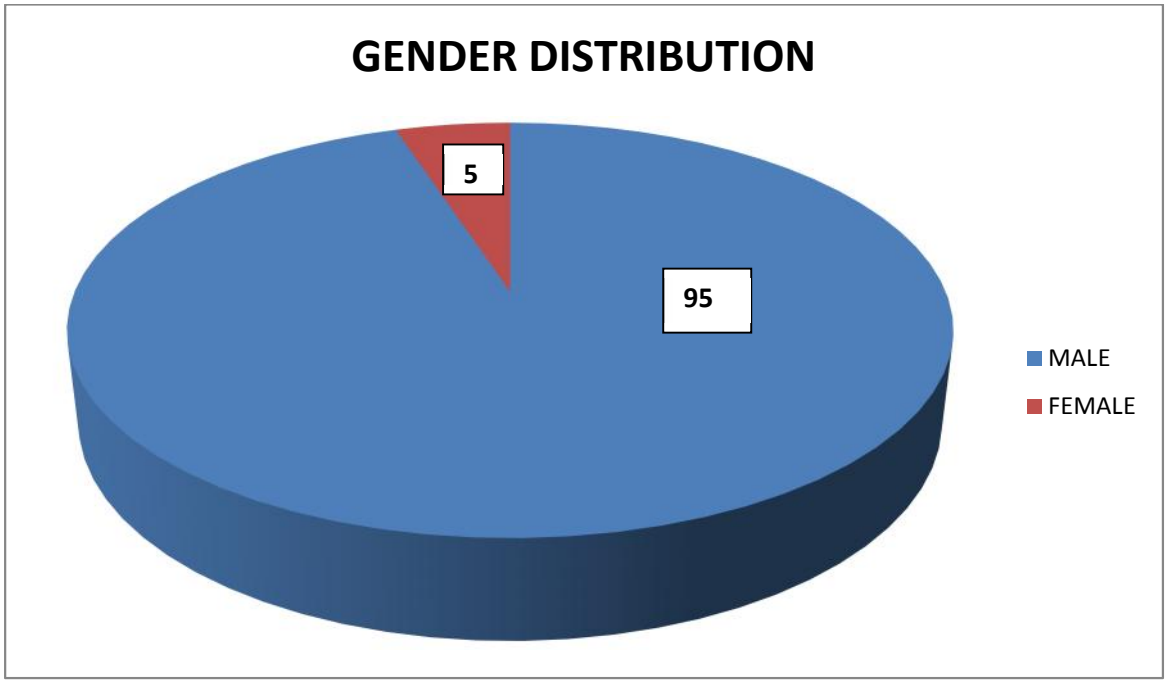


Figure 12: Distribution of the patients based on their Gender (n = 20).

ASA RISK DISTRIBUTION

As shown in figure 13, 3 patients were ASA risk II (which is 15%) and 18 were ASA I (85%). ASA III and above were excluded from the study. Out of three patients two of them were diabetics and one was both diabetic and asthmatic patient. All of them were well controlled with oral hypoglycemic agents.

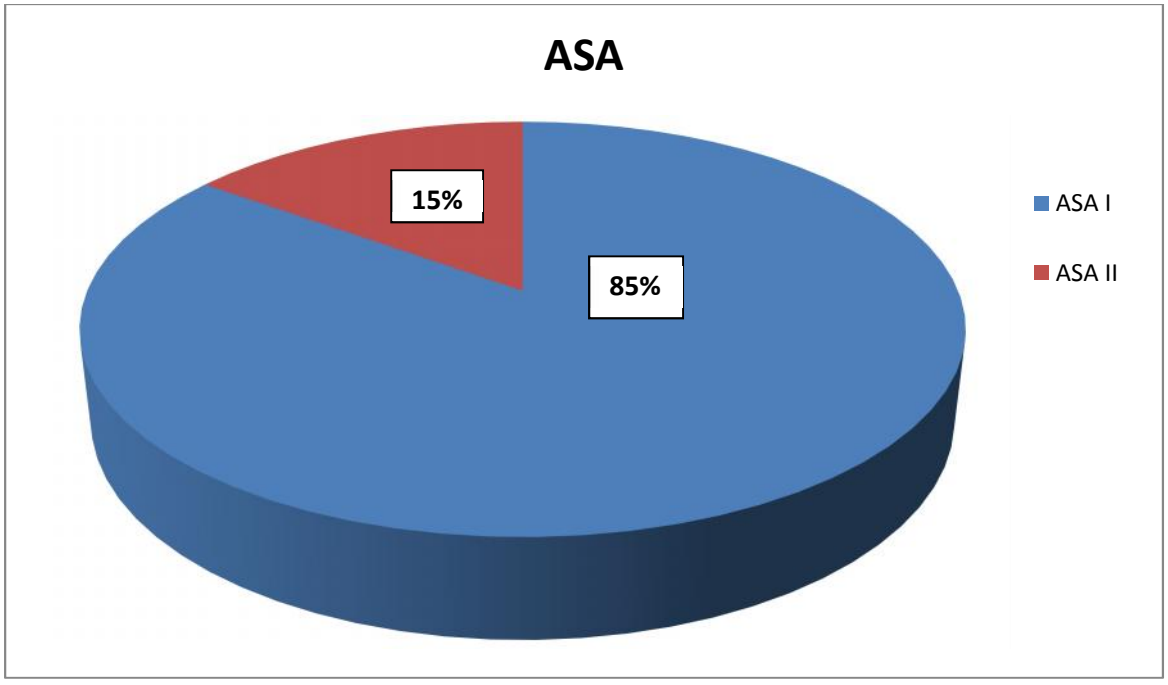


Figure 13: Distribution of patients according to ASA risk

PRIMARY OUTCOMES

1: CHANGE IN MEAN ARTERIAL PRESSURE AFTER POSITION CHANGE (FROM SUPINE TO BEACH CHAIR)

