http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v10i5.20

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Retrospective Evaluation of Anaesthesia Management in Surgical Procedures for Moyamoya Disease - A Single Institute Experience

Authors
Dr Pratima Kothare[1], Dr Roshni Cheema[2]
[1]MD, DNB Anaesthesiology, Consultant Anaesthesiologist, BHIMS & MRC, Mumbai, India
MBBS, Seth GSMC/KEMH, Mumbai, India
[2]MD Anaesthesiology Resident, BHIMS, Mumbai, India

Abstract

Background: *Moyamoya disease (MMD) is a progressive cerebrovascular disease with stenosis of intracranial internal carotid artery and its proximal branches leading to the gradual development of a collateral circulation to compensate for the blockage. This neovasculature is weak and prone to bleeding and aneurysms. Since we are dealing with a fragile cerebral vasculature on one hand and stenosed vessels on the other, anaesthesia management is challenging.*

Aim: *Aim of this case series was to formulate an ideal plan for the anesthesia management of patients undergoing surgery for Moyamoya disease by understanding the perioperative factors so as to deliver safe and hemodynamically stable anesthesia with minimal effect on perioperative oxygenation and blood flow leading to reduction in neurological complications, length of ICU and hospital stay.*

Material and Method: *Retrospective evaluation of 25 pediatric patients for surgery for MMD from Feb 2005 to June 2021 was undertaken. Direct vascularization was performed in 3 patients (12%) and 22 (88%) underwent Indirect vascularization procedures. Anaesthesia protocol designed was applied to all the patients. Data related to anaesthesia monitoring and outcomes was charted to assess the efficiency of anaesthesia plan.*

Observations and Results: *A total of 25 pediatric patient records were evaluated. Ischemic presentations were 80% and Haemorrhagic presentations were 20%. Endtidal CO2 was maintained at 35 to 40 mmHg, There was no hypocapnia or hypercapnia and oxygen saturations were maintained at 100%. Intraoperatively two patients n= 2(8%) had hypertension and tachycardia at the time of scalp incision. 4 patients n= 4(16 %) had hypotension after induction of anaesthesia . None of the patients had low urine output, n= 0(0%) and no patient developed hypothermia. Two patients required blood transfusion, n=2 (8%). Postoperatively haemoglobin dropped to 10 ± 0.52, seizures were seen in 4 patients (16%), one child developed infarction n=1 (4%) and none had haemorrhage or hematoma (0%). It was thus observed that with vigilant and targeted perioperative protocol, ICU stay was just 2 days (1.84 ±0.39) and discharge from hospital was within 11 days (10.34±1.039) without any new neurological deficits with the exception of one patient who had an unfortunate event of infarction, n=1 (4%) requiring emergency decompression surgery.*

Conclusion: *Cerebral haemodynamics is at the core and an extremely crucial determinant of success rate in revascular- ization procedures used in MMD. Perioperative anesthesia care has a direct effect on these hemodynamic parameters. We designed and titrated anaesthetics for achieving and maintaining normotension, normo- capnia, normovolemia and normothermia. This was to ensure that we maintain adequate Cerebral Blood Flow (CBF) and Cerebral Perfusion Pressure (CPP) and Cerebral Metabolic Rate of oxygen consumption (CmRO2), thereby avoiding perioperative cerebral ischemic consequences and neurological worsening, and thus minimising the ICU/Hospital stay.*

Keywords: *MMD, revascularisation surgery, anaesthesia risk factors, length of hospital stay.*

Background

Moyamoya is a Japanese word which means something hazy like a puff of cigarette smoke drifting in the air, as can be seen in figure 1 and was first described by Takeuchi and Shimuzui in 1957[1, 2]. It is defined as a cerebral arteriopathy with occlusion of supraclinoid terminal Internal Carotid Arteries (ICA) or proximal Middle Cerebral Arteries (MCA) causing constriction mainly at the ICA and often extending to the MCA and Anterior cerebral artery (ACA). When ICA gets completely blocked patients survive on the collateral circulation from the posterior circle of Willis arising from the basilar artery, but this neovasculature is fragile and prone to bleeding and aneurysms. On angiography these new collateral vessels are hazy and have a reticulate appearance resembling a puff of smoke.

