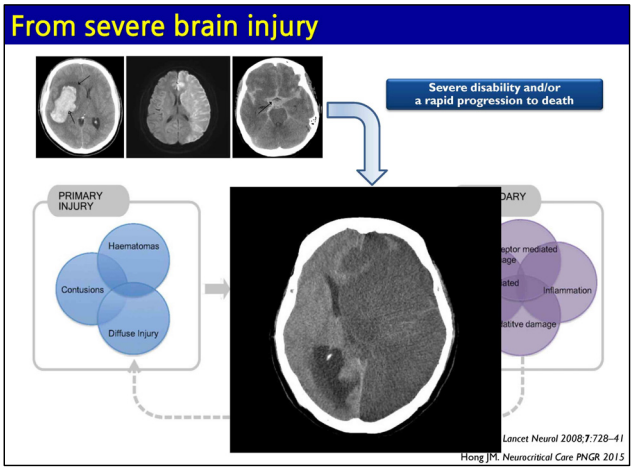
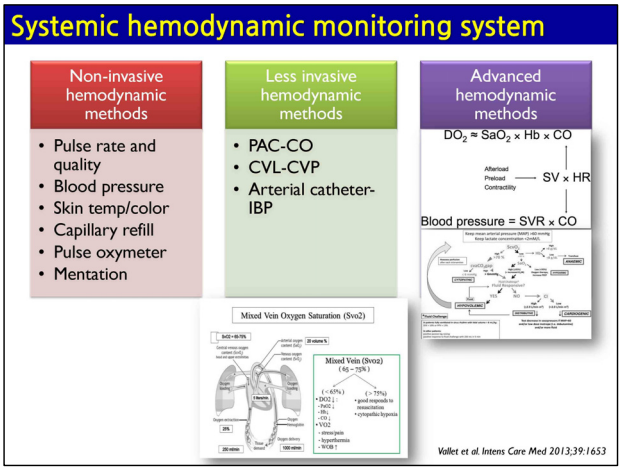
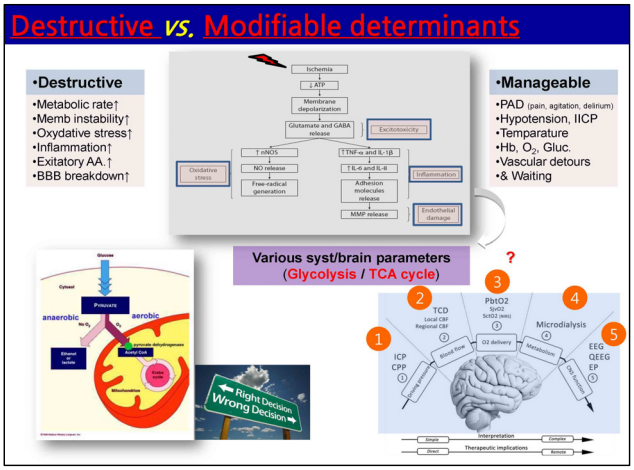
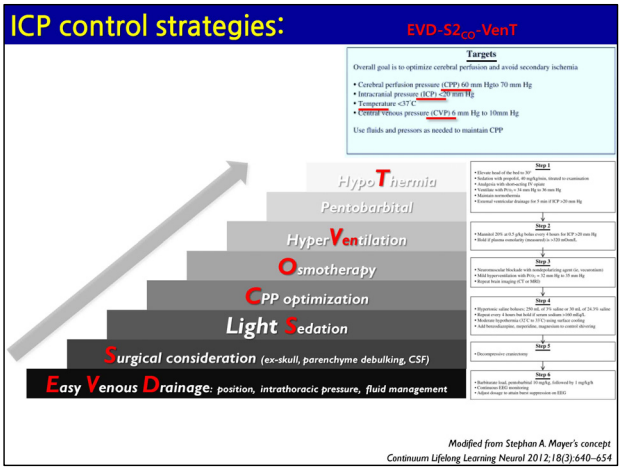
Minimally-Invasive Neuromonitoring in NCC

