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Neuroanesthesia Updates 2020

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Recommended Citation

D'Souza S. Neuroanesthesia Updates 2020. UMMs-Baystate Anesthesia Grand Rounds, Sep 2020, Springfield, MA.

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Neuroanesthesia Updates 2020

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 BP target in neurosurgical patients: How high and how low should we go?
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Anti-seizure prophylaxis: TBI, brain tumor surgery, aSAH, AIS
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History of cerebral autoregulation Curve

Fog in 1930 first observed cerebral autoregulation(constriction and dilatation)of blood arteries for varying degrees of blood pressure on a cat's pia mater through a cranial window.

Ref: 1.Fog M: cerebral circulation. The reaction of of the pial arteries to fall in blood pressure. Arch Neurol Psych 1937: 37: 351-64

2. Fog M: Cerebral circulation II. The reaction of the pial arteries to increase in blood pressure. Arch Neurol Psych 1939; 41;260-68

History of cerebral autoregulation Curve Journey from 1959 to 2020: from Static to dynamic

- Lassen in in 1959 first described the lower limit of autoregulation.
- His concept was based on over 200 clinical studies based on
- a) assessing CBF flow using inert gas technique
- b) indicator dilution method
- Studies of controlled hypotension in humans Fick principle and Kety-Schmidt technique

Ref: Lassen NA: Cerebral blood flow and oxygen consumption in man. Physiol rev 1959; 39:183-89

Lassen 1959 concept of autoregulation

Inert gas technique: Measures arteriovenous difference

Indicator dilution method: Measures the venous dilution of an arterially injected indicator

Lassen 1959 concept of autoregulation

Fick principle:

Blood flow to the brain= Amount of the substance injected X arterial and venous difference of the substance

Kety-Schmidt technique: Quantitative measurement of arterial and venous blood flow from washout curves of the diffusible tracer material

History of cerebral autoregulation curve

Upper limit of the cerebral was described in 1971.

Ref: 1. Europ Neurol 1971; 6; 6-10 2. Stroke 1973; 4: 139-47 History of cerebral Autoregulation curve From 1959(static) to 2020(dynamic) Other methods:

TCD Positron emission tomography Plethysmography Cerebral angiography MRA

Ref: Journal of Cerebral Blood Flow & Metabolism 2008 : 28, 1071–1085

Cerebral Autoregulation Curve from 1959 to 2020?

Do we need to revise the lower limit of autoregulation?

Do we need to revise the lower limit of autoregulation?

Drummond JC:Correspondence to the editor The Lower Limit of Autoregulation:Time to revise our Thinking?

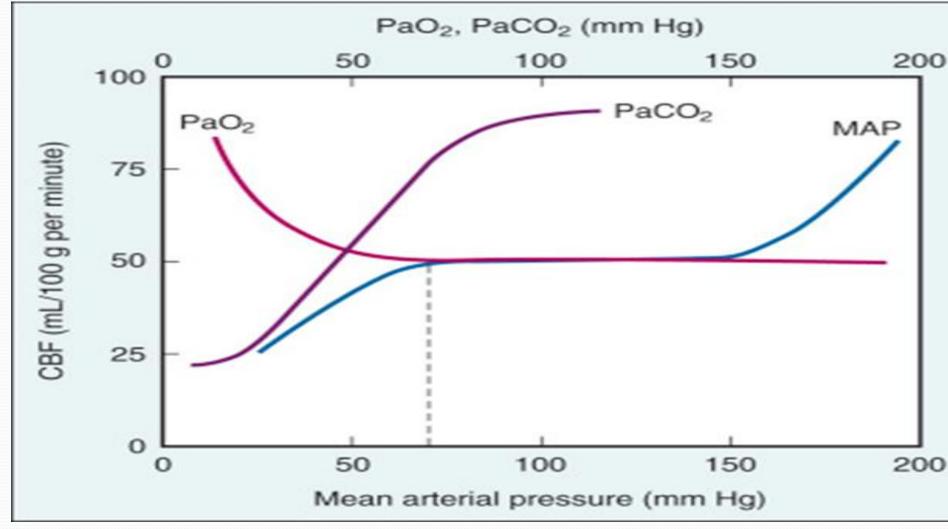
Ref: Anesthesiology 1997;86:1431-33

Author	Hypotensive Technique	CBF Method	LLA Mean (Range)
McCall* ⁴	Hydralazine	K-S/N2O	<64 (33-80)
	Veratrum viride	K-S/N2O	<57 (40-72)
Moyer, <i>et al.</i> ⁶	Hexamethonium	K-S/N2O	>62 (53-80)
	Trimethaphan	K-S/N2O	>57 (44–75)
	Pendiomide	K-S/N2O	<61 (54–72)
Strandgaard ⁸	Trimethaphan/tilt	1/A-VDO2	73 ± 9
Waldemar, <i>et al.</i> ¹⁰	Trimethaphan/lower body negative pressure ± captopril	1/A-VDO2	79 (57–101)
Larsen, et al.11	Lower body negative pressure/labetalol	1/A-VDO2	79 (53–113)
		CBFVmca	91 (41–108)
Olsen, et al.12	Labetalol/lower body negative pressure	1/A-VDO2	88 (76–101)
Olsen, <i>et al.</i> ¹³	Lower body negative pressure/labetalol	1/A-VDO2	73 (60–100)
	-	A-NIRS diff	79 (73–101)

Data are presented as mean values with ranges (when available) or standard deviations.

* The subjects were 42 pregnant women near term, 24 of whom had toxemia of pregnancy.

K-S/N20 = Kety-Schmidt technique using nitrous oxide as the tracer; CBFVmca = mean CBF velocity in the middle cerebral artery; 1/A-VDO2 =the constancy of cerebral metabolic rate was assumed and that CBF was determined by the formula CBF = a constant \times 1/(arterial-jugular venous oxygen content difference); A-NIRS = the constancy of cerebral metabolic rate was assumed, and a decrease in CBF was inferred when the arterial to regional saturation difference (the latter determined by near infrared spectroscopy) increased; < = the LLA was not identified, but that CBF was unchanged from the control value at the MAP indicated; > = the LLA was not identified, but that CBF was less than the control value at the MAP indicated. **Cerebral autoregulation curve Image from Lemkuil BP, Drummond JC, Patel PM. Central nervous system physiology: Cerebrovascular. In Hummings HC, Egan TD, eds.** *Pharmacology and Physiology for Anesthesia: Foundations and Clinical Application*. Philadelphia, PA: Elsevier; 2019:174-192, Fig. 9.7.



Lower limit of Cerebral autoregulation

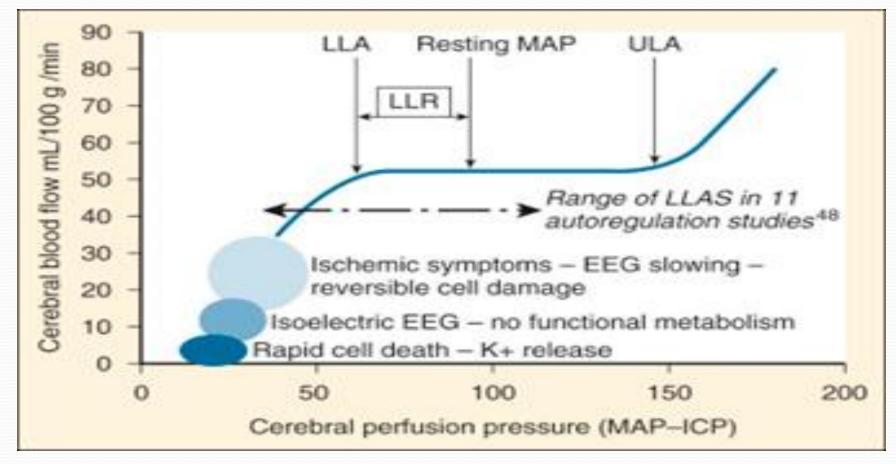
Lower Limit of cerebral Autoregulation= 70 mm Hg

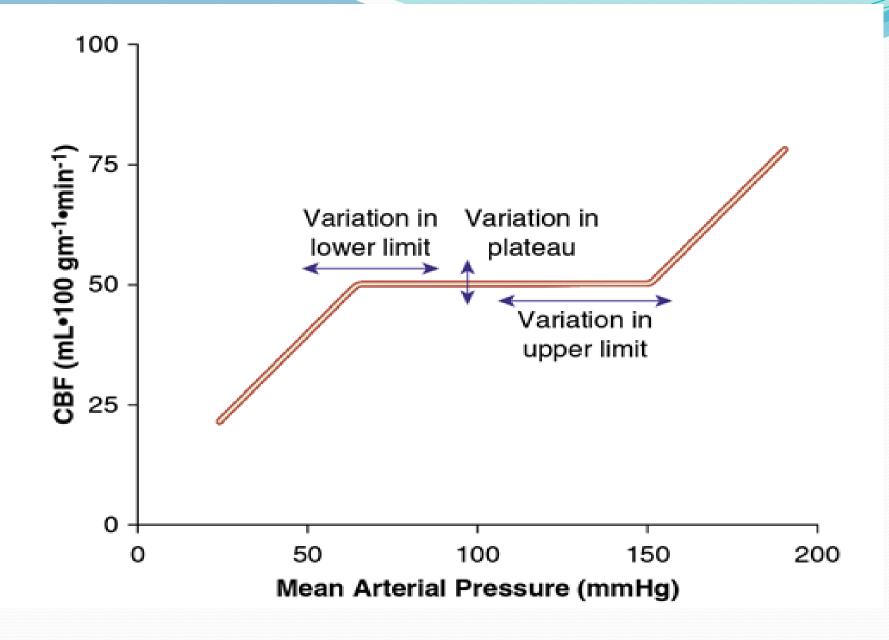
Ref: 1. Anesthesia for Neurosurgery, Smith's Anesthesia for Infants and Children 2017; 744-72