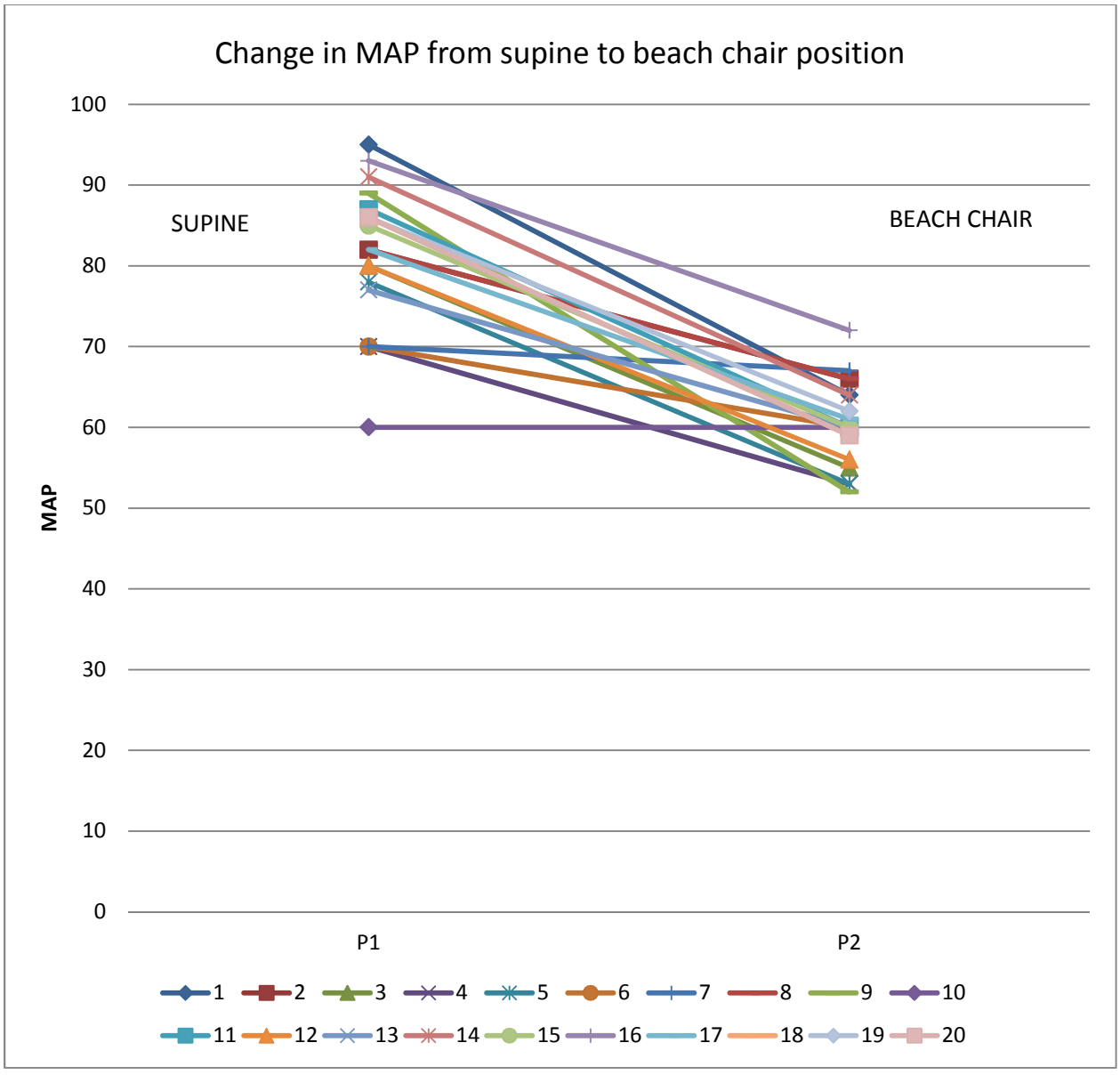


Figure 14: Line graph to show the pattern of mean arterial pressure for individual patients

2: CHANGE IN MIDDLE CEREBRAL ARTERY BLOOD FLOW VELOCITY (V_{MCA}) AFTER POSITION CHANGE (FROM SUPINE TO BEACH CHAIR)