Moyamoya disease has an Asian predominance, is an autosomal dominant hereditary disorder with a mutation of q25.3 on chromosome 17 with an incidence of 0.35 cases per 100,000 people, and a female to male ratio of 1.8:1[3].

Moyamoya Syndrome is when it occurs in association with other disorders like congenital heart disease, Downs syndrome, Neurofibromatosis type 1 or Sickle cell anaemia among many others. It can be acquired post irradiation or post infections[4]. Microscopically, there is a fibrocellular thickening of the intima, irregular induration of the internal elastic laminae and attenuation of the media. No inflammatory infiltrates are seen, however there is a role of auto immune complexes in developing this pathology.

Age at presentation can be bimodal, pediatric patients present with ischemic symptoms and adults in 4th decade mainly have hemorrhagic presentation. Clinically MMD can thus present as Transient Ischemic Attacks (TIA) that reverse within 24 hours without any deficits or stroke - ischemic or hemorrhagic with chief complaints being headache, seizures, weakness, monoparesis or hemiparesis, visual impairment or a combination of any or all[5]. Besides CT/MRI, a six vessel DSA remains the gold standard for diagnosis and also to see the integrity of vessels for bypass surgery. Treatment options suggested are either conservative or surgical revasularisation.

Conservative treatment suggested is antiplatelet therapy for preventing stroke in MMD patients, however evidence for the same is lacking. The TIA episode is mainly due to haemodynamic changes rather than platelet adhesions or embolic events, therefore the benefit of antiplatelet therapy remains controversial in the treatment of MMD[6,7,8].

Revascularisation surgery either direct or indirect is indicated in cases of refractory symptoms due to TIA or stroke. Treatment aims at revascularization to improve cerebral blood flow and prevention of further ischemic and haemorrhagic episodes[9,10].

Understanding the disease and surgical procedures gives the anesthesiologist a perspective in managing these patients perioperatively. Proper preoperative evaluation will help to achieve good results[11].

Material and Methods

The aim of presenting this case series was to evaluate the perioperative findings based on the best anesthetic technique evolved in our institute to maintain the physiological requirements for adequate cerebral circulation and oxygenation during the course of revascularization. Only those patients whose complete data record was available were included in the series. Statistical analysis was done and data is presented as percentage (%), Mean +/- SD or Median (range).

Clinical presentations, surgical procedure conducted, intraoperative course, postoperative outcome, ICU stay and discharge from hospital with or without neurological deterioration was charted for each patient as seen in table 1. Retrospective data of 25 children with MMD (n=25), age ranging between 1 to 16 years for revascularization surgery between 2005 to 2021 was evaluated.

Table 1: Perioperative data analysis

Preoperative Data	Intraoperative Data	Monitoring	Intraoperative Complications	Postoperative Parameters
Age	Anaesthesia technique	ECG	Brady/Tachycardia	Extubated/Ventilated
Weight	Fluids management	NIBP/IBP	Hypo/Hypertension	Seizures
Gender	Blood transfusion	Pulse Oximetry	Hypo/Hyperthermia	Other Neuro. complications
Preexisting complaints	Urine output	EtCO2	Blood Loss	Duration of ICU stay
Unilateral Disease	Mean BP	Rectal temperature	Low urine output	Discharge from hospital
Bilateral Disease	EtCO2		Bronchospasm	Neurological status on discharge
	Temperature			

Methodology

A thorough preanesthetic check up was done for all 25 patients. Parental counseling of the pediatric patients was done and an informed anesthesia consent was obtained. Our anesthesia team was in constant consultation with the surgical team about their ongoing requirements for a safe outcome. This is an overview of our institutional anesthesia management for MMD revascularisation surgeries.

Prilocaine patch applied at iv site half an hour prior in wards, in the presence of parents. No sedation or premedication given in wards. Preoxygenation done for 5 minutes. Two peripheral veins secured. Arterial line secured after induction. Adjuvant drugs used were Fentanyl (1mcg/kg), Glycopyrrolate (0.004 mg/kg), Dexamethasone and antibiotics followed by induction with Thiopentone (4mg/kg), Vecuronium (0.08 mg/kg). Endotracheal tube secured after intubation and ensured bilateral air entry. Anesthetic plane maintained with O2 : N2O (1:1) and Sevoflurane (MAC 0.8 to 1) with intermittent Vecuronium top ups. Tidal volume of 10-12 ml/kg and respiratory rate of 12 to 16, to maintain an End Tidal CO2 of 35 to 40 mmHg. Oxygen saturations maintained at 100% at all times.