2. Miller's anesthesia 2020, 9th edition

Cerebral Autoregulation curve

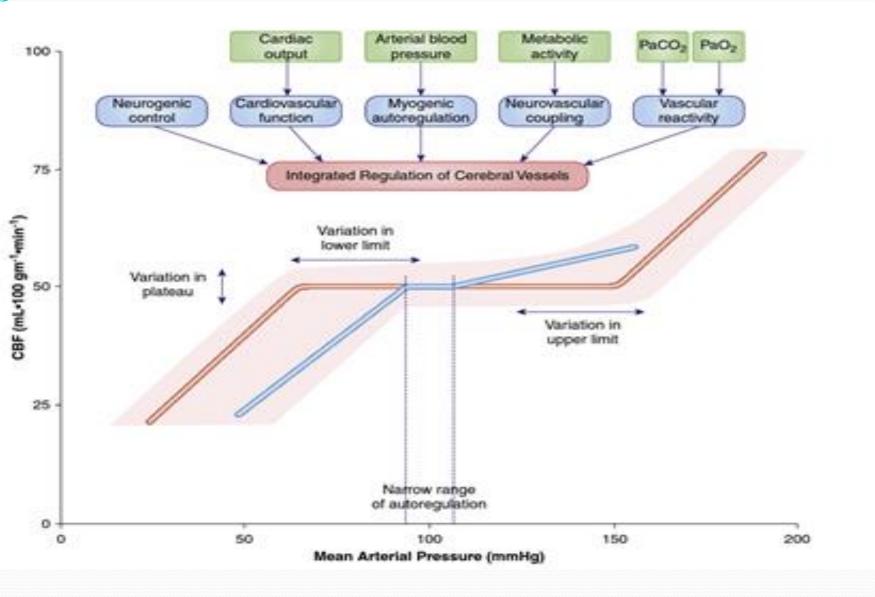
Anesthesia for Neurosurgery, Smith's Anesthesia for Infants and Children 2017; 744-72



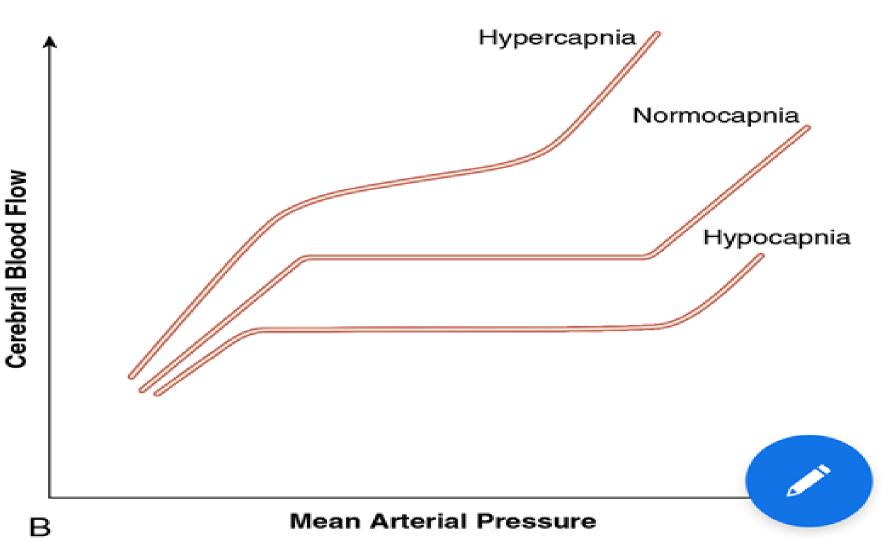


Dynamic Cerebral Autoregulation : Miller's anesthesia 2020.

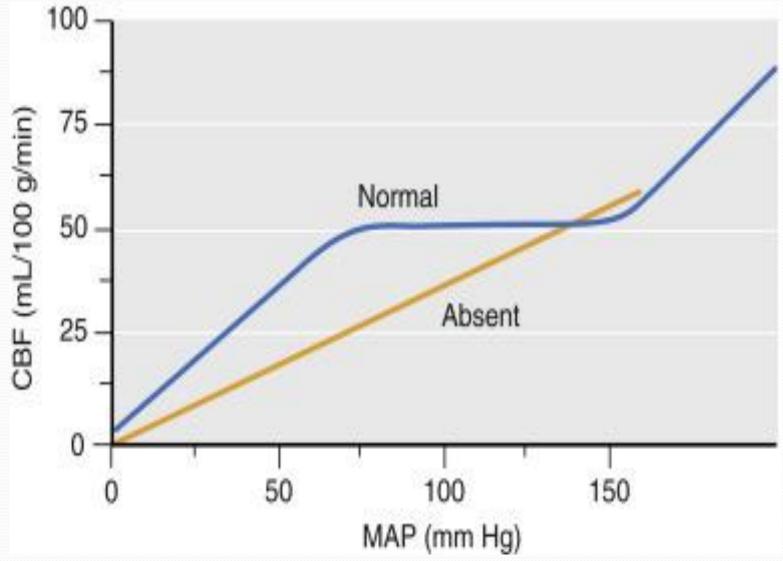
9th edition



Effect of PaCO2 on cerebral autoregulation: Miller's Anesthesia 2020, 9th edition



Cerebral autoregulation curve in acute intracranial event Ref: Miller's Anesthesia 2020, 9th edition



Pathophysiology of EBI

Early brain injury (EBI) formerly known as primary brain injury.

Loss of cerebral autoregulation Loss of blood brain integrity

EBI: Past historical concept of Systemic Hypertension

CPP = MAP - ICP

Systemic hypertension secondary to Cushing's reflex.

EBI: Hypertension current concepts

- Following EBI, massive sympathetic discharge and catecholamine release resulting in hypertension and tachycardia
- Cytokine release can result in leaky capillaries: pulmonary edema
- Lower BP: cerebral ischemia, at higher MAP, cerebral edema
- **Ref: 1 Intensive care 2016; 4: 29**
- 2. Neurosurg Focus 2019; 45;5
- 3. J Neurosurg Anesthesiol 2017; 29:382-87

EBI: Pathophysiology

- Catecholamine surge: EKG changes, wall motion abnormalities on TEE: Takotsubo cardiomyopathy(1)
- Cytokine release: leaky capillaries, neurogenic pulmonary edema.(2)

References:

- Krishnamoorthy V, Mackensen GB, Gibbons EF, Vavilala MS. Cardiac Dysfunction After Neurologic Injury: What Do We Know and Where Are We Going?. Chest 2016;149:1325-31
- 2. Bust K, Bleck T: Neurogenic pulmonary edema. Crit Care Med 2015:43; 1710-15

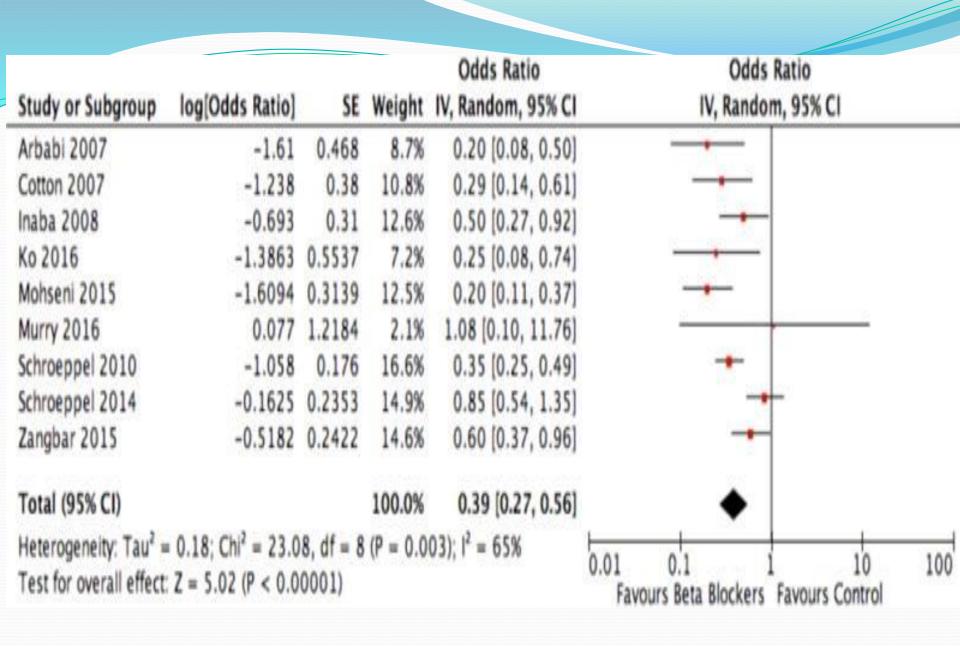
TBI :?Beta Blockers

TBI with beta blockers: Systematic review and metaanalysis.(9 studies n= 8245)

Results: Low quality of evidence of reduction in hospital mortality (OR: 0.39, CI 95%)

Long term functional outcomes were not measured in these studies.