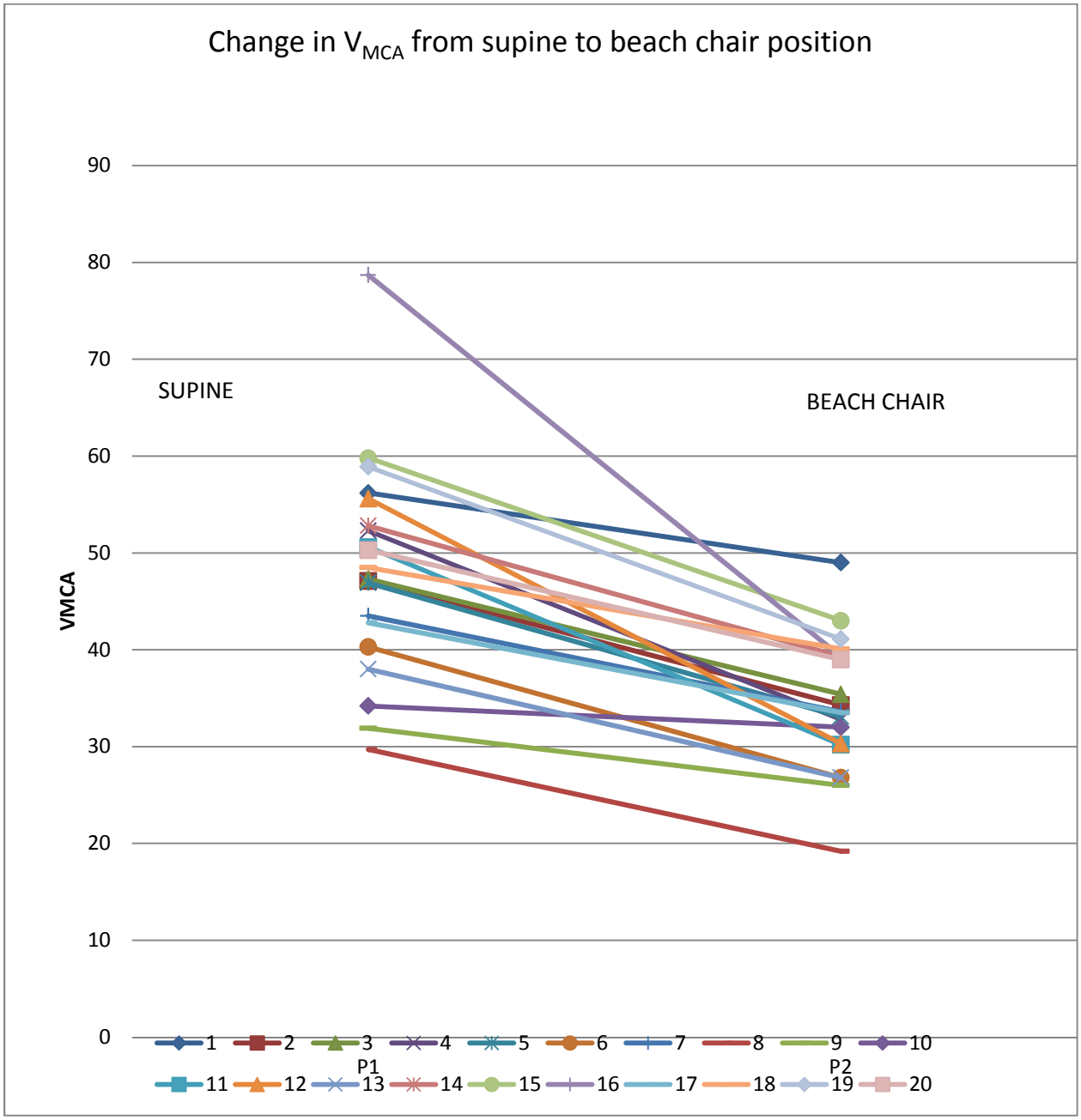


Figure 15: Line graph to show the pattern of middle cerebral artery blood flow velocity for individual patients

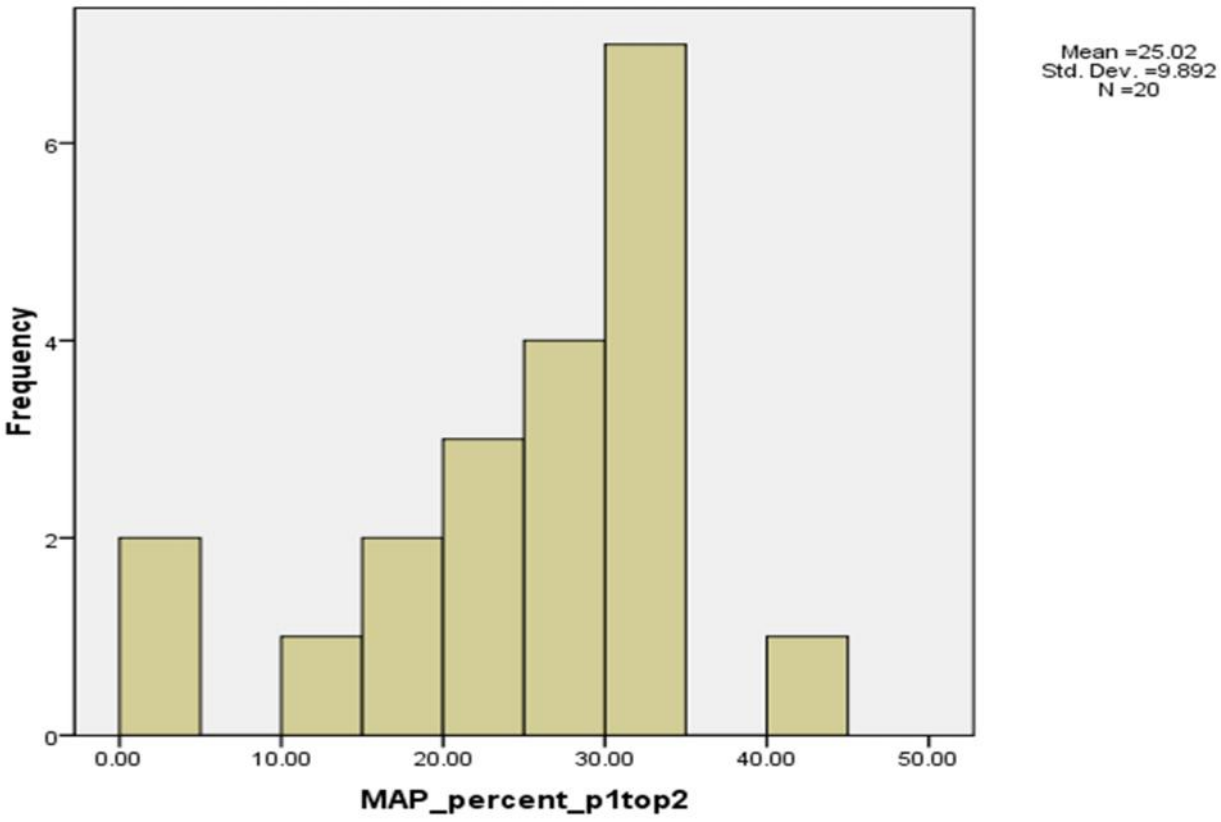


Figure 16: Distribution of MAP percentage reduction from baseline.