Monitoring: Standard (ECG, pulse oximetry, NIBP), invasive BP monitoring in all cases, right Internal jugular vein (IJV) central venous access secured in STA-MCA bypass cases only, urine catheter for urine output monitoring, rectal temperature probe, warming blanket and warm fluids infused to maintain core temperature at 36.6 to 37.2 degrees celcius. IV Fluids - crystalloids 4 to 6 ml/kg/hour to maintain a urine output at 2ml/kg/hour. Reversal was standard with measures to avoid any sudden tachycardia/ hypertension during extubation. Pain relief with intravenous Paracetamol and Diclofenac suppository.

Surface markings, as can be seen in figure 3 were done with the use of a doppler (figure 2) by the neurosurgeon to map the course of superficial temporal artery in supine position on a horse shoe rest. No lignocaine infiltration over incision, before and after surgery, as per neurosurgical protocol to avoid spasm of Superficial temporal artery (figure 4,5).

Results and Observations

Table 1: Patient Data Details

Data is presented as number (%) or Mean +/- SD or Median (range).

Sr. No.	Variable	
1	Number of Patients (n)	25
2	Gender - Male : Female	13:12
	Age group: 1-16 yrs	n = 25 (100%)
3	1-5 yrs 6-10 yrs	n = 12 (48%) n = 8 (32%)
	10-16 yrs	n = 5 (20%)
	Weight(kg)	
4	1-5 yrs 6-10 yrs	11 +/- 1.8 19.5 +/- 2
	10-15 yrs	26 +/- 1.2

Table 2: Clinical Presentation

Sr. No.	Variable	n = 25
1	Preoperative Hemolobin	10.83 +/- 00.26
2	Preoperative Hematocrit	35.95 +/- 1.14
3	Ischemic Presentation	20 (80%)
4	Hemorrhagic Presentation	5 (5%)
5	Unilateral Disease	22 (88%)
6	Bilateral Disease	3 (12%)
	Clinical Presentation	
	a) Headache	8 (32%)
	b) Seizures	12 (48%)
7	c) Neurological Weakness	14 (56%)
	d) h/o Loss of consciousness	1 (4%)
	e) Transient ischemic attack (TIA)	22 (88%)
	f) Hemorrhage	3 (12%)

Table 3: Types of Surgical Procedures Performed

Sr. No.	Sr. No.	n = 25 (100%)
A	Direct Vascularisation, External carotid artery (EC) - Internal carotid artery (IC) Bypass) i.e, Superficial temporal artery (STA) - Middle cerebral artery (MCA) Bypass	3 (12%)
B	Indirect Vascularisation	22 (88%)
1	Encephalo-duro-arterio-synangiosis (EDAS)	12 (48%)
2	Encephalo-duro-arterio-myosynangiosis (EDAMS)	8 (32%)
3	Encephalomyosynangiosis (EMS)	0 (nil)
4	Multiple Burrholes	0 (nil)
5	Multiple Burrholes + EDAS	4 (16%)

Table 4: Intraoperative Data Analysed

Sr. No.	Variable	n = 25
1	Duration of Anesthesia (hours)	5.03 ± 0.25
2	Duration of surgery (hours)	4.03 ± 0.25
3	Intravenous fluids (ml)	1430 ± 182.45
4	Urine output (ml)	451 ± 54.45
5	Blood loss (ml)	97.2 ± 9.64
6	Patients requiring blood transfusion	2 (8%)
7	Rectal temperature (°celcius)	35.2 - 36.8
8	Extubated	25 (100%)
9	Post-operatively ventilated	0
10	Central venous access	3 (12%)
11	Arterial access	25 (100%)
	Intraoperative complications	
	a) Hypotension	4 (16%)
	b) Hypertension	2 (8%)
12	c) Tachycardia	2 (8%)
	d) Hypothermia	3 (12%)
	e) Low urine output	0
	f) Hypocapnia	0
	g) Bronchospasm	0