홍 지 만
아주대학교병원 신경과

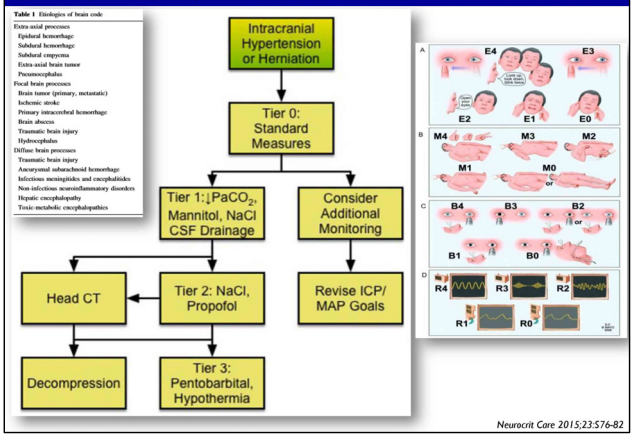






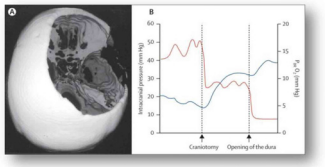


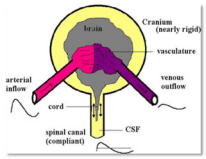
ENLS (intracranial hypertension or herniation protocol)



Neurocrit Care 2015;23:S76-82

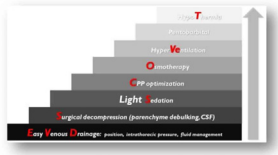
Early surgical decompression





Combination of the respective methods!!

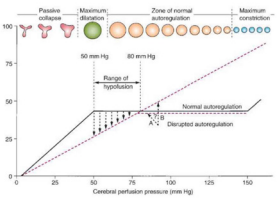
- EVD, easy venous drainage
- Early lumbar drainage in special cases
- Sedation
- CPP optimization
- Osmotherapy
- Hyperventilation
- Hypothermia



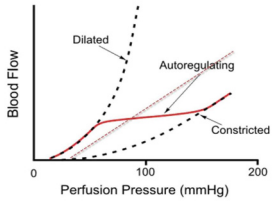
Modified from Stephan A. Mayer's Concept

Cerebrovascular autoregulation

: Intrinsic ability of an organ (especially the brain) to maintain a constant blood flow despite changes in perfusion pressure



Under the laws of Ohm and Poiseuille: rigid pipe vs. elastic bio-tube



Ginsberg MD, Bogousslavsky J. Cambridge, MA: Blackwell Science, 1998: 319–332.

Autoregulation & CPP optimization

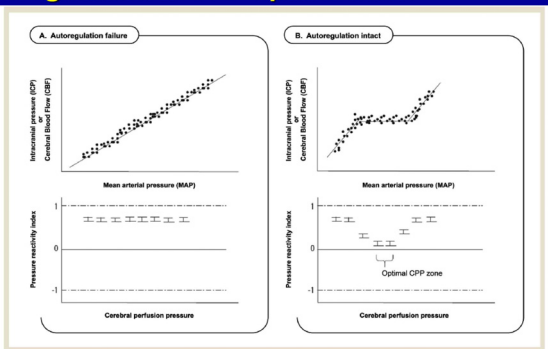
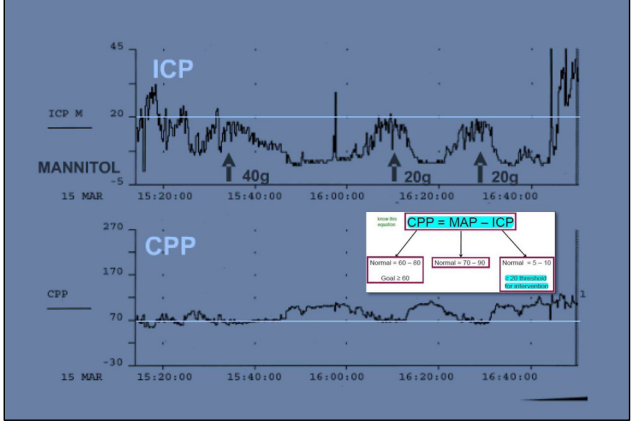