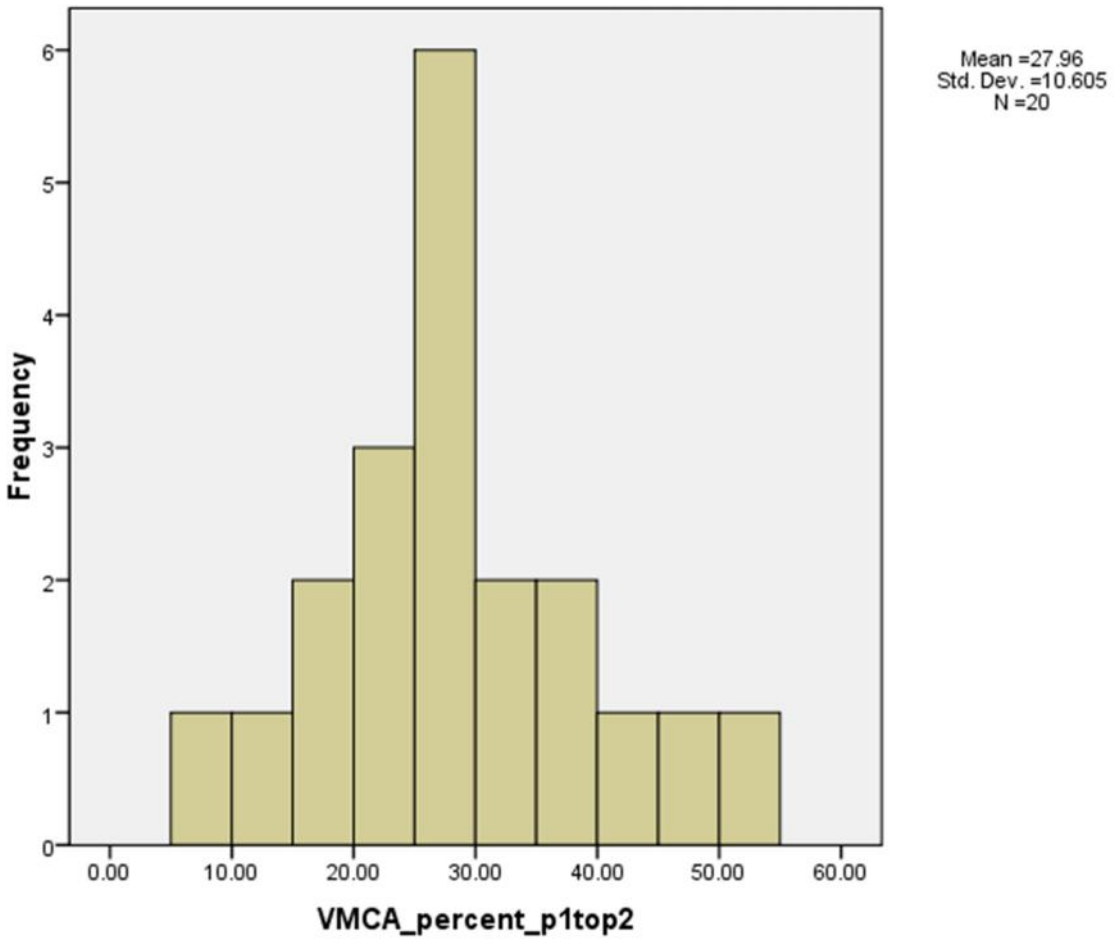


Fig 17: Distribution of VMCA percentage reduction from Baseline.

The percentage reduction of V_{MCA} ranges from 6.43% to 50.06% with the mean value of 50.06% standard deviation of 10.6%.

Variable	**Mean**	**SD**	**Min**	**Max**
MAP percentage reduction	25.02	9.89	0	41.6
V_{MCA} percentage reduction	27.95	10.60	6.43	50.06

Table2: Descriptive statistics of percentage reduction in MAP and V_{MCA} from

The percentage reduction in the mean arterial pressure ranges from 0 to 41.6% with the mean value of 25.02%. For one patient other than the initial drop in the blood pressure as soon as change in the position there was no further hypotension at all but still he had a minor drop in $V_{MCA.}$

The paired samples t test was performed to test the significance of difference between supine and beach chair positions and found it was statistically significant ($p < 0.001$) for both mean arterial pressure (MAP) and middle cerebral artery blood flow velocity (VMCA)

COMPARING THE CHANGES IN MEAN ARTERIAL PRESSURE AND THE MCA BLOOD FLOW VELOCITY

The correlation between the MAP change from baseline and VMCA change from the baseline is performed using Pearson's Correlation Coefficient and it is found that there is no linear correlation. But they are in quadratic relationship. The correlation if presented graphically as a scatter plot in Figure 18.

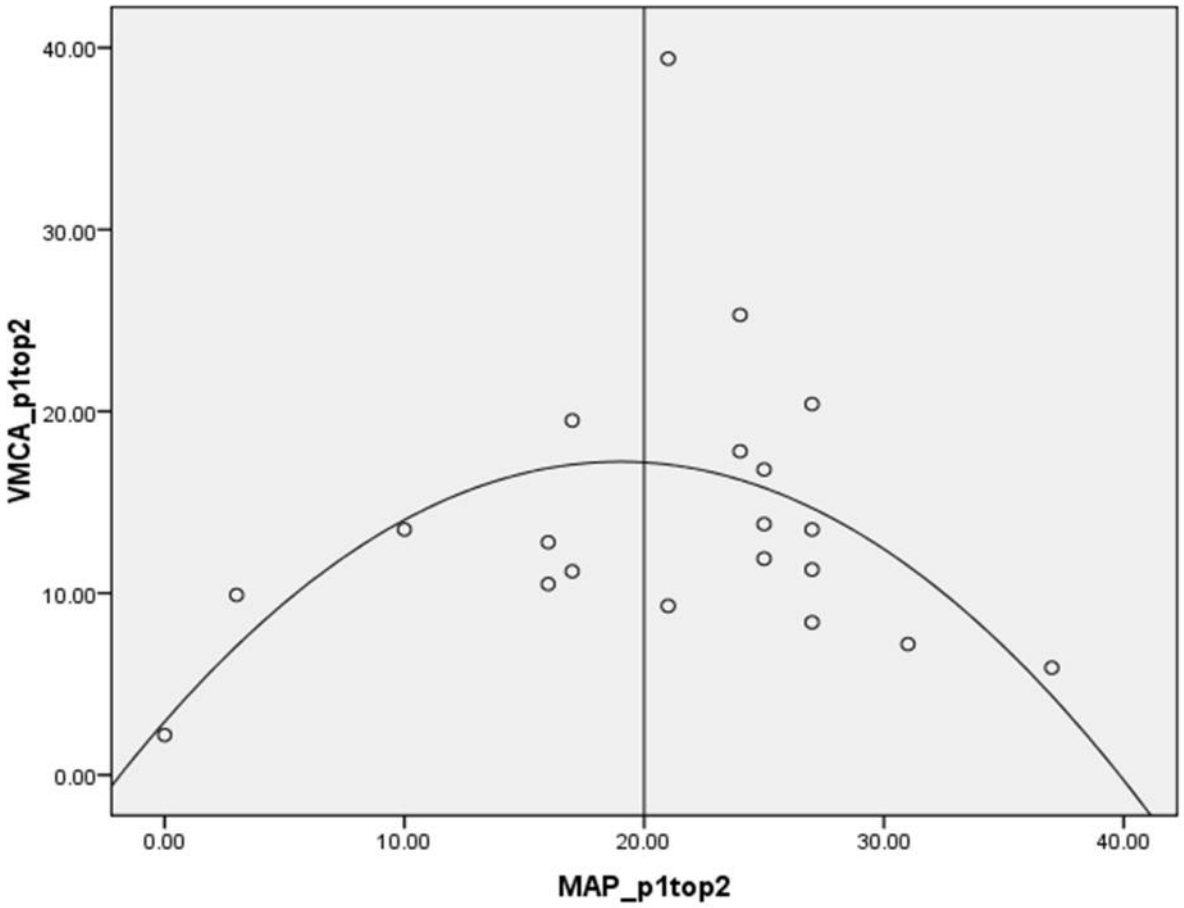


Figure 18: Plot of MAP change and VMCA change showing quadratic correlation .