Table 5: Postoperative Data Analysed

Sr. No.	Variable	Result
1	Post operative Hb	10 ± 0.52
2	Post operative Hematocrit	35 ± 0.83
3	Seizures	4 (16%)
4	Hemorrhage	0
5	Infarction	1
6	Re-exploration/Decompression Bone flap removal	1
7	Postoperative ICU stay in days	1.84 ± 0.39
8	Discharge from hospital	10.34 ± 1.039

A total of 25 patients records were evaluated. Ischemic presentations were n = 20 (80%) and haemorrhagic presentations were n = 5 (20%). At the desired tidal volume and respiratory rate delivered during controlled ventilation there was no hypocapnia or hypercapnia and oxygen saturations maintained at 100%. Intraop- eratively two patients n= 2 (8%) had hypertension and tachycardia at the time of scalp incision which was treated with required amount of IV Labetalol (not to use xylocaine infilteration as artery is superficially located may go in spasm) however 4 patients (n = 4(16%) had hypotension after induction of anaesthesia which responded to IV Mephenteramine. None of our patients had low urine output, n = 0 (0%) and no one suffered hypothermia. Blood transfusion was required in 2 patients, n = 2 (8%). Postoperatively Haemoglobin dropped to 10 ± 0.52. Seizures was seen in 4 patients (16%) and treated as per standard protocol. One child developed infarction (4%), and none had haemorrhage or hematoma (0%). It was thus observed that with vigilant and targeted perioperative protocol ICU stay was just 2 days (1.84 ±0.39) and discharge from hospital was within 11 days (10.34±1.039) with no new neurological deficits, with the exception of one patient who had an unfortunate event of infarction, n = 1 (4%) requiring decompression surgery. Hence it was observed with the given anaesthesia protocol there were no significant complications and all patients were extubated on table with no new neurological deficit. Above information can be seen in tables 1,2,3,4 and 5. This was in accordance with a study published by Vivek B Sharma et. al. at AIIMS New Delhi[12].

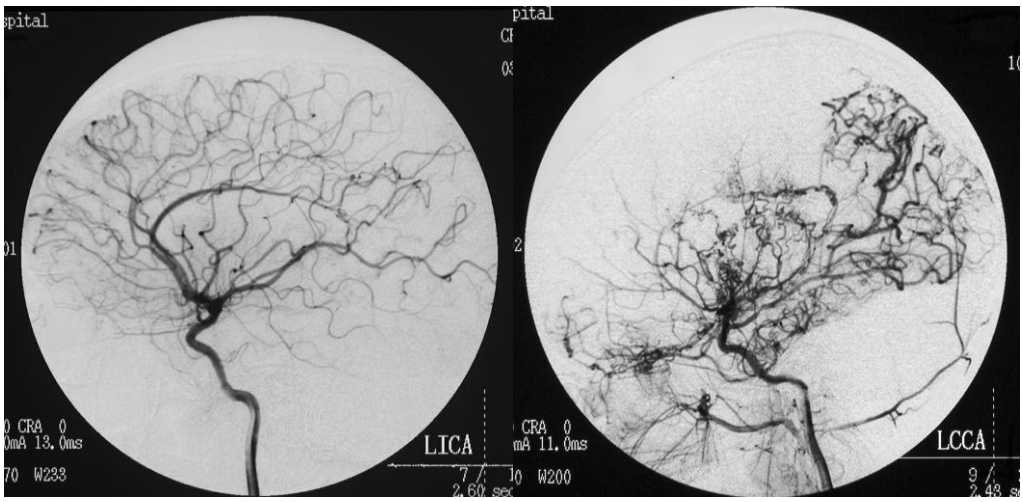

Figure 1: *Left*: Normal DSA Image, *Right*: MMD DSA Image.

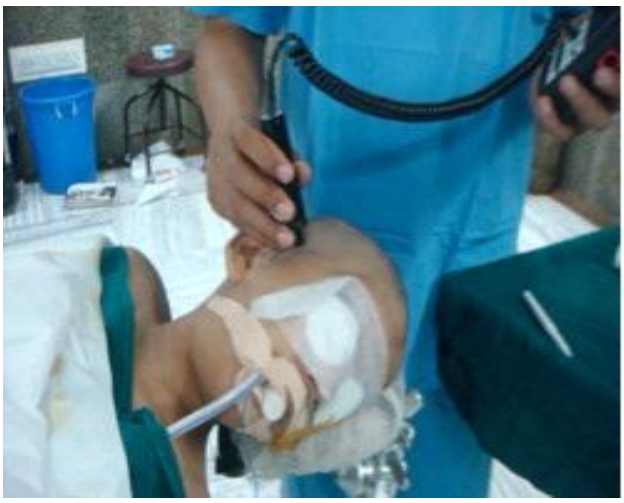

Figure 2: Doppler guided locating of STA (superficial temporal artery).