Ref: Alai AS et al: Beta-Blockers and Traumatic Brain Injury: A Systematic Review and Meta-Analysis. Ann Surg 2017; 266; 952-61



aSAH and post event Beta blockade

A meta-analysis and systematic review reported in 2020 16 studies, 4 RCT's 12 prospective observational n=6702 Results: In house mortality benefit (OR 0.50 CI 95%),

Limitations: Heterogeneity

Ref:Ramesh, A.V., Banks, C.F.K., Mounstephen, P.E. *et al.* Beta-Blockade in Aneurysmal Subarachnoid Hemorrhage: a Systematic Review and Meta-Analysis. *Neurocrit Care* 2020. https://doi.org/10.1007/s1₂₀₂₈₋₀₂₀₋₀₀₉₁₅₋₅

Reference point to measure ABP in neurosurgical patients

Past: External auditory meatus
 Current: Highest point of the head

Ref: Drummond: ASA refresher course lecture, Boston 2018

EBI:hypertension management in neurosurgical Patients: What drugs?

- Vasodilator drugs: Hydralazine, nitroprusside and nitroglycerine should not be used.
- Calcium channel blockers: Clevidipine, nicardipine are preferred
- Beta blockers can be used except in DBS macro electrode implantation

References:

- 1. Neurocrit Care 2011;15:211-240
- 2. Stroke 2012;43:1711–37

Target BP in neurosurgical patients: How high and How low should we go?

TBI
AIS
sICH
aSAH
DBS

Target BP in TBI as per BTF guidelines

Brain trauma foundation(BTF) guidelines developed by the American congress of Neurosurgeons for management of Traumatic Brain Injury(TBI)

Third edition in 2007

Fourth edition in 2017 (called 2016 guidelines)

2016 BTF guidelines: What is new? <u>Redefining hypotension in TBI</u> BTF guidelines(third edition in 2007): Defined hypotension as systolic BP less than 90 mmHg.

• **BTF guidelines(Fourth edition 2016 guidelines):** defined hypotension as systolic BP less than 110 mm Hg

Ref: 1 BTF guidelines 2016: 4th edition

2. Berry C, Ley EJ, Bukur M, et al. Redefining hypotension in traumatic brain injury. *Injury*. Nov 2012;43:1833-37

3 Brenner M, Stein DM, Hu PF, Aarabi B, Sheth K, Scalea TM. Traditional systolic blood pressure targets underestimate hypotension-induced secondary brain injury. *J Trauma Acute Care Surg*. May 2012;72: 1135-39

Redefining hypotension......

BTF analysed the data from

- Two class II retrospective studies(n=22594)(ref 1, 2, 3)
- 16 Class III retrospective and prospective observational data(n=2444)(ref 3)

Ref 1.Berry C, Ley EJ, Bukur M, et al. Redefining hypotension in traumatic brain injury. *Injury.* Nov 2012;43:1833-37.

- 2. J Trauma Acute Care Surg 2012;72:1135-1139
- 3. Brain Trauma Foundation Guidelines 2016, 4th edition

TBI: Target pressure

Target cerebral perfusion (CPP)

Third edition: 50- 70 mm Hg (BTF 2007) Fourth edition 60-70 mm Hg (BTF 2016)

Ref: BTF guidelines 2016:4th edition

Redefining CPP......

BTF analysed the data from..... 1 RCT

- 15 Retrospective data
- (total n= 4619)

Ref Brain Trauma Foundation Guidelines 2016, 4th edition

A patient with severe head injury with GCS of 4 presents for emergency evacuation of an hematoma. What is your target MAP?

Target MAP during Acute TBI management

CPP = MAP - ICP

MAP = CPP + ICP

MAP = 60+ (20 or 25)= 80-85

MAP = 70+ (20 or 25)= 90-95

TBI: Why narrow range MAP?

- Loss of cerebral autoregulation
- Loss of blood-brain integrity
- CBF depends on MAP
- Hypotension results in hypoperfusion
- Hypertension results in hyperperfusion and cerebral edema

AIS: Target BP prior to EVT

Target BP prior to EVT(endovascular thrombectomy)
 140-180 mmHg systolic

Why higher BP in AIS prior to EVT? : for collateral blood flow through circle of Willis

Ref: J Neurosurg Anesthesiol 2014;26:95-108

Target BP after EVT in AIS

- A retrospective analysis of data from 3 RCT's(comparing GA vs MAC for EVT) reported in 2020;
- n= 365 (SIESTA, ANSTROKE, GOLIATH)
- MAP less than 70 over 10 min and MAP greater than 90 over 45 min is associated with poor functional outcome at 90 days(mRankin score).

Ref: JAMA Neurol 2020; 77:622-631

Target BP in Spontaneous Intracerebral hemorrhage (sICH)

INTERACT 2 trial: Compared systolic BP less 140 mm Hg(intensive group) Vs systolic BP less than 180 mm Hg

n= 2794 (randomised within 6 hours of sICH)

Outcome measures death and disability rate on mRankin score at 90 days

Results: Mortality 11.9 % intensive group vs 12.0% and non fatal disability rate 23.3% vs 23.6%

Ref:N Engl J Med 2013; 368:2355-2365

Target BP in sICH

ATTACH 2 trial

Compared 110-139 mmHg systolic to 140-179 mm Hg systolic within 4.5 hours of ICH symptoms (mean 128 mmHg Vs 160 Hg)

n= 1000

Primary outcome measures: death and disability rate at 90 days did not differ between the two groups.

Ref: N Engl J Med 2013; 368:2355-2365

Target BP in ICH

Analysis of Data from ATTACH 2 trial:

Elevation of creatinine within 7 days(1) Neurological deterioration within 24 hours(2) Adverse cardiac events within 7 days (2)

Adverse events were higher in intensive group

References:

- 1. N Engl J Med 2016; 375:1033- 1043
- 2. Stroke 2018;49:1412–1418

Target BP for aSAH prior to

securing aneurysm

Prior to 2011 : Systolic BP 180 mm Hg prior to coiling or clipping

2011: 160 mm Hg as systolic as per neurocritical care consensus statement (1)

2012: Above statement endorsed by ASA of the AHA(2)

Currently based on results of INTERACT 2(3) and ATTACH 2 trials: 140 mm Hg(4)

References: 1,Neurocrit Care. 2011;15:211–240. 2.Stroke 2012;43:1711–1737 3.N Engl J Med 2013; 368:2355-2365 4. N Engl J Med 2016; 375:1033-1043

Target BP for temporary clipping

• Maintain the BP or elevate above the MAP.

Maintain burst suppression on EEG.

Aneurysm clipping Target BP for induced hypotension

BP of 50 mm Hg: achieved by clevidipine

Adenosine 0.3-0.4 mg/kg

aSAH: Target BP for DCI after securing aneurysm

HHH therapy is part of the history now! Hypervolemia and hemodilution not used now. What about induced hypertension? : Its role is controversial

Induced hypertension to prevent DCI after secring aneurysm in aSAH?

RCT(n=240) was prematurely terminated after recruiting 42 patients : after observing no benefit

Ref 1: Stroke 2018;49:76-83

Induced hypertension to prevent DCI after secring aneurysm in aSAH?

Retrospective data analysis reported in 2018

3 academic centers from Netherlands, 2006-2015) n= 1647

479 showed DCI

300 showed cerebral infarction on CT scan

201 were treated with induced hypertension, 99 were not treated with induced hypertension

With induced hypertension 41/201(20%) compared 33/99(30%) without induced hypertension developed cerebral infarct.

Ref: Stroke 2018; 49:2630-2636

Target BP for Deep Brain Stimulation

Less than 140 mm Hg , not below 120 mm Hg for macro/microelectrode implantation.

BP above 140 mm Hg is associated with intracerebral hemorrhage and worse neurological outcome as per case series.

 Hemodynamic changes(hypertension/hypotension can occur during Macro/micro electrode implantation during DBS

Ref:Front Neurosci. 2017; 11: 477

Target BP: Summary

Neurosurgical condition	Target BP/CPP in mm Hg
тві	CPP: 60-70 Avoid systolic BP below 110
AIS prior to EVT	140-180 systolic
AIS after EVT	70-90 MAP
sICH	120-140 systolic
aSAH	120-140 systolic
DBS	120-140 systolic

PaCo2 management

Normocarbia, Paco2 35-40 mm Hg is the goal
 Hypercarbia increases cerebral blood flow and abolishes cerebral autoregulation curve
 Hypocarbia preserves the autoregulation curve with

decrease in CBF.