CHANGES IN DIFFERENT ASA GROUPS

ASA	Median	IQR	P value
1	27.9070	20.79 – 31.32	0.842
2	29.4118	14.28 – 41.57	

Table 3: MAP percentage reduction among different ASA groups

The percentage reduction in MAP between different ASA group was analyzed. There is no significant difference in the fall in blood pressure between ASA I and ASA II patients.

ASA	Median	IQR	Pvalue
1	27.1762	22.09 – 36.31	0.921
2	28.0936	18.49 – 33.49	

Table 4: VMCA percentage reduction among different ASA groups.

There is no significant difference noted between ASA I and ASAII patients in V_{MCA}.

SR. NO.	AGE	BASELINE MAP	HYPOTENSION MAP	TREATED MAP
1	20	95	64	75
2	65	82	66	77
3	18	70	53	65
4	24	78	53	65
5	60	82	66	75
6	55	87	60	76
7	55	91	64	84
8	34	85	60	70
9	23	93	62	72
10	33	82	61	73
11	28	86	59	70
12	45	86	62	73
13	29	80	59	76

Table 5: Distribution of mean arterial pressure (MAP) at various point of time

The above table shows the mean arterial pressure values in supine, during hypotension in sitting position and after treatment with ephedrine or phenylephrine bolus.

Out of the 20 patients, the six patients who were overzealously treated during hypotension were excluded along with one patient who had no hypotension . Among the thirteen patients the MAP requirement was an average of 73.15mmHg (with a minimum of 65 mmHg and a maximum of 84mmHg) to keep the middle cerebral artery blood flow velocity around baseline. Hence can maintain adequate cerebral perfusion above 73.15mmHg.

Distribution among age groups

Serial No.	Age	Percentage reduction of MAP	Percentage reduction of V_{MCA}	MAP(mmHg) to maintain normal V_{MCA}
1	65	19.51	27.17	77
2	52	14.28	33.49	75
3	60	19.51	35.35	75
4	55	31.03	40.31	76
5	55	29.67	25.56	84

Table 6: Percentage reduction of MAP and V_{MCA} in older age group

Average reduction of MAP is 22.8%; average reduction of V_{MCA} is 32.37%. average MAP = 77.4 mmHg

MAP: mean arterial pressure; V_{MCA} : middle cerebral artery blood flow velocity

Serial No.	Age	Percentage reduction of MAP	Percentage reduction of V_{MCA}
1	20	32.63	12.81
2	28	31.25	25.15
3	18	24.28	37.28
4	24	32.05	29.42
5	37	4.28	22.75
6	37	41.57	18.49
7	40	0	6.43
8	30	30	45.50
9	39	22.07	29.47
10	34	29.41	28.09
11	23	22.58	50.06
12	33	25.60	21.72
13	28	31.39	17.31
14	45	27.90	30.22
15	29	26.25	22.46

Table 7: Percentage reduction of MAP and V_{MCA} in younger age group

Average reduction of MAP is 23.78%; average reduction of V_{MCA} is 26.477%

MAP: mean arterial pressure; V_{MCA} : middle cerebral artery blood flow velocity

DISCUSSION

Discussion:

This observational study was aimed at assessing the correlation between the reduction in mean arterial pressure and cerebral blood flow in patients undergoing shoulder surgery in beach-chair position.

22 patients were enrolled in the study. Out of which 2 patients were excluded from the study due to poor trans-temporal Doppler window and wide swing in blood pressure recordings.

Mc Culloch et al(29) did a similar observational study in 19 patients .All the patients were more than 55 years of age and known cases of cardiovascular or cerebrovascular disease. All patients were given interscalene block for analgesia and standard intravenous induction and intubation was done. Anesthesia was maintained with desflurane. They did controlled hypotension with remifentanil and phenylephrine infusion and monitored NIBP and invasive blood pressure and continuous middle cerebral artery blood flow velocity by transcranial Doppler.

In this study. they kept systolic blood pressure (NIBP) at around 90mmHg for most of their patients. It was demonstrated that a 47+/ -7% decrease in mean arterial pressure was accompanied by 22 +/- 7% decrease in middle cerebral artery blood flow velocity.

In our study we included patients from age of 18 to 65 years, with a mean age of 38(standard deviation 14). 25% of the patients were more than 45 years and 75% of them were less than 45 years. None of our patients were given regional anesthesia. We gave general anesthesia using standard intravenous induction and intubation. Anesthesia was maintained with isoflurane (miminum alveolar concentration: 0.8 – 0.9) and 0.1 to 0.15mg /kg morphine was given for analgesia. We monitored invasive blood pressure and measured middle cerebral artery blood flow velocity by transcranial Doppler at various points namely before induction; after intubation in supine; then during hypotension in sitting position and after the hypotension was treated. We considered a 20% reduction in mean arterial pressure in order to treat it. In elderly patients we treated if mean arterial pressure was less than 70mmHg.

We noticed that the percentage reduction of mean arterial pressure varied from 0 to a maximum of 41.6%. The mean percentage reduction in mean arterial pressure was 25.02%.

The mean arterial pressure change was accompanied by reduction in middle cerebral artery blood flow velocity measured by transcranial Doppler

We measured middle cerebral artery blood flow velocity whenever there was hypotension in sitting position. We found that during significant reduction in mean

arterial pressure, middle cerebral artery blood flow velocity reduced by an average of 27.95%. The minimum drop was about 6.43% and a maximum of 50%.