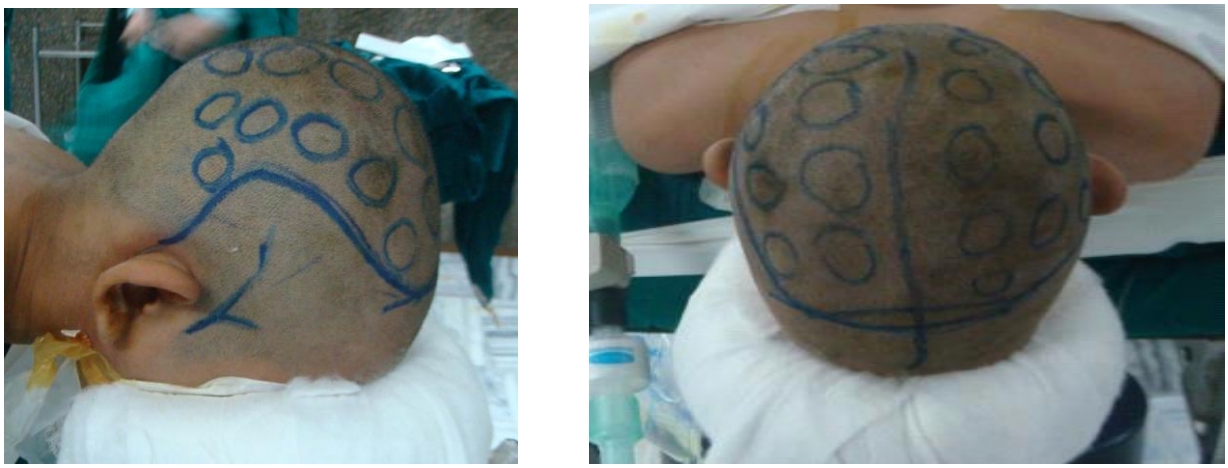

Figure 3: Left : Surface Marking of STA with Doppler. Right : Multiple Burrhole Markings.

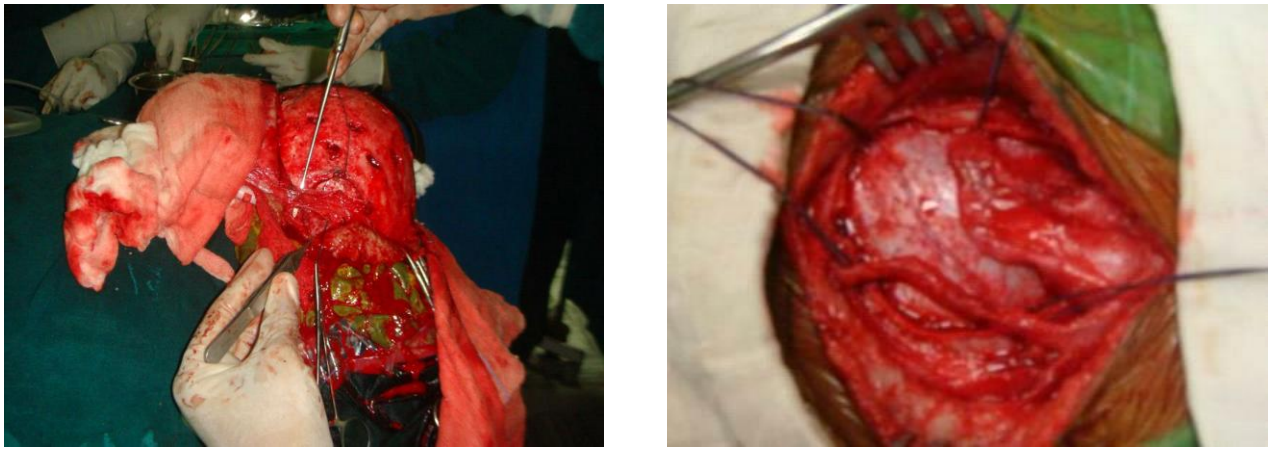

Figure 4: Ultrasound guided Superficial Temporal Artery Dissection.