Ref: Miller's Anesthesia 9th edition ; 2020

PaCO2 management In TBI

Cerebral blood flow decreases to 50% of the normal at the end of 4 hours and reaches to 80% of the normal at the end 24-72 hours based on severity of the TBI

Hyperventilation contraindicated for the first 24 hours after TBI.

Ref: 2016 BTF guidelines 4th edition

PaCO2 management: Why frequent measurement of arterial PaCO2 is essential?

- Patient with acute intracranial event: catecholamine surge
- Decreases pulmonary capillary blood flow
- Increases dead space ventilation
- Increases PCo2- End- tidal Co2 differences
- Hourly estimation of PaCo2 to identify PaCo2-End Tidal CO2 gradient.

TIVA vs Volatile anesthetics for Neuroanesthesia Which is the best technique?

Anesthesia technique: TIVA vs Volatile anesthetic agents

- A meta-analysis of 14 RCT's
- **n**= 1891
- TIVA: Propofol/fentanyl and Propofol/remifentanil
- Volatile anesthetic agents: Isoflurane/sevoflurane in air/oxygen mixture
- ASA 1-3
- Patients had no or minimal midline shift on CT scan

Ref: Can J Anaesth 2014; 61:347-56

Results in TIVA group compared volatile agent group

- ICP= -5.2 mm/Hg less(95% confidence interval -6.81 to -3.6 mm HG)
- 2. CPP was +15.3 mm Hg (95% confidence interval

12.2 to 20.46 mm Hg)

3. Limitations: Outcome measures were not studied in these trials of this meta-analysis.

Ref: Can J Anaesth 2014; 61:347-56

TIVA or Volatile agents?

A recent study reported in 2019

- n= 90 patients with TBI
- 45 received propofol based TIVA 100-150 mcg/kg/min
- 45 received Isoflurane 1 MAC or less
- Results: ICP was lower in TIVA group and brain relaxation at dural opening and closure was better with TIVA group

Ref:*Eur J Trauma Emerg Surg* 2019 https://doi.org/10.1007/s00068-019-01249-4

Neurological recovery: TIVA vs Volatile agents?

A Cochrane review in 2016 (15 RCTs, n=1833) reported that neurological recovery is similar in both TIVA volatile anesthetic agent based technique in patients undergoing brain tumor surgery.

Ref: Cochrane Database Syst Rev 2016;9:CD010467

IV vs inhalational technique?

TIVA alone or with lower concentration of volatile anesthetic most commonly used popular technique in neuroanesthesia.

References;

- 1. Miller's anesthesia 9th edition 2020
- 2. Uptodate 2020

3. Pasternak JJ: Neuroanesthesiology Update. J Neurosurg Anesthesia 2019; 2; 178-9**8**

Why TIVA is preferred in Neuroanesthesia?

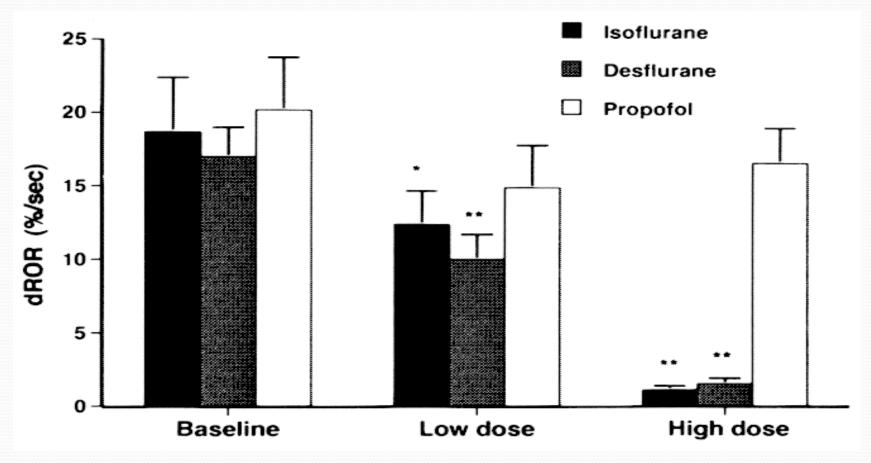
- Propofol based TIVA maintains cerebral autoregulation curve and decreases the ICP.
- Volatile anesthetic agents suppress the cerebral autoregulation.

Ref:

- 1. Armstead WM: Cerebral autoregulation and dysregulation. Anesthesiol Clin. 2016 34: 465–477
- 2. Miller's Anesthesia 2020; 9th edition

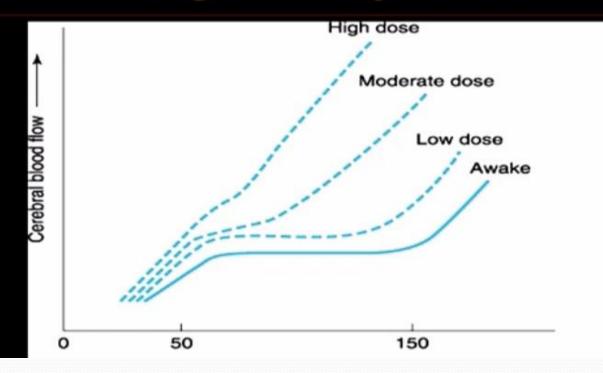
Effect on cerebral autoregulation

Anesthesiology 1995;83, 66-76 (n=42, TCD)



Ref Sharma D: ASA refresher course lecture 2019

Dose-dependent Depression of Cerebral Autoregulation by Volatile Anesthetics



Cerebral blood flow: TIVA vs Sevoflurane

• A Study from Finland: reported in 2003

ASA grade 1 healthy male volunteers (8)

Ref: Anesthesiology 2003; 99:603-613

- Induced with sevoflurane 8% and intubated with rocuronium 0.6mg/kg body weight
 Maintained with sevoflurane
 Then added 70%N20 after 50 min
- TCI of propofol and then added 70% N20 after 50 min

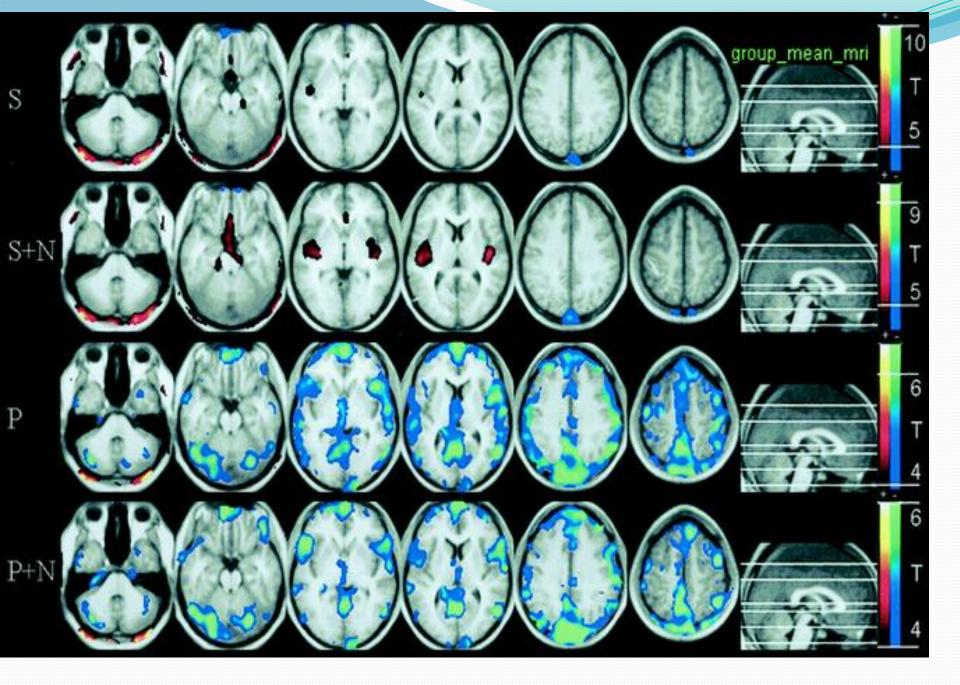
Ref: Anesthesiology 2003; 99:603-613

Anesthesia depth BIS 40

 (Average sevo 1.5% end tidal (range 1.1%-1.9%, propofol tcI 4ng/ml)

Cerebral blood was observed on PET scan

Ref: Anesthesiology 2003; 99:603-613



Volatile anesthetic agents concentration greater than 1 MAC should be avoided.

Ref: Miller's 9th edition, 2020

Which volatile anesthetic agent?

Vasodilatory effect: desflurane > Isoflurane > sevoflurane (under 1 MAC clinically not significant)

Miller's Anesthesia 9th edition; 2020

IV vs inhalational technique?: Current concept

- In patients with elevated ICP: Volatile anesthetic agent concentration should be limited to 0.5 MAC.
- In acute expanding intracranial events such as TBI/ aSAH, persistently elevated ICP in brain tumors: volatile anesthetic agents should be avoided altogether at least until the dura is opened.