It was found that one of our patients had very stable blood pressure throughout the procedure but had a minor drop in middle cerebral artery blood flow velocity.

We performed paired sample t test to test the significance of difference of mean arterial pressure and middle cerebral artery blood flow velocity in supine and beach-chair positions. It was statistically significant with $p < 0.001$.

It was also one of the objectives to analyze the correlation between the percentage reduction of mean arterial pressure and middle cerebral artery blood flow velocity. We used Pearson's correlation coefficient and found that there is no linear correlation between the two variables.

Mean arterial pressure and middle cerebral artery blood flow velocity reduction are in quadratic relationship with statistically significant constants. Quadratic linear regression analysis were used to predict the correlation between middle cerebral artery blood flow velocity reduction and mean arterial pressure reduction.

The equation we derived is as follows,

$$Vmca = a + (b * MAP) + (c * MAP^2)$$

Where a, b and c are constants and their values were found to be as, a = 2.934; b = 1.507; c = -0.04

V_{MCA}: Percentage reduction of Middle cerebral artery blood flow velocity

MAP: Percentage reduction of Mean arterial pressure

To explain, there was a direct linear relationship between the percentage reduction of middle cerebral artery blood flow velocity and mean arterial pressure till 20%, following which they did not correlate directly.

While doing the study, we over-treated six patients' blood pressure beyond the baseline pressure, so we were not able to determine the lower limit of mean arterial pressure required to maintain cerebral blood flow near normal in those patients. Hence we excluded those values in our analysis. In the rest of the patients, in order to maintain a near normal middle cerebral artery blood flow velocity the average required mean arterial pressure was 73.1mmHg. This has inter-individual variation with a minimum value of 64mmHg and maximum value of 84mmHg.

We found variations in different age group also. We had 25% patients above 45 years and 75% were below 45 years. In patients above the age of 45 the average percentage reduction was 22.8% which was accompanied by a 32.38% reduction in middle cerebral artery blood flow velocity in sitting position.

Whereas, in the younger age group, the percentage reduction in mean arterial pressure was 23.78%, with a 26.47% reduction in middle cerebral artery blood flow velocity in sitting position.

We can say that, though the drop in the mean arterial pressure was similar in both the age groups, the fall in the cerebral blood flow was much larger in the older age group who would require a higher mean arterial pressure to maintain cerebral perfusion. The calculated average value of mean arterial pressure to maintain cerebral blood flow near the baseline was 77.4mmHg in the older age group whereas the average mean arterial pressure in all patients was 73.15mmHg.

In the study by McCulloch et all (29) all patients were known cases of cardiovascular disease or cerebrovascular disease but in our study we included ASAI and II patients. 15 % of our patients were ASA risk II (diabetes in two patients and one was an asthmatic) and 85% patients were ASA I.

We found that there was no significant difference among the two ASA groups. None of our patients were hypertensives also. If we could have done the study in patients with higher ASA risk we might have obtained a significant difference among the ASA groups.

None of our patients had neurological disability in the postoperative period and complications related to the arterial cannulation.

CONCLUSION AND LIMITATION

Conclusion:

This observational study in patients coming for shoulder surgery in sitting position showed correlation between mean arterial pressure and middle cerebral artery blood flow velocity

Mean arterial pressure can be taken as a surrogate, especially in sitting position, for cerebral perfusion thus necessitating active treatment in the event of hypotension.

We conclude that an average of 73.15mmHg is the minimum mean arterial pressure required to maintain cerebral perfusion in beach-chair position. In the older age group it is better to maintain the mean arterial pressure above 77.4mmHg.

Limitations

1. Although the calculated sample size was 45, we were able to recruit only 20 patients. Hence the power of the study is reduced. The number of patients above the age of 45 was only 5.

2. We could not do a continuous transcranial Doppler monitoring.

3. In six cases hypotension was treated overzealously.

BIBLIOGRAPHY

1. Marilyn J. Cipola. The Cerebral Circulation. Morgan and Claypool Life Sciences;

2. Kim. E. Barrett, Susan M. Barman, Scott Boitano, Heddwen Brooks. Ganong's Review of Medical Physiology. 24th ed. LANGE;

3. James E. Cottrell, William L. Young. Cottrell and Young's Nueroanesthesia. 5th ed. MOSBY ELSEVIER;

4. Richard D. Hoge, Jeff Atkinson, Brad Gill, Gérard R. Crelier, Sean Marrett, and, G. Bruce Pike. Linear coupling between cerebral blood flow and oxygen consumption in activated human cortex. PNAS. 1999 Jun 14;96(16):9403–8.

5. Richard B Buxton and Lawrence R Frank. A Model for the Coupling Between Cerebral Blood Flow and Oxygen Metabolism During Neural Stimulation. J Cereb BLOOD FLOW Metab. 1996 Jul 8;17:64–72.

6. Ronald D. Miller. Miller's Anesthesia. seventh edition. ELSEVIER; 305-340 p.

7. Alifia Tameem, MBBS MD FRCA, Hari Krovvidi, MD FRCA Specialist Registrar, Queen Elizabeth Hospital Birmingham, Edgebaston, Birmingham, West Midlands, B15 2TH, et al. Cerebral physiology. BJA [Internet]. 2013 Mar 1; Available from: http://ceaccp.oxfordjournals.org/content/early/2013/02/28/bjaceaccp.mkt001.full

8. Paul G. Barash, Bruce F. Cullen, Robert K. Stoelting, Michael K. Cahalan, M. Christine Stock. Clinical Anesthesia. 6th edition. Wolters Kluwer/ Lippincott Williams & Wilkins;

9. S. Strandgaard, J. Olesen, E. Skinhøj, N. A. Lassen. Autoregulation of Brain Circulation in Severe Arterial Hypertension. Br Med J. 1973 Mar 3;1.

10. L WOLLNER, S T McCARTHY, N D W SOPER, D J MACY. Failure of cerebral autoregulation as a cause of brain dysfuction in the elderly. Br Med J. 1979: 1.

11. Hiroaki Naritomi, MD; John Stirling Meyer, MD; Fumihiko Sakai, MD; Fumie Yamaguchi, MD; Terry Shaw, PhD. Effects of Advancing Age on

Regional Cerebral Blood FlowStudies in Normal Subjects and Subjects With Risk Factors for Atherothrombotic Stroke. JAMA Neurol. 36(7).