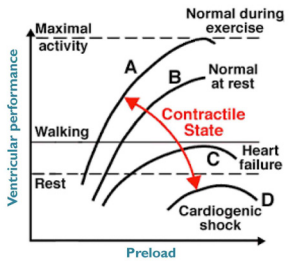
그림 3. 손상된 대뇌 혈류자동조절기능 vs. 정상인 대뇌 혈류자동조절기능.
(A) 혈압의 변동에 대해 뇌혈류 혹은 뇌압의 변동이 같이 일어난다. 따라서 압력 변동비(PRx)가 혈압변동에 대해 일정하다.
(B) 혈압의 변동에 대해 뇌혈류 혹은 뇌압의 변동이 적게 일어나며 일정구간에서 가장 낮은 대뇌 관류압(cerebral perfusion pressure, CPP) 지점을 나타내는 구역이 있다 (CPP optimization).

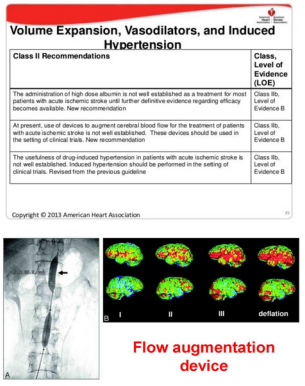
Neurocritical Care PNGR 2015

Cerebral autoregulation: Association with system & neuro



Frank-Starling curve & cerebral flow driving

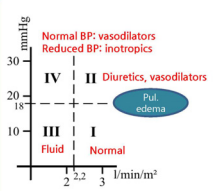




Flow augmentation device

Therapeutic interventions

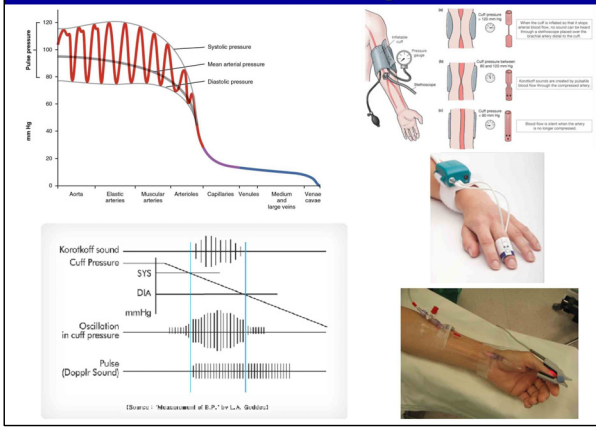
- Therefore, optimizing cardiac output
- Optimize HR and SV (preload, afterload, contractility)
  1. Chronotopic medications
  2. Diuretics/volume
  3. Vasodilators/vasocontrictors
  4. Inotropic factors and medications:
     positive (glucagon, Epi, T3, digitalis) VS. negative (acidosis, rising K^+, Ca^{++} b)
- ✓ IABP & non-invasive counter pulsation



Low status	Performances	High status	Variables
• Atropine • Pacemakers	Heart rate	• Betablockers • Calcium blockers	• HR
• Fluids	Preload (filling pressure & volume)	• Diuretics • Venodilators	• CVP, PAO(W)P
• Vasopressors	Afterload (resistance to outflow)	• Arterial dilators • Calcium blockers • ACE inhibitors	• SVR, PVR, MAP
• Inotropics	Contractility (strength of contraction)		• RVSV, LVSV

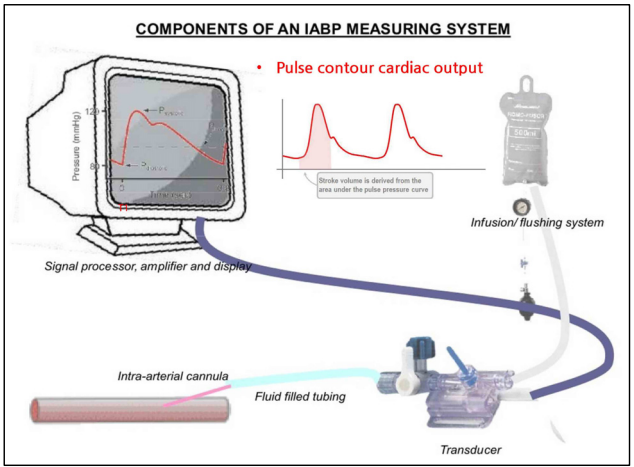
Forrester etl al. Am J Cardiol 1977;39:137