Ref:

- 1. Miller's Anesthesia 2020; 9th edition
- 2. Armstead WM: Cerebral autoregulation and dysregulation. Anesthesiol Clin. 2016 Sep; 34: 465–477

Dexmedetomidine and remifentanil

- Commonly used adjuncts with propofol when EEG, MEP and SSEP monitoring is used.
- Reduces the dosage of propofol and enable to monitor EEG without causing burst suppression

Dexmedetomidine

- Widely used in neuroanesthesia
- Provides analgesia
- Reduces emergence delirium
- Reduces dosage of propofol
- Doesn't affect EEG monitoring significantly
- Infusion dosage should be limited to 0.5 mcg/kg/hr when MEP monitoring used.
- Part of the regimen for awake-asleep-awake craniotomies and DBS.

Ref: 1 J Neurosurg Anesthesiol 2019; 31: 366-77 2. J Clin Anesth. 2019; 54:114-125.

Remifentanil in Neuroanesthesia

Reduces sympathetic response to intubation and insertion of Mayfield pins

- 2. NeuroMorfeo Trial: Reduced the urinary catecholamine levels (ref 1)
- 3. Reduction in in-house mortality rate in patients with clipping of ICA aneurysms when remifentanil was used as per a retrospective Japanese database analysis (ref 2)

References:

- **1.** Eur J Anaesthesiol. 2012; 29:371-9
- 2. J Neurosurg Anesthesiol. 2014;26:291–298

Fluid Management in Neurosurgery

Fluid management in

Neurosurgery

NS osmolality closer to plasma

- LR is slightly hypotonic.
- Chloride rich solutions that contribute toward hyperchloremic acidosis and impaired renal blood flow.(**Ref 1,2,3**)

As transfusion of NS associated with metabolic acidosis and renal dysfunction LR or plasmalyte are commonly used in neurosurgery.(**Ref 1, 2, 3**)

Ref: 1. Miller's 9 the edition, 2020

Pasternak JJ: Neuroanesthesiology Update. J Neurosurg Anesthesia 2019;
 178-98

3. Dabrowski W., Wise R., Malbrain M.L.N.G. Fluid Management in Neurosurgical Patients.Textbook of Neuroanesthesia and Neurocritical Care 2019; 373-382

Fluid management in Neurosurgery

Replace half of the urine as fluids when mannitol is used.

Goal is to maintain Euvolemia.

If large amount of fluid transfusion is needed (more than two liters alternate NS with LR.

Fluid warmer is not needed for a routine craniotomy for tumor surgery, as large volume of fluids are not needed in routine craniotomy for tumor surgery

Ref: Miller's Anesthesia 2020, 9th edition.

Albumin in neurosurgical patients : TBI

Subset analysis of SAFE trial in severe TBI (GCS 3-4)patients in 2009 reported higher morality with 4% albumin at 30 days.

Ref: N Engl J Med 2007; 357: pp. 874-884

? Albumin in TBI

Limitation for this conclusion of SAFE trial:

- 1. Not randomized to include TBI (1)
- 2. 4% albumin was hypoosmolar (osm 274 mOsmol/L)(2)

References:

- 1. Anesth Analg 2011; 113: pp. 426-427
- 2. Curr Opin Anaesthesiol 2012; 25: pp. 563-565

? Albumin in TBI

A single center retrospective analysis (n=93) from Sweden reported a lower 28 day and 18 month mortality in severe TBI patients when albumin was used with zero or negative fluid balance in TBI patients.

Ref: Acta Anaesthesiol Scand 2008; 53: pp. 18-25

Albumin in aSAH

- 25% albumin was found to be beneficial in aSAH as per ALISAH pilot trial(n=47)(1)
- Lower incidence of TCD vasospasm, DCI and cerebral infarction at 90 days.(2)

References:

- Suarez JI, Martin RH, Calvillo E, et al. The Albumin in Subarachnoid Hemorrhage (ALISAH) multicenter pilot clinical trial: safety and neurologic outcomes. *Stroke*. 2012;43(3):683-690
- 1. Suarez J.I., Martin R.H., Calvillo E., Bershad E.M., and Venkatasubba Rao C.P.: Effect of human albumin on TCD vasospasm, DCI, and cerebral infarction in subarachnoid hemorrhage: the ALISAH study. Acta Neurochir Suppl 2015; 120: pp. 287-290

Albumin in AIS: ALIAS phase 3 trial

Age 18-82, NIHSS score <or = 6 n=830

NS vs

25%albumin (2gm/Kg) infused over 2 hours within 5 hours of stroke onset.

Results: Symptomatic ICH (at 24 hours) and CHF/pulmonary edema was more common with albumin group.

Overall neurological outcome was similar in both groups.

Ref: PLoS One 2015; 10: e0131390

Hyperosmolar therapy: Mannitol Vs Hypertonic saline Which is better?

Hyperosmolar therapy In TBI: Mannitol Vs hypertonic saline

- HTS decreases the ICP better than Mannitol without short term outcome difference
- 2007 BTF guidelines: HTS was preferred over Mannitol in TBI patients.

2016 BTF guidelines:Either mannitol or HTS is acceptable.

HTS vs Mannitol in TBI

• A meta-analysis in 2019 (12 RCT's n= 464) found decrease in ICP was better with HTS at 30-120 min interval.

No outcome difference at discharge
Long term outcome was not studied in the trials of this meta-analysis.

Ref : J Neurosurg Anesthesiol 2019; 00:000–000 DOI; DOI: 10.1097/ANA.00000000000644

HTS vs Mannitol in TBI

A Cochrane review in 2020(n=287, 6 RCT's) comparing Mannitol vs HTS

91% of these patients had GCS of less than 8 (severe TBI)

3 trials reported outcome with regard to hospital discharge, out of which 2 reported in house mortality.

Conclusion: No difference in outcome Limitations: Heterogeneity of methodology of the trials.

Ref: Cochrane Database of Systematic Reviews 2020; CD010904.

Mannitol Vs HTS for brain tumor surgery

• A meta-analysis in 2015 of 7 RCT's reported better brain relaxation with HTS compared to mannitol.

 Limitation: Heterogeneity of the trials, other factors were not standardized.
 Outcome measures were not studied.

Ref: PLoS One 2015; 10: e0117314. PMID:25635862

Mannitol

Mannitol 20%
Dose: 0.25 gm/kg to 1 gm/kg
1Gm/ Kg is the optimum dose

Ref: Miller's 9th edition 2020 Uptodate 2020 J Neurosurg Anesthesiol 2020;32:307-314

Mannitol In neurosurgery

FDA has also approved mannitol 25% in neuroanesthesia.

Rate of administration 20% or 25%: over 30-60 min.

Ref: FDA 2019 recommendation of Mannitol.

Mannitol in Brain tumor surgery

Optimum dose of mannitol is 1 gm/kg(Ref 1,2, 3).

Tumor size and peritumoral edema are primary determinants of mannitol dose rather than midline shift (Ref 2)

Ref:

- 1. Miller's Anesthesia, 9th edition, 2020
- 2. J Neurosurg Anesthesiol 2020;32:307-314
- 3. J Neurosurg 2017; 126:1747-2058

Mannitol in Brain tumor surgery

Higher doses of mannitol produces better brain relaxation.

 Higher doses of mannitol is associated with electrolyte abnormalities: Hyponatremia, hyperkalemia and increased serum osmolality.

 Higher dose of mannitol may cause postoperative cerebral edema

Ref:

- 1. Uptodate 2020
- 2. J Neurosurg Anesthesiol 2020;32:307–314
- 3. J Neurosurg 2017; 126:1747-2058

Anti-Seizure Prophylaxis

Anti- seizure prophylaxis TBI

TBI: early benefit for 1 week to prevent seizures, no long term benefit.

Ref; BTF guidelines, 4th, edition 2016

Anti- seizure prophylaxis TBI

• A recently published meta-analysis and systematic review in 2019

3 RCT's(n= 750) and 6 observational studies (n=3362)

Results: Benefit was limited early seizure prevention only.

Ref: World Neurosurg 2019;122:433-440

Prophylactic Anti-seizure medications: Brain tumor surgery

A meta-analysis reported in 2019

2 RCT's and 4 retrospective analysis.

454 received anti-seizure medications

333 did not receive anti-seizure medications, or placebo in RCT

Results : No benefit from anti-seizure medication

Ref: Journal of Neurology, Neurosurgery & Psychiatry 2019;90:599-607.

Prophylactic Anti-seizure medications: Brain tumor surgery

A 2020 Cochrane review also did not find the benefit of anti-seizure prophylaxis(10 trials, n= 1815)

Ref: Cochrane Database of Systematic Reviews 2020; CD007286.

Antiseizure prophylaxis for aSAH?

A retrospective data analysis (n-363, 43% received antiseizure prophylaxis): No benefit

Ref: Stroke 2016; 47:1754-60

Anti-seizure prophylaxis in sICH

- Not recommended as per 2015 AHA clinical guidelines(Class 3 Level B evidence)
- Not recommended as per 2014 European Stroke Association guidelines
- There is no RCT data to support Anti-seizure prophylaxis use in sICH.