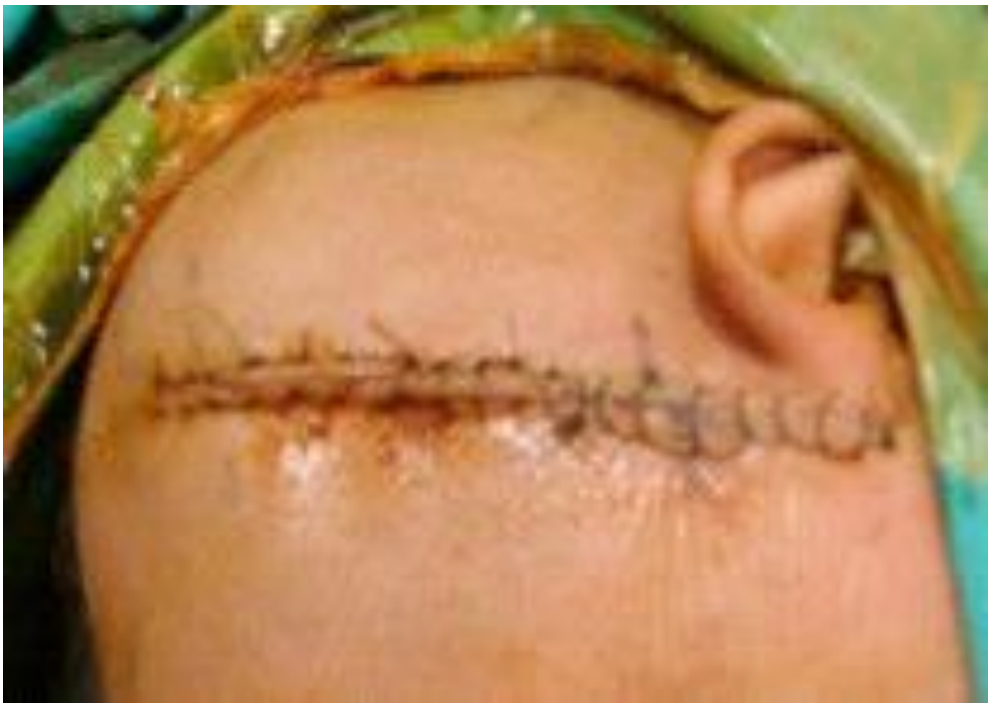

Figure 5: No infilteration. No bandage. No mask over incision to prevent compression on the artery.

Discussion

In Japan's epidemiological survey, the prevalence of MMD was 6.03 and incidence was 0.54 per 100000, however in India incidence is not very clear[13]. Thus suffice to say that MMD being rare, its anaesthesia management has to gradually evolve by retrospectively analysing the results of current anesthesia practices that are in use. Anaesthesia for revascularization for MMD is all about maintaining cerebral oxygenation by strict regulation of all variables that impact CBF, CPP and CMRO2.

The risk factors to develop cerebral ischemia peri or postoperatively in patients with MMD can be categorized as,

- **Patient Factors:** History of repeated transient ischemic attacks (TIA), severity of disease (unilat- eral/bilateral), extent of stenosis, type of revascularization procedures, long duration of surgery and thus anaesthesia and timing of surgery. Although indications and timing of surgery is controversial, it is advisable to wait for atleast 6 months after stroke for revascularisation surgery In a bilateral disease wait for 4 weeks in between procedures for a better neurological outcome. Pediatric patients predominantly presented with ischemic stroke as can be seen in the demographics table whereas adults commonly present with hemorrhagic stroke. This can be explained by the development of more collaterals in adults compared to pediatric population with MMD. Collateral neovasculature is fragile and more predisposed to hemorrhage.
- **Operative Factors:** Avoid significant reduction in hematocrit, intraoperative hypotension, hypocap- nia, hypercapnia, hypoxia, hypovolemia and reduced urine output, mismatch in oxygen supply and demand[14,15]. Hypocapnia leads to cerebral vasoconstriction leading to reduced CBF whereas hypercapnia causes vasodilation of cerebral vasculature. Since the diseased vasculature is already maximally dilated, this causes steal phenomenon further worsening blood flow to the already af- fected part of brain. Hence it is vital to maintain normocapnia. Urine output is a good measure of body's intraoperative blood volume and overall perfusion which is centric to the success of a revascularisation surgery. To Reduce O2 demand (decrease CMRO2), avoid excessive crying of child, depth of anaesthesia and post operative analgesia, avoid tachycardia, hypertension, hypercapnia and hyperthermia[16] and to ascertain good O2 supply correct anaemia, maintain adequate CBF and CPP by avoiding hypotension, hypocapnia, hypovolemia and low urine output. School of thought is divided when it comes to pre op anxiolysis. In theory, an anxious crying child will have hypocapnia and hence cerebral vasoconstriction which would advocate use of anxiolytics. However that is a double edged sword because a drowsy child might develop hypercapnia, hence vasodilation and steal phenomenon. Hence we did not include in our protocol any preoperative anxiolysis in ward before shifting the child to the operating room.