A-Line and arterial monitoring



Indications (A-line)

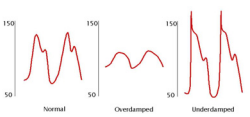
- Continuous, beat-to-beat blood pressure measurement
  - Hemodynamically unstable patients
  - ICU patients requiring inotropic support
  - Neurocritical patients
- Frequent arterial blood gas analysis
  - Patients with respiratory failure on ventilator
  - Severe acid/base disturbance
- Radial artery has low complication rates, but the ulnar, brachial, axillary, dorsalis pedis, posterior tibial, femoral arteries are alternatives



Mechanism of action

- The column of saline in the A-line tansmits the pressure changes to the diaphragm in the transducer
- The transducer: at level of 4th intercostal space, at the mid-axillary line (phlebostatic axis)

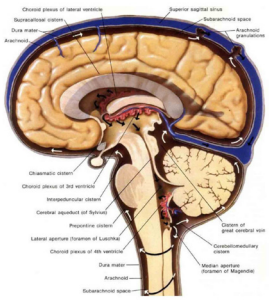
- Zeroing
  - Flush the device and turn it off to patients but open to atmosphere
- Damping due to
  - Air bubbles
  - Catheter kinks
  - Clots
  - Injection ports
  - Low flush bag pressure or no fluid
  - Improper scaling
  - Severe hypotension if everything else is ruled out
- Resonant due to
  - Long tubing
  - Overly stiff, non-compliant tubing
  - Increased vascular resistance
  - High systolic and low diastolic
  - Non-fully opened stopcock valve



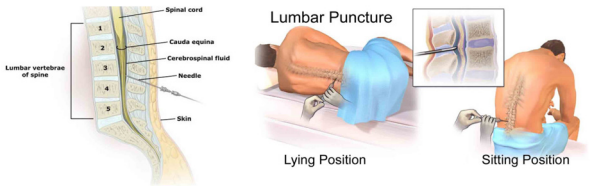
- A-line BP: 5 mmHg higher in systolic BP and 8mmHg lower in diastolic BP

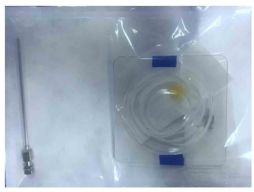
Lumbar drainage (LD) & anatomical basis

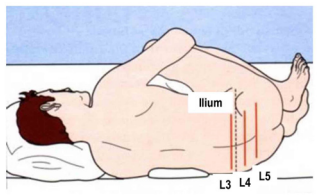
- Simple and less invasive bedside technique
- Reducing EVD duration and avoiding EVD exchange
- Decrease of development of vasospasm after SAH
- Diminish of permanent hydrocephalus after ICH with severe ventricular involvement
- Theoretically, LD-ICP = EVD-ICP
- Drainage in acute status?



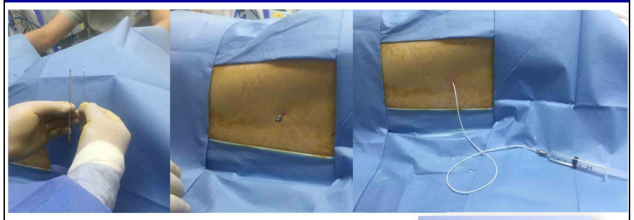
Lumbar drainage (LD) - approach







Placement



- **Complications**
  - Over-drainage
  - Infection at insertion site
  - Tube dislodgement
  - Lower extremity weakness
  - Changes in bowel and bladder patterns
  - Tracking in tubing

Malignant ICP by controlled CSF LD

Therapy of malignant intracranial hypertension by controlled lumbar cerebrospinal fluid drainage

Münch, Elke C, MD; Bauhuf, Christian MD; Horn, Peter MD; Roth, Harry R, MD; Schmiedek, Peter MD; Vajkoczy, Peter MD

Abstract

Objectives: To evaluate the effect of controlled lumbar cerebrospinal fluid drainage in adult patients with refractory intracranial hypertension.

Design: Prospective, pre- vs. postintervention study.