12. Wagner M, Jurcoane A, Volz S, Magerkurth J, Zanella FE, Neumann-Haefelin T, et al. Age-related changes of cerebral autoregulation: new insights with quantitative T2'-mapping and pulsed arterial spin-labeling MR imaging. AJNR Am J Neuroradiol. 2012 Dec;33(11):2081–7.

13. Kewal K. Jain. THE HANDBOOK OF NEUROANESTHESIA. New York: Humana press;

14. Leroy C. Abbott ; John B. deC. M. Saunders ; Helen Hagey ; Ellis W. Jones Jr. SURGICAL APPROACHES TO THE SHOULDER JOINT. J Bone Jt Surg. 1949 Apr;31(2):235–55.

15. Xinning Li, MD; Josef K. Eichinger, MD; Timothy Hartshorn, MD; Hanbing Zhou, MD; Elizabeth G. Matzkin, MD; Jon P. Warner, MD. A Comparison of the Lateral Decubitus and Beach-chair Positions for Shoulder Surgery: Advantages and Complications. J Am Acad Orthop Surg. 2015 Jan 1;

16. Nicolas Bonnaig, MD, Steven Dailey, MD, and Michael Archdeacon, MD, MSE. PROPER PATIENT POSITIONING AND COMPLICATION PREVENTION IN ORTHOPAEDIC SURGERY. J Bone Jt Surg. 2014 Jul 2;96 A(13):1135–40.

17. Christopher J. Winfree, MD, David G. Kline, MD,. Surgical Neurology Intraoperative positioning injuries. ELSEVIER. 2005 Jan;63(1):5–16.

18. Sarah Gerken, MD.,. Preventing positioning injuries: An Anesthesiologist's perspectives.

19. Christina L. Beecroft, FRCA, FDS RCS, David M. Coventry, FRCA. Anaesthesia for shoulder surgery. Oxf J. CEACC pain(2008) 8(6):193–8.

20. Andre P. Boezart. Anesthesia and Orthopaedic Surgery. Mc Graw Hill Medical; 81-90 p.

21. Albert K. H. Ng, Richard S. Page. Greater aricular nerve neuropraxia with beach chair position during shoulder surgery.

22. Pohl A, Cullen DJ. Cerebral ischemia during shoulder surgery in the upright position: a case series. J Clin Anesth. 2005 Sep;17(6):463–9.

23. Darren J. Friedman, MD; Nata Z. Parnes, MD; Zachary Zimmer; Laurence D. Higgins, MD; Jon J.P. Warner, MD. Prevalence of Cerebrovascular Events During Shoulder Surgery and Association With Patient Position. Helio Orthop. 32(4).

24. L. K. Moppett, R. P. Mahajan. Transcranial doppler ultrasonography in anaesthesia and intensive care.

25. Nicoletto, Heather A; Burkman, Marilyn H. Transcranial Doppler Series Part II: Performing a Transcranial Doppler. Am J Electroneurodiagnostic Technol. 2009 Mar 1;49(1):14–27.

26. C C Bishop, S Powell, D Rutt and, N L Browse. Transcranial Doppler measurement of middle cerebral artery blood flow velocity: a validation study.

27. F.J. Kirkham†, T.S. Padayachee‡, S. Parsons§, L.S. Seargeant†, F.R. House|,, R.G. Gosling‡. Transcranial measurement of blood velocities in the basal cerebral arteries using pulsed Doppler ultrasound: Velocity as an index of flow.

28. Bathala L, Mehndiratta MM, Sharma VK. Transcranial doppler: Technique and common findings (Part 1). Ann Indian Acad Neurol. 2013;16(2):174–9.

29. McCulloch TJ, Liyanagama K, Petchell J. Relative hypotension in the beach-chair position: effects on middle cerebral artery blood velocity. Anaesth Intensive Care. 2010 May;38(3):486–91.

ANNEXURES

PROFORMA

DATA COLLECTION SHEET

SERIAL NO: DATE:

HOSPITAL ID: AGE: SEX:

DIAGNOSIS:

PROPOSED SURGERY:

ASA:

COMORBIDITIES MEDICATIONS

TIME	HR Beats/min	MAP mmHg	SPO_2 %	V_{MCA}	$ETCO_2$ mmHg	VASOPRESSOR USED
PREINDUCTION(T_0)						
POST INTUBATION(T_1)						
AFTER SITTING POSITION(T_2)						
T_3						
T_4						
T_5						
T_6						
T_8						
T_9						
T_{10}						

Informed Consent Form for Subjects

Informed Consent form to participate in a research study

Study Title:

Study Number: _____

Subject's Initials: _____ Subject's Name: _____

Date of Birth / Age: _____

(Subject)

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions.

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published.

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

(v) I agree to take part in the above study.

(vi) I am aware of the Audio-visual recording of the Informed Consent.

(Click here for Audio Visual guidelines)

Signature (or Thumb impression) of the Subject/Legally Acceptable

Date: _____/_____/_____

INFORMATION SHEET

Research Title: **The effect of beach chair position on cerebral blood flow in patients undergoing shoulder surgery**

Information sheet

Introduction

I am Dr. Jesudoss A, M.D post graduate with the Department of Anaesthesiology and I have ten months of experience in the department. I am doing a research on measurement of blood flow to brain in patients undergoing shoulder surgery in sitting position (beach chair position). I am going to give information and invite you to be part of this research. You do not have to decide today whether or not you will participate in the research. Before you decide, you can talk to anyone you feel comfortable with about the research.

There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have questions later, you can ask me or the anaesthetist on the day of surgery.

Purpose of the research: For patients who are planned for shoulder surgery, sitting position (or beach chair position) is chosen because of better visualization of surgical site. But this position can cause decrease in pressure in the blood vessels to brain and also blood flow to brain. If there occurs a significant reduction in blood flow brain it will affect the brain function. So we will treat blood pressure to bring back to normal. In order to prevent this I am going to do a Doppler scan (an ordinary ultrasound scan) of your head and also measure blood pressure by radial artery (blood vessel in the wrist) cannulation.