Ref: Stroke 2016;47:2666-72

Antiseizure prophylaxis in AIS

 Anti-seizure prophylaxis is not recommended in AIS as per 2018 ASA/AHA guidelines.

Ref: Stroke. 2018;49:e46-e99.

Summary: Anti-seizure prophylaxis

Neurosurgical Category	Antiseizure Prophylaxis indication
ТВІ	Yes
Brain tumor surgery	No
aSAH	No
sICH	No
AIS	No

A Patient with Severe TBI

Protocol for patient with severe Traumatic Brain injury with GCS 3 with emergency surgery to evacuate extradural hematoma. Let us assume that patient is an young adult otherwise healthy.

Anesthetic management

Management of severe head injury

- Work along with surgeon, consider that quick surgery is important.
- Preoxygenate
- Discontinue clevidipine/nicardipine prior to induction
- Circulate NIBP every 1-2 min
- 1 mg hydromorphone

Management of severe TBI

- Start phenylephrine at 0.3 mcg/kg/min
- Propofol 200 mg along with 80-160 mcg phenylephrine
- Goal is not to drop the BP below 110 mm Hg(as per BTF guidelines)
- CPP of 60-70 is the target(MAP 85-95 mm Hg)(as per BTF guidelines.

Management of severe TBI

- Rapid sequence induction if the patient at high risk of aspiration
- Administer defasciculating dose of Rocuronium before succinylcholine
- Use Rocuronium if the patient is not at risk of aspiration
- Gently hyperventilate the patient with low peak airway pressures for 3 min.
- Administer 1 mcg/kg of remifentanil and repeat it if needed as per the heart rate
- Quick gentle intubation in single attempt.

Management of severe head injury

- Use TIVA with propofol/remifentanil infusion
 Avoid volatile anesthetic until dura is opened(Ref Miller's 9th edition, 2020)
- Secure the endotracheal tube well
- Allow the surgeon to turn the bed to 90 or 180 degrees
- Administer antibiotics.

Management of severe head injury

Large bore IV

- Arterial line
- Do and ABG: Figure out PaCO2-end tidal CO2 difference and target end-tidal CO2
- Manage ventilation as per this end tidal CO2 target
- Do frequent ABG to figure out target CO2 based on above difference
- Maintain CPP 60-70 mmHg as per BTF guidelines.

Management of severe head injury

- Administer mannitol ıgm/kg over 15-20 min
 Administer Keppra 1 Gm IV over 1 hour.
- Post procedure transfer the patient to ICU, well sedated and with close monitoring of vital signs

How to reduce the ICP quickly when the dura is opened and bulging

- Discontinue volatile anesthetic agents
 Administer a bolus of propofol with phenylephrine
 Start TIVA as your primary anesthetic.
 Temporarily hyperventilate to 30-35 min until the
 - problem settles and then maintain normocarbia.

Neuromonitoring: EEG, Evoked potential monitoring

Volatile anesthetic agents sensitivity

VEP> MEP> SSEP> BAEP

Anesthetic agent and evoked potentials

Agent	Amplitude	Latency
Etomidate	Increases	increases
Ketamine	Increases	No change
Nitrous oxide	Decreases	No change

EEG

- Surgical anesthesia: Delta waves
- Opiates maximum effects: Delta waves
- EEG monitoring during neuroanesthesia: Target delta waves
- Dexmedetomidine max effect: alpha waves
 Avoid high dose of propofol infusion, use remifentanil and dexmedetomidine



Try to avoid volatile anesthetic agents

If you are using dexmedetomidine limit the infusion rate at 0.5mg/kg/hour

Ischemia monitoring on evoked potentials

Decrease in amplitude and increase in latency
Loss of evoked potentials

VAE updates

Updates on Venous air embolism(VAE)

A single center report: Mayo clinic report of extreme air embolism in sitting position from 2010-2013.

8 cases(6 suboccipital craniotomies, 2 DBS cases

Multiorifice CVP, TEE, Doppler used.

Cases were associated with hypotension and decrease in end-tidal Co2

Reference:

Abcejo AS, Pasternak JJ, Perkins WJ. Urgent Repositioning After Venous Air Embolism During Intracranial Surgery in the Seated Position: A Case Series. *J Neurosurg Anesthesiol* 2019; 31:413-421.

VAE updates

Recommendation: 1. Emergency position change to supine

2.Air aspiration through CVP; not successful in this case series.

3. Monitoring for embolism is indicated. Placement of CVP line to aspirate air is not indicated.

References:

- 1. Abcejo AS, Pasternak JJ, Perkins WJ. Urgent Repositioning After Venous Air Embolism During Intracranial Surgery in the Seated Position: A Case Series. *J Neurosurg Anesthesiol*. 2019;31:413-421
- 1. Pasternak JJ: Neuroanesthesia updates. J Neurosurg Anesthesiol 2020; 32; 97-119

AIS updates

Year	Title	Centers	number	Time	Reference
2015	MR CLEAN	16, Dutch	500	6	Ref28: N Engl J Med 2015; 372:11-20
2015	EXTEND IA	14, Australia, New Zealand	70	4.5	Ref 29: N Engl J Med 2015; 372: 1009-18
2015	ESCAPE	21, International	316	12	Ref 30:N Engl J Med 2015; 372:1019-30
2015	REVASCAT	4 Spanish centers	206	8	Ref 31: N Engl J Med 2015; 372:2296-2306
2015	SWIFT PRIME	39, international	196	6	Ref 32: N Engl J Med 2015; 372:2285-2295
					59

<u>tPA really necessary in AIS? Is it a</u> <u>placebo!!</u>

AIS: EVT vs EVT with tPA(combination therapy)

Multicenter RCT 41 tertiary academic centers(n=656)
Outcome measures: Reperfusion:before thrombectomy (2.4% vs 7.9%)
Successful reperfusion after thrombectomy: 79.4% vs 84.5%
Mortality 17.7% vs 18.8% at 90 days
Conclusion: Endovascular thrombectomy alone is non-inferior to with regard to functional outcome at 90 days.

Ref: N Engl J Med 2020; 382:1981-1993

AIS: EVT vs EVT with tPA(combination therapy)

	EVT alone(%)	EVT with tPA(%)
Reperfusion before EVT	2.4	7.9
Reperfusion after EVT	79.4	84.5
Mortality at 90 days	17.7	18.8

EVT 6-24 hours: DAWN trial

n=206 (107 thrombectomy group, 99 control group) (multicenter)

Outcome measure:

- Functional independence(mRS o-2) was better with thrombectomy group compared to standard of care (49% vs 13%)
- 2. 90 day mortality did not differ between the two groups(19% vs 18%)

Ref: N Engl J Med 2018; 378:11-21

EVT 6-24 hours: Diffuse 3 trial

Multicenter US trial (38 centers) was terminated after recruiting 182 patients(90 EVT group, 90 medical therapy group)

Outcome measures

- Functional outcome better with thrombectomy compared to medical therapy (45% Vs 17%)
- Mortality rate at 90 days was lower with thrombectomy group compared to medical therapy group(14% vs 26%)

Ref: N Engl J Med 2018; 378:708-718

GA Vs MAC for EVT: Does it matter in 2020?

GA vs MAC for EVT: Data from observational/retrospective studies

Observational trials and retrospective analysis reported better outcome with MAC.

Limitations:

- GA patients had worst underlying neurological status
- 2. Time to intervention was slower in GA group
- ^{3.} BP was lower in GA group.

Ref:

- 1.Am J Neurol 2019; 40-10011-5
- 2. Stroke 2015; 46:1257–1262
- 3. Am J Neuroradiol 2015; 36:525-529

GA vs MAC: Single center RCT's

- 1. JAMA 2016; 316:1986-96
- 2. Stroke 2017; 48:1601-7
- 3. JAMA Neurol 2018; 75:470-77

RCT	Year reported	Country	n
SIESTA(1)	2016	Germany	150
AnStroke (2)	2017	Sweden	90
Goliath(3)	2018	Denmark	128

RCT	MAC	GA
SIESTA	Not standardized	Not standardized
ANSTROKE	Propofol remifentanil	Propofol/remifentanil for induction Sevoflurane/remifentanil for maintenance
GOLIATH	propofol/Fentanyl	Propofol /alfentanil/ Succinylcholine for induction Propofol/remifentanil for maintenance

GA vs MAC: RCT data

A meta-analysis(reported in 2019) of three RCTs comparing GA vs MAC

SIESTA(Germany), ANSTROKE(Sweden), and GOLIATH(Denmark)

n= 365, large vessel occlusion in anterior circulation

Outcome measures: GA group had favourable functional independence at 90 days.

Ref:

- 1. Front Neurol. 2019;10:1131
- 2. J Am Heart Assoc 2019; 8e011754



- **n**= 88
- TIVA was used in both groups
- Conversion of sedation to GA: 9.52%
- (SIESTA 14.3%, Goliath 15.6%)
- No difference in functional outcome or mortality rate at 90 days

Ref: Frontiers in Neurology: doi.org/10.3389/fneuro2020.00170

GA vs MAC for EVT: What we should in 2020?