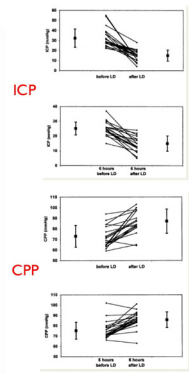
Setting: Surgical intensive care unit of a university hospital.

Patients: Twenty-three patients with severe traumatic brain injury or delayed ischemia after subarachnoid hemorrhage with intracranial hypertension refractory to aggressive treatment, including repeated applications of tromethamine, hypertonic saline solution, barbiturate coma, and decompressive craniectomy. Patients were considered for controlled lumbar cerebrospinal fluid drainage if basal cisterns on computerized tomography scan were discernible.

Interventions: After institution of a lumbar drain, cerebrospinal fluid was gradually aspirated, and then, continuous cerebrospinal fluid drainage was maintained under control of intracranial pressure (ICP) and pupillary status.

Measurements and Main Results: ICP and cerebral perfusion pressure before and after initiation of lumbar cerebrospinal fluid drainage and related complications were documented. The neurologic outcome of the patients was assessed according to the Glasgow Outcome Scale 6 months after injury. As a result of lumbar cerebrospinal fluid drainage, all patients demonstrated an immediate and lasting decrease of ICP and a concomitant increase of cerebral perfusion pressure. Two patients temporarily showed a unilateral fixed and dilated pupil 6 and 8 hrs after onset of lumbar cerebrospinal fluid drainage, respectively. Ten patients showed a favorable outcome, four patients survived with a severe permanent neurologic deficit, one patient remained in a persistent vegetative state, and eight patients died.

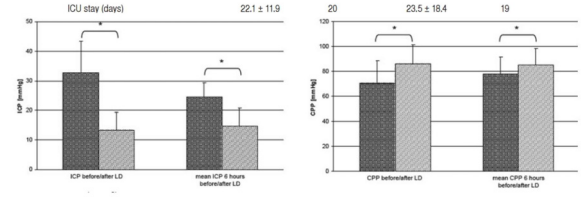
Conclusions: Controlled lumbar cerebrospinal fluid drainage significantly reduces refractory intracranial hypertension. The danger of transtentorial or tonsillar herniation is minimized by considering lumbar drainage in the presence of discernible basilar cisterns only.



Crit Care Med 2001;29:976-981

Refractory ICP by controlled CSF LD

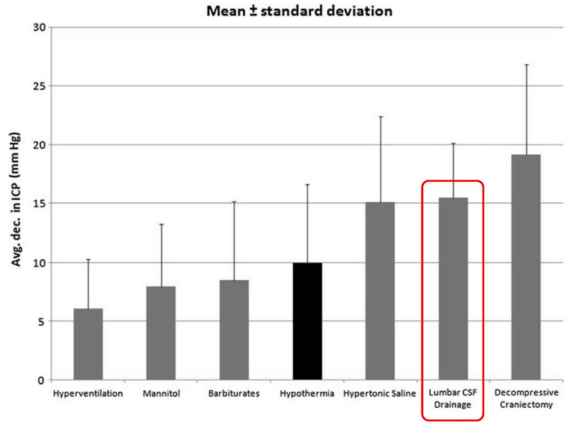
Parameter	TBI Mean ± SD	TBI Median	SAH Mean ± SD	SAH Median
age (yrs)	37.6 ± 18.1	35	48.4 ± 11.8†	49
GCS score	6.4 ± 3	6	7.5 ± 3	5
ICU stay (days)	22.1 ± 11.9	20	23.5 ± 18.4	19



directly before LD	64.3 ± 16.5	65	75.5 ± 18.1	76
directly after LD	80 ± 9.2	79	91.1 ± 17.5	88
6 hrs before LD	71.1 ± 10.2	71	83.3 ± 13.9	80
6 hrs after LD	79 ± 8.3	78.5	89.8 ± 14.7	89.5

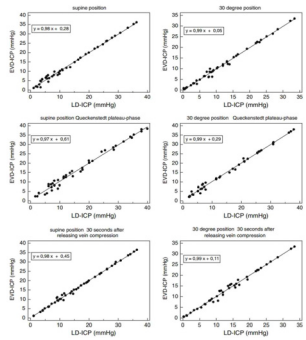
Open and well-defined basal cisterns (+)