Participant selection: You have been invited to participate because you have surgery in your shoulder in sitting position.

Voluntary participation: your participation in this research is entirely voluntary. Whether you choose to participate or not, the management of anaesthesia and your safe recovery will not change. If you choose not to participate in this research project, you will be offered the same anaesthesia routinely given in this hospital for shoulder surgery.

Information on the procedures:

1. You will be made to lie down straight on the operation table. A needle will be inserted into your vein for giving fluids and drugs. Also, a needle will be inserted into your blood vessel in the wrist to measure blood pressure before giving any injection to induce sleep. But this procedure will be done under local

anaesthesia i.e. by injecting a small amount of lignocaine in the skin to numb it. This might cause a little discomfort to you.

2. Then in the same position we will scan your head using Doppler scan (ultra sound).
3. Then anaesthesia will be given as per the standard protocol followed in this hospital. Surgery will be done in sitting position and if required the Doppler scan will be repeated during surgery also.

Side effects: commonly hematoma (collection of blood) and bleeding can happen due to the injection put in your blood vessel at the wrist. Temporary spasm of the artery, catheter-site- infection, ischemic damage, psuedoaneurysm formation are the other rare complications. We will take all precautions necessary to keep you safe.

Benefits: while measuring blood flow to your brain during the procedure, if there is any significant reduction we can treat immediately.

Reimbursements: Your will not be charged for the Doppler scan or for the injection that we put in your artery to measure blood pressure.

Confidentiality: Your name will not be mentioned anywhere neither the data sheet nor the final published study. Your data will bear a study number and the number will be used till analysis. The master sheet will have your study number.

Sharing the result: The result of this research is a property of Christian medical college and I'm entitled to publish it in a journal or present it in a conference.

Right to refuse or withdraw: You do not have to take part in this research if you do not wish to do so. You may also withdraw participating in this research even inside the operating room. It is your choice and all of your rights will be respected.

This proposal has been reviewed and approved by IRB, Christian Medical College, which is a committee whose task is to make sure that research participants are protected from harm. If you wish you can find more about the IRB.

Contact

1. Dr. Jesudoss A (Primary investigator)

PG registrar,

Department of Anaesthesiology,

Christian Medical College, Vellore.

Email: jesudoss@cmcvellore.ac.in

Mobile: 9843391144

Office: 0416 2282105

2. Dr. Melvin Alex Abraham (Co-investigator)
Department of Anaesthesiology, CMC Vellore.

Phone: +91 9894540049

Email ID: melvinalexabraham@gmail.com

3. Research office,

II floor, Carman block,

Christian medical college,

Bagayam, Vellore 632 002.

Email: research@cmcvellore.ac.in

Telephone: 04162284294

It has also been reviewed by the Ethics Review Committee CMC Vellore, which is supporting the study.

DATASHEET

S.NO	HOSPITAL NO.	AGE	SEX	P1_TIME	P1_MAP	P1_VMCA	P2_TIME	P2_MAP	P2_VMCA	P3_TIME
1	257050B	20	M	7:42	95	56.2	8:11	64	49	8:15
2	301722G	65	M	7:52	82	47.1	7:59	66	34.3	8:03
3	229545G	28	M	11:44	80	47.3	11:59	55	35.4	12:03
4	256516G	18	M	7:45	70	52.3	8:05	53	32.8	8:08
5	257520G	24	M	7:45	78	46.9	8:11	53	33.1	8:15
6	351721F	52	M	16:10	70	40.3	16:33	60	26.8	16:35
7	213736G	37	F	10:41	70	43.5	10:58	67	33.6	11:02
8	623676A	60	M	7:45	82	29.7	8:40	66	19.2	8:45
9	211054G	37	M	9:55	89	31.9	10:45	52	26	10:50
10	202629G	40	M	7:35	60	34.2	8:37	60	32	8:40
11	344311G	55	M	7:32	87	50.6	7:57	60	30.2	8:00
12	872560F	30	M	9:20	80	55.6	10:12	56	30.3	10:15
13	952972B	39	M	10:32	77	38	11:30	60	26.8	11:34
14	178569G	55	M	12:20	91	52.8	13:03	64	39.3	13:10
15	370908G	34	M	10:10	85	59.8	11:05	60	43	11:09
16	307808G	23	M	9:13	93	78.7	9:45	72	39.3	9:58
17	417998G	33	M	11:05	82	42.8	11:56	61	33.5	12:02
18	447661G	28	M	7:39	86	48.5	8:59	59	40.1	9:06
19	150762D	45	M	13:39	86	58.9	14:58	62	41.1	15:02
20	322477D	29	M	7:35	80	50.3	8:50	59	39	8:55

P3_MAP	P3_VMCA	P1TOP3MAP	P1TOP2MAP	P1TOP2VMCA	HOSPITAL NO	SR. NO.
75	53.9	21.05263158	32.63157895	12.8113879	257050B	1
77	48.3	6.097560976	19.51219512	27.17622081	301722G	2
86	46.3	-7.5	31.25	25.15856237	229545G	3
65	47.6	7.142857143	24.28571429	37.28489484	256516G	4
65	37.9	16.66666667	32.05128205	29.42430704	257520G	5
75	34.5	-7.142857143	14.28571429	33.49875931	351721F	6
78	39.1	-11.42857143	4.285714286	22.75862069	213736G	7
75	22.5	8.536585366	19.51219512	35.35353535	623672A	8
109	39	-22.47191011	41.57303371	18.49529781	211054G	9
69	37.5	-15	0	6.432748538	202629G	10
76	53.4	12.64367816	31.03448276	40.31620553	344311G	11
80	47.5	0	30	45.50359712	872560F	12
83	39.4	-7.792207792	22.07792208	29.47368421	952972B	13
84	44.1	7.692307692	29.67032967	25.56818182	178569G	14
70	56.4	17.64705882	29.41176471	28.09364548	370908G	15
62	60.4	33.33333333	22.58064516	50.0635324	307808G	16
73	40.5	10.97560976	25.6097561	21.72897196	417998G	17
70	45.6	18.60465116	31.39534884	17.31958763	447661G	18
73	55.6	15.11627907	27.90697674	30.22071307	150762D	19
76	56.4	5	26.25	22.46520875	322477D	20