Conclusion

Based on the above observations, suggested aim of anaesthesia management is as follows[16],

- **Detailed preoperative evaluation**.
- **AIM of Premedication**: Non crying child (excessive crying leads to hyperventilation and decreases CO2) keep the child with the parent as far as possible and use local prilocaine patch for a pain free iv accessing, no sedation from wards as we do not want the child to be drowsy and hypoventilate.
- **Aim of Induction**: Use agents causing less or no hypotension, treat with low doses of mephen- teramine immediately if hypotension occurs. Avoid tachycardia to laryngoscopy by using Labetalol appropriately[14].

- **AIM of Maintainance**:
  - Deep plane of anaesthesia with Normocapnia EtCO2 35 to 40[17].
  - Use of N2O is controversial and subjective to the anesthetist.
  - Inhalational agent Sevoflurane with MAC < 1 is advocated, however Sato et. al. [18] suggested use of Total Intra Venous Anaesthesia (TIVA) over Inhalational Anesthesia for revascularization surgeries.
  - Oxygen saturations have to be maintained at 100% at all times, hence keep a check on normaliz- ing haemoglobin and hematocrit values.
  - Maintain urine output at minimum 2ml/kg/hr by maintaining Normovolemia with crystalloids and by replacing blood loss with blood products. Take a central line to measure CVP, especially in STA-MCA anastomosis cases.
  - Maintain normothermia by maintaining warm operating room temperatures and using warm fluids and warming devices for the patient.
  - Maintain Normotension, Mean arterial pressure (MAP) either equal to or more than 20% of baseline.
- **Aim of Reversal**: avoid tachycardia, give multimodal analgesia, avoid narcotic analgesia to avoid hypoventilation/ hypercapnea, no xylocaine/adrenaline infilteration at incision site as STA may go in spasm and no tight bandage or dressing as STA will get compressed.
- **Aim of Postoperative care in ICU**: warm room, vigilant monitoring of intake and output, vitals, pain control, maintain haemoglobin and haematocrit, do not use nasal mask cannula as its strings will sit on STA site and can cause compression.

Conflict of Interest: None.
Financial Aid: None

Acknowledgement
BHIMS (Bombay Hospital), Dept of Neurosurgery - Consultant Anesthesiologist - Dr. Ranjana Das, Consultant Neurosurgeons - Dr. Chandrashekhar Deopujari, Dr. Vikram Karmarkar, Dr. Naresh Biyani.

References

1. Jiro Suzuki and Akira Takaku. Cerebrovascular moyamoya disease: disease showing abnormal net-like vessels in base of brain. *Archives of neurology*, 20(3):288–299, 1969.
2. K. Takeuchi and K. Shimizu. Hypoplasia of the bilateral internal carotid arteries. *Brain Nerve*, pages 37–43, 1957.
3. Youhei Mineharu, Katsunobu Takenaka, Hiroyasu Yamakawa, Kayoko Inoue, Hidetoshi Ikeda, KI Kikuta, Yasushi Takagi, Kazuhiko Nozaki, Nobuo Hashimoto, and Akio Koizumi. Inheritance pattern of familial moyamoya disease: autosomal dominant mode and genomic imprinting. *Journal of Neurology, Neurosurgery & Psychiatry*, 77(9):1025–1029, 2006.
4. R Scott and Edward Smith. Moyamoya disease and moyamoya syndrome. *The New England journal of medicine*, 360:1226–37, 04 2009.
5. Tariq Parray, Timothy W Martin, and Saif Siddiqui. Moyamoya disease: a review of the disease and anesthetic management. *Journal of neurosurgical anesthesiology*, 23(2):100–109, 2011.
6. Markus Kraemer, Peter Berlit, Frank Diesner, and N Khan. What is the expert's option on antiplatelet therapy in moyamoya disease? results of a worldwide survey. *European journal of neurology*, 19(1):163–167, 2012.
7. Satoshi Yamada, Koichi Oki, Yoshiaki Itoh, Satoshi Kuroda, Kiyohiro Houkin, Teiji Tominaga, Susumu Miyamoto, Nobuo Hashimoto, Norihiro Suzuki, et al. Effects