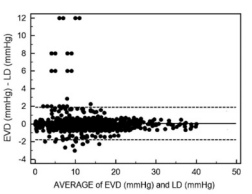
J Neurosurg 2009;110(6):1200-8



Neurocrit Care 2009;11(3):427-36

LD for ICP in communicating hydrocephalus



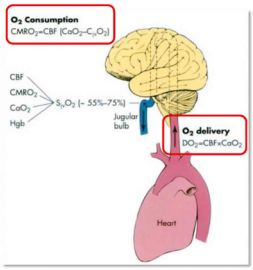


- Most feared complications – downward herniation
- Avoid
  - > 20mmHg
  - Midline shift (+)

Neurocrit Care 2011;14:208–215

Cerebral oxygenation and physiology

Balance of O_2 supply and consumption of the brain
= Balance of O_2 delivery and metabolism of the brain



Oxygen dynamics

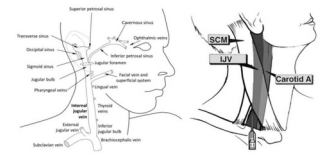
- O_2 delivery (DO_2) = (CO_2) x CO x 10
- Arterial O_2 content (CO_2) = [(Hb x 1.34 x SaO_2) + (PaO_2 x 0.0031)]
- Jugular venous O_2 content (CO_2) = [(Hb x 1.34 x $SjvO_2$) + (PvO_2 x 0.0031)]
- $CMRO_2$ (VO_2) = CBF x (SaO_2 – $SjvO_2$)
- $CMRO_2$ (VO_2) = CBF x (A – V)DO_2
- Oxygen extraction ratio (O_2ER) = VO_2 /DO_2

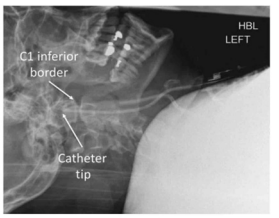
→ When $CMRO_2$ is constant, any change in CBF is associated with a reciprocal change in the cerebral (A – V) DO_2

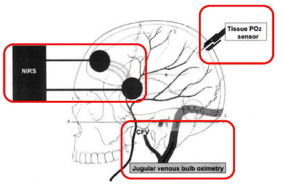
- (A – V) DO_2 = ($CMRO_2$/CBF)

Monitoring in Neurocritical Care 2013

Jugular bulb venous oxygen saturation







- Global venous Oximetry
  - SvO_2 – mixed venous oximetry
- Regional venous Oximetry
  - $ScvO_2$ – head and upper extr.
- Organ specific venous Oximetry
  - $SjvO_2$ – cranial jugular bulb oximetry
  - $ShvO_2$ – hepatic venous oximetry
  - $ScsO_2$ – coronary sinus venous oximetry

Calculations of Oxygenation

- ABGA (Ph: 7.34, PaO_2: 293, O_2Sat: 100%, HCO3: 21 ...), Hb: 8.5 mg/dL
- Arterial O_2 content = CaO_2 = 8.5(1.34)1 + 0.003(293)
  = 11.39 + 0.88
  = 12.27 ml O_2/dL (O_2%)
- Venous O_2 content = CvO_2 = 8.5(1.34)(.76) + 0.003(45)
  = 8.66 + 0.14
  = 8.67 ml O_2 /dL
- A-V O_2 difference = Ca-v O_2 = 12.27 - 8.67 = 3.6 ml O_2 /dL

Category	Normal range
Content (CaO2)	16-19 ml O2%
Delivery (DO2)	520-720 ml O2/min/m2
Consumption (VO2)	110-160 ml O2/min/m2
Extraction (ER)	22-32 %
Mixed venous pO2	33-53 mmHg
Mixed venous saturation	68-77 %