Choice of the technique depends on patients baseline neurological status as per 2019 ASA of AHA statement. **Stroke 2019; 50:e344-e418**

GA vs MAC for EVT in AIS: Ongoing trials

 AMETIS trial (Anesthesia Management in Endovascular Therapy for Acute Ischemic Stroke (n=270)

 SEGA (SEdation Versus General Anesthesia for Endovascular Therapy in Acute Ischemic Stroke)(n=270)

Ref:

- 1. BMJ open 2019;9:e027561 NCT 03229148
- 2. NCT 03263117

Elective Decompressive Craniectomy(DC)

Elective DC for TBI

Decompressive Craniectomy (DECRA, n= 155)(Ref 1)
 Craniectomy for Uncontrollable Elevation of ICP (RESCUEicp n=)(Ref 2)

Results;No benefit for early elective DC

Ref 1: N Engl J Med 2011; 364:1493-1502 Ref 2: N Engl J Med 2016; 375:1119-1130

DECRA: Elective DC for TBI

Diffuse TBI GCS 3-8 Standard Rx vs Standard Rx + DC n= 155, multicenter, 15 centers DC: Bifrontotemporal DC within 72 hours of randomization with a target ICP of 20 mm Hg (15 min period in hour period)

Results: DC group had worse neurological outcome and death rate at 6 months.

Ref: N Engl J Med 2011; 364:1493-1502

Rescue ICP trial: DC for TBI

Standard Rx Vs Standard rx+DC n= 408, multicenter RCT Target refractory persistent ICP 25 mmHg for 1-12 hours

Results:

Lower mortality, but higher vegetative state/ severe disability in survivors in DC group.

Good recovery and moderate disability was similar in both group.

Ref: N Engl J Med 2016; 375:1119-1130

Elective DC for TBI ?

Based on DECRA and Rescueicp trial: Elective DC is not indicated in severe diffuse TBI

Elective DC for AIS

- The Hemicraniectomy After Middle Cerebral Artery Infarction With Life-threatening Edema Trial [HAMLET]): A Multicentre, Open, Randomised Trial
- n-64, 32 DC and 32 best medical treatment
- MCA hemispheric infarction were included in the trial
- DC was done within 4 days
- Assessment: mRankin score at 1 year
- Results; Benefit of DC if done within 48 hours

DHC for MCA hemispheric stroke

- A meta-analysis reported in 2020
 9 RCTs including HEMLET, DECIMAL and DESTINY trials
- N= 425, 210=DHC, 215= best medical management

Results: DHC performed with 48 hours was associated with better survival rate and functional outcome.

Ref: *Bioscience Reports* 2020; 40 BSR 20191448 PMID 31854446

Deep Brain stimulation(DBS)

- GABAergic drugs midazolam should be avoided.
- Long acting opiates should be avoided.
- Dexmedetomidine 1 mcg/kg bolus followed by 0.2-0,7 mcg/kg/hr
 - Remifentanil 0.01-0.05 mcg/kg/min
- Propofol infusion, avoid bolus
- Stop everything for assessment.
- Keep systolic BP 120-140 mm Hg for electrode implantation and MAP above 70 mmHg.

Limit the fluids

Updates on Intracranial aneurysms

Clipping of aneurysms

During application of an temporary clip

Maintain MAP

Administering additional propofol is a common practice, but benefit of such practice is not proven(Ref 1)

Ref 1: Anesthesiology. 2010; 112:86-101

Optimum duration of temporary clipping

Common practice:

Optimum duration: 10 min for MCA and upto 20 min in other locations.

Optimum duration of temporary clipping; What is new?

A prospective observational trial(n=24)(13 ruptured and 11 unruptured MCA aneurysms)

MEP monitoring

Safe duration was 2.4 min with 95% confidence interval based on MEP changes

Ref: Surg Neurol Int 2017;8:79

Clipping of aneurysms Induced hypotension Clevidipine Adenosine: 0.3-0.4 mg/kg

Clipping of aneurysms

Adenosine precautions

Pacer pads

Prolonged effect in AV blocks, digoxin and verapamil

Contraindications: CAD, Severe asthma, AV block

Ref:

- 1.<u>Stroke Vasc Neurol</u>. 2017; 2:204–9
- 2. J Neurosurg 2018; 129:3
- 3. Stroke 2012; 43; Supp 1

Endovascular techniques for the treatment of aneurysms

Endovascular coiling
 Endovascular stent assisted coiling for larger aneurysms
 Endovascular flow diversion techniques

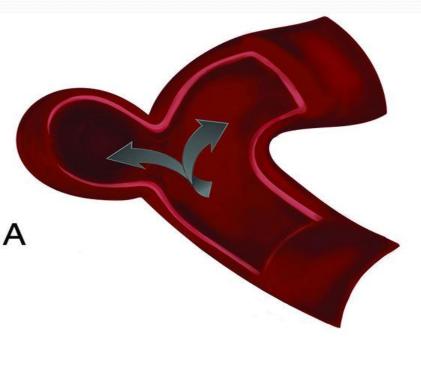
Endovascular Flow diversion: Updates

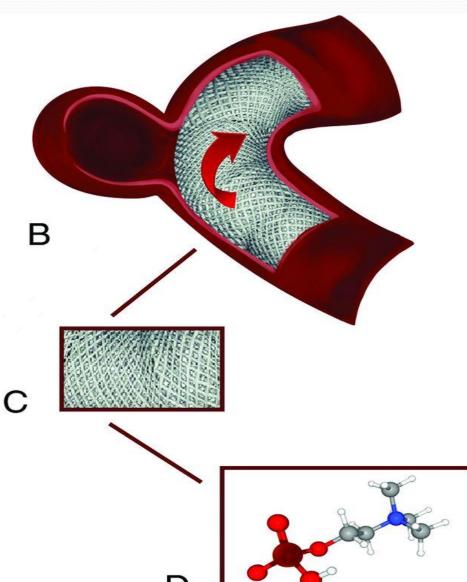
Traditional endoluminal flow diversion: Deploys the diverter within the lumen of the parent vessel at the neck of the aneurysm (PED, Silk, Surpass, FRED)

Intrasaccular Flow diversion: Deployment of self expanding new diverters within the aneurysm.(WEB, Medina, LUNA)

Ref: Neurosurgical Focus 2017; 42: 1

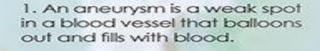
PED illustration from AJNR Am J Neuroradiol : 2019 www.ajnr.org





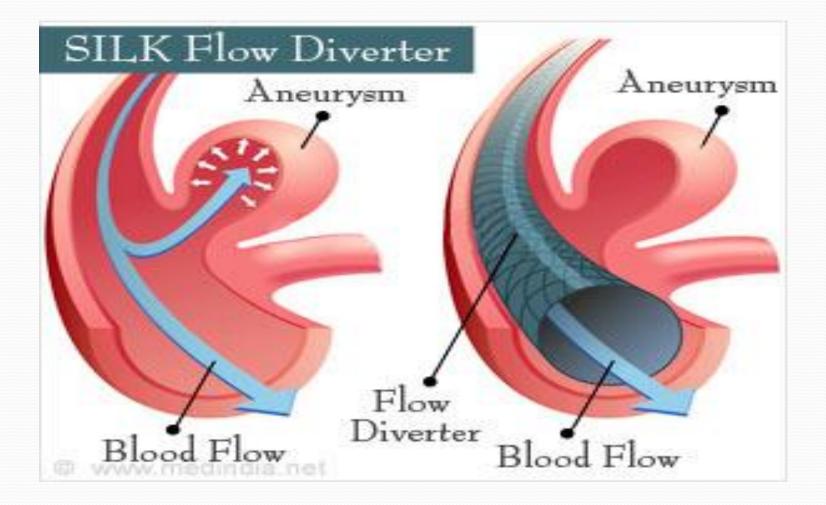
PED: Illustration from University of Iowa

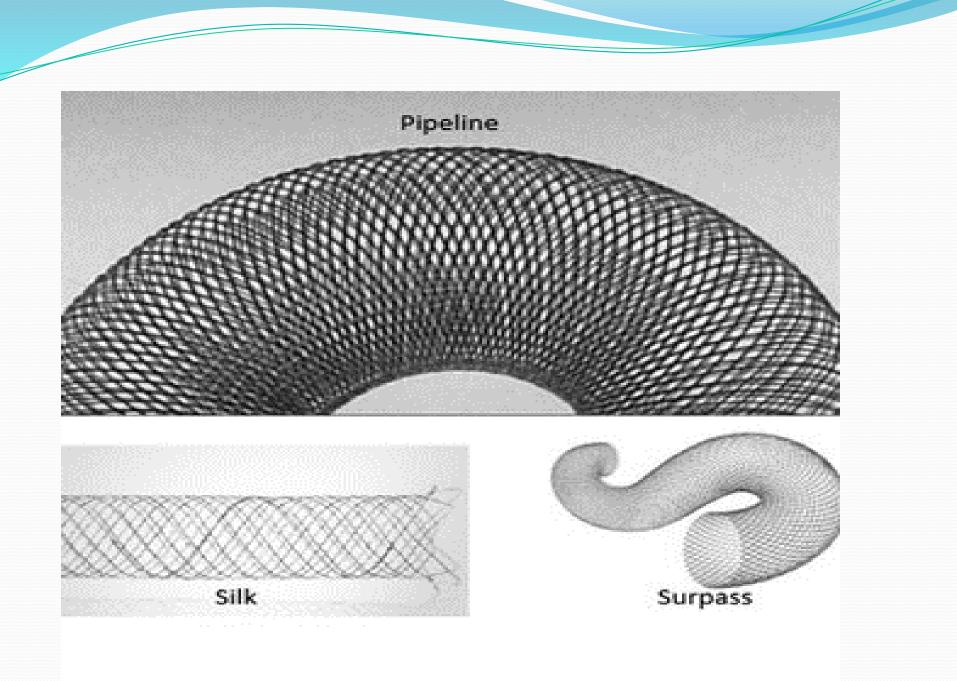
3.