of surgery and antiplatelet therapy in ten-year follow-up from the registry study of research committee on moyamoya disease in japan. *Journal of Stroke and Cerebrovascular Diseases*, 25(2):340–349, 2016.

8. Soumya Sundaram, PN Sylaja, Girish Menon, Jayanand Sudhir, ER Jayadevan, Sajith Sukumaran, Sapna Erat Sreedharan, and Sankara Sarma. Moyamoya disease: A comparison of long term outcome of conservative and surgical treatment in india. *Journal of the neurological sciences*, 336(1-2):99–102, 2014.
9. Lai-Wah Eva Fung, Dominic Thompson, and Vijeya Ganesan. Revascularisation surgery for paediatric moyamoya: a review of the literature. *Child's Nervous System*, 21(5):358–364, 2005.
10. Kyu-Chang Wang, Ji Hoon Phi, Ji Yeoun Lee, Seung-Ki Kim, and Byung-Kyu Cho. Indirect revascu- larization surgery for moyamoya disease in children and its special considerations. *Korean journal of pediatrics*, 55(11):408, 2012.
11. Rabail Chaudhry, George Williams, Shilpa Dhabade, Jones William, Greesha Pednekar, Vasanth Ruthiramani, Carlos Artime, and Lara Ferrario. Effect of intraoperative anesthesiology management on patient outcomes for surgical revascularization for moya moya disease: A 7-year retrospective study, 12 2016.
12. Vivek B Sharma, Hemanshu Prabhakar, Girija Prasad Rath, and Parmod K Bithal. Anaesthetic manage- ment of patients undergoing surgery for moyamoya disease–our institutional experience. *Journal of Neuroanaesthesiology and Critical Care*, 1(02):131–136, 2014.
13. Kenji Wakai, Akiko Tamakoshi, Kiyonobu Ikezaki, Masashi Fukui, Takashi Kawamura, Rie Aoki, Masayo Kojima, Yingsong Lin, and Yoshiyuki Ohno. Epidemiological features of moyamoya disease in japan: findings from a nationwide survey. *Clinical neurology and neurosurgery*, 99:S1–S5, 1997.
14. Toru Iwama, Nobuo Hashimoto, and Yasuhiro Yonekawa. The relevance of hemodynamic factors to perioperative ischemic complications in childhood moyamoya disease. *Neurosurgery*, 38(6): 1120–1126, 1996.
15. Anita Shetty, Avinash Bajaj, Shashikant Shinde, Shrikanta Oak, and Madhu Garasia. "unveiling the puff of smoke": An observational study of anesthesia management of patients of moyamoya disease at a tertiary care centre. *Journal of Neuroanaesthesiology and Critical Care*, 1(01):A29, 2014.
16. Sulpicio G Soriano, Navil F Sethna, and R Michael Scott. Anesthetic management of children with moyamoya syndrome. *Anesthesia & Analgesia*, 77(5):1066–1070, 1993.
17. Koukichi Kurehara, Hideyuki Ohnishi, Hajime Touho, Hitoshi Furuya, and Takao Okuda. Cortical blood flow response to hypercapnia during anaesthesia in moyamoya disease. *Canadian journal of anaesthesia*, 40(8):709–713, 1993.
18. Kiyotaka Sato, Reizo Shirane, Masato Kato, and Takashi Yoshimoto. Effect of inhalational anesthesia on cerebral circulation in moyamoya disease. *Journal of neurosurgical anesthesiology*, 11(1):25–30, 1999.