Jugular venous oximetry

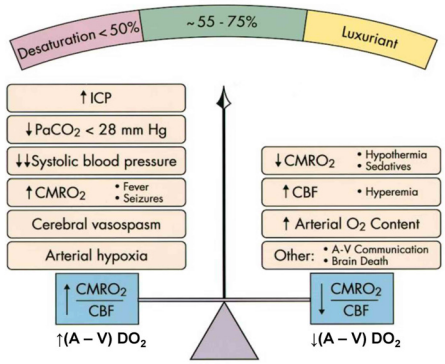
$SjvO_2$	Normal Hyperemia Low flow	60 – 80% > 80% < 50%
(A – V) DO_2 = ($CMRO_2$/CBF)	Normal Hyperemia (CBF > $CMRO_2$) Low flow (CBF < $CMRO_2$)	5 – 7.5 vol % < 5 vol % > 7.5 vol %
Cerebral O_2ER	Normal Hyperemia Low flow	24 - 40 % < 24 % > 40 %

Interpretation of $SjVO_2$

Factors increasing VO_2	Factors decreasing VO_2
· Surgery · Trauma · Burns · Inflammation · Sepsis · Pyrexia · Shivering · Seizures · Agitation/anxiety/pain · Adrenergic drugs · Weaning from ventilation	· Sedation/analgesics · Muscle paralysis · Shock/hypovolaemia · Hypothermia/cooling · Mechanical ventilation · Antipyretics · Starvation/hyponutrition

Neurocritical Care PNGR 2015

Interpretation of $SjVO_2$



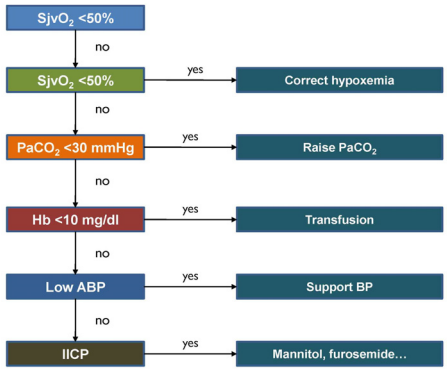
Anesth Analg 2000;90:559-66

Limitations of Jugular Venous Oximetry

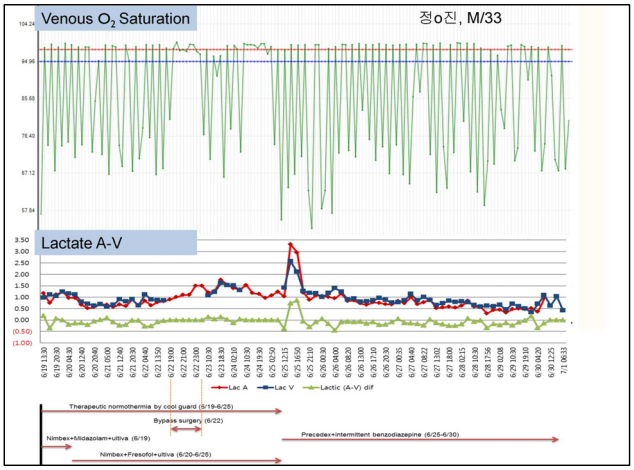
Limitation	Rationale	Management
Incomplete mixing	Asymmetric venous drain	Cannulate dominant side (usually right)
Extracerebral contamination	3% of jugular blood is contaminated from scalp, meninges, and skull	Radiograph confirmation Catheter tip: lower border of C1 and withdraw sample slowly
Bohr effect	Falsely high due to alkaline condition	Detect by measuring low jugular bulb PCO_2 (<27 mmHg)
Global measure	It may not provide information about regional injury	Measurement of arteriovenous lactate: anearobic metabolism!
Insensitive to infratentorial flow	Brainstem and cerebellum contribute little to the venous outflow from the brain	Limited value from brainstem injuries
Monitoring errors	Occlusion and fibrin formation at tip	Repositioning Recalibration, heparinized saline flush

Anesth Analg 2000;90:559-66

Suggested algorithm for $SjVO_2$

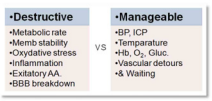


Monitoring in Neurocritical Care 2013

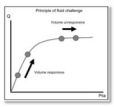


Conclusions: Minimally-invasive Neuromonitoring

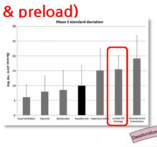
- Destructive and manageable components



- A-line and systemic driving force
  - Frank-Starling curve (stroke volume & preload)



- Lumbar drainage
  - Theoretically, LD-ICP = EVD-ICP



- Jugular bulb venous oxygen saturation & physiology
  - O_2 delivery & consumption