2. The Pipeline Embolization Device prevents blood flow into the aneurysm, allowing the blood vessel to heal.

3. Over time, the aneurysm will shrink on its own.





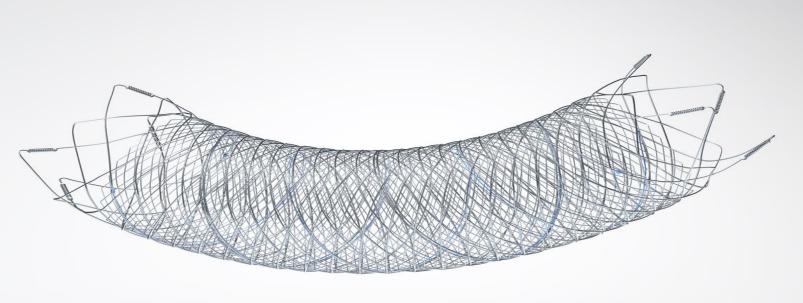
Ideal flow diverter

In order to achieve complete occlusion of the aneurysm ideal flow diverter should have

- 1. High metal content
- 2. Decreased porosity
- 3. Optimum porosity in order to prevent attenuation and occlusion of pores

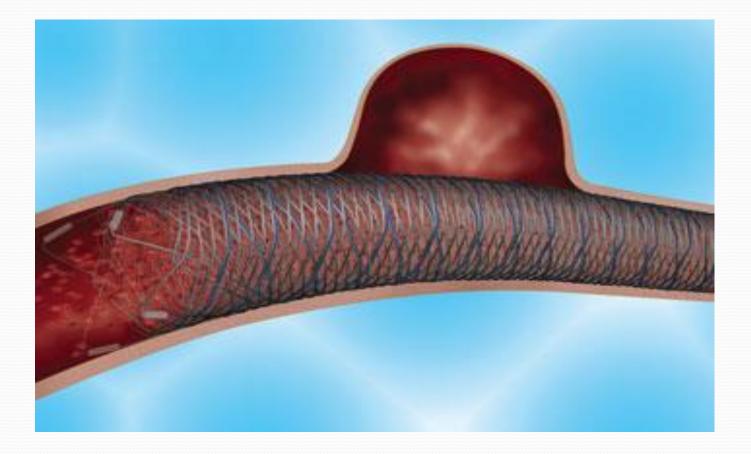
FRED dual layer flow diverters

The device consists of a braided self-expandable closed-cell dual-layer stent (also referred to a "stent within a stent")



FRED flow diverter

Illustration from UCLA



PED vs FRED

- Retrospective analysis
- 375 posterior circulation unruptured aneurysms in 369 patients.
- PED was used in 285 (77.2%) and FRED in 84 (22.8%) procedures
- Limitations: PED was used in larger and fusiform aneurysms.
- In Order to eliminate propensity score matching was used to compare 33 FRED and 33 PED aneurysms

Results: Occlusion rate and complication rate were similar in both groups.

Functional outcome was better in FRED group.

Ref: J Intervent Surg 2020<u>http://dx.doi.org/10.1136/neurintsurg-2020-016055</u>

Coiling vs Flow diversion

Endovascular coiling vs flow diversion: a meta-analysis 11 studies (10 retrospective, 1 prospective) n=611 coiling, 576 Flow diversion

Results: Flow diverters, compared to coiling had

- Higher rate of aneurysm occlusion(immediate and delayed)
- No difference in overall intraoperative complication rate
- No difference in functional outcome score measured on mRankin
- Flow diverts reduce cost and time of fluoroscopy per case

Ref: World Neurosurgery, 2019; 128: 464-72

Problems with endoluminal

flow diverters

- Dual Antiplatelet therapy is needed to prevent stent thrombosis.
 Clopidogrel typically started 5 days prior to intervention
- Aspirin 325 mg and Plavix continued post procedure until 6 months.

Problems with traditional Endoluminal Flow diverters

Stent thrombosis

Distant parenchymal hemorrhage

- Hemorrhagic transformation of AIS.
- Stent migration

Endoluminal flow diverters

A prospective observational trial from France with PED and Silk flow diverters recently reported in 2019 at 12 months (n=398 patients, 477 aneurysms): **Diversion study**. (Oct 2012- Oct 2014)

Satisfactory occlusion rate 80%
Major complication rate 5.6%
Mortality rate as 1.2%

Ref: Stroke. 2019;50:3471-3480

Flow diverters for intracranial aneurysm

A meta-analysis of 60 studies(3125) reported in 2017,

The neurological complication rate was 4.5%.
Mortality rate was 2.8%
No difference between PED and Silk flow diverters
Complication rate was higher in ruptured aneurysms in this analysis.(Odds ratio of 2.3 with 95% CI, 1.2-4.2)

Ref: Neurosurgical Focus 2017; 42:6

Intrasaccular WEB Flow diverter:

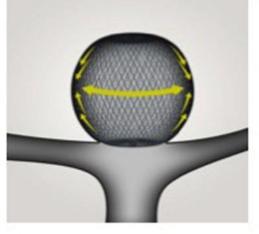
Illustration from Stroke and Vascular Neurology 2016;1:e000027





WEB-SL (Single Layer)

WEB SLS (Single Layer Spherical)



Radial compression holds WEB in aneurysm sac while conforming to aneurysm wall and sealing the neck



Endovascular Flow diversion: Updates

Advantages of intrasaccular flow diverters over intra luminal diveters

- Dual antiplatelet therapy not required
- Less risk hemorrhagic transformation of AIS from platelet therapy
- Less chance of stent thrombosis of flow diverter
- Less chance Flow diverter migration

Ref: Neurosurgical Focus 2017; 42: 1

DCI after aSAH: Pathophysiology

EBI: Loss of BBB, autoregulation Vasospasm CSD(Cortical spreading depression) DIC as part of SOFA(old terminology: (SIRS)

References:

1. J Clin Neurophysiol. 2016; 33 :196–202

2. Cereb Blood Flow Metab 2017; 37:1595-1625

DCI management

IAD (Intra-arterial vasodilator) therapy with vasodilators(milrinone etc) is beneficial as per a meta-analysis(n= 1561, 56 studies)

Immediate angiographic reversal(89%), improved outcome 66% and mortality 5%.

Ref: *J Neurointerv Surg* 2018;10:380-387.

DCI management

HHH part of the history now.

Prophylactic Induced was used until recently.

DCI management

RCT(n=240) was prematurely terminated after recruiting 42 patients : after observing no benefit.

Ref 1: Stroke 2018;49:76–83

DCI management:current concept

Prophylactic induced hypertension: Not indicated

Prophylactic oral nimodipine is indicated

 Early angioplasty and vasodilator therapy is indicated

Trigeminal vagal reflex

Temporal craniotomy Trigeminal neurolysis Jannetta procedure maxillofacial surgeries

Blood management: DCR

A Large vascular tumor: A Double edged sword!!

Goal: 1. Prevention of coagulopathy with blood management to prevent post op intracranial bleeding and hematoma.

- 2. Maintain CPP.
- 3. Decrease ICP.

4. Do not lower BP to prevent bleeding as in trauma DCR principle

DCR in Neurosurgery

Avoid large amount of crystalloids
Use blood/blood products
Consider TXA
Maintain CPP.

Elective surgery after AIS: How long we have to wait?

Elective surgery after AIS: How long we have to wait?

National registry analysis from Denmark 2005-2011, reported in 2014 n= 7137(with AIS)

n= 474046 (without AIS)

Risk of AIS and MI was 54.4% with prior AIS and 4.1% without prior AIS if the surgery was done within three months.

Ref: JAMA 2014;312:269-277

Elective surgery after AIS: How long we have to wait?

All elective surgery should be postponed for 9 months after Stroke as per

- 1. American College of Surgeons 2018 guidelines(Ref 1)
- 2020 Guidelines From the Society for Neuroscience in Anesthesiology and Critical Care (Ref 2)

Ref:

- 1. J Am Coll Surg 2018; 227:521-36
- 2. J Neurosurg Anesthesiol 2020; 32: 210-26