Cerebral hemodynamics

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Cerebral Blood Flow...
CBF = CmrO2 / (CaO2 - CvO2)
CBF = CPP / CVR

Cerebral Blood Flux...

Cerebral Blood Supply...

Cerebral \textit{perfusion}...

Cerebral arterial \textit{perfusion}...

Cerebral arterio-venous \textit{perfusion}...
CBF heterogeneity
Difference regarding the sensibility to the hypoxia and the ischemia
Peripheral compartment
Convexity and cerebral lobes (grey matter)

Central compartment
Circle of Willis
Anterior high Posterio low perfusion pressure perfusion pressure compartment compartment (carotid supply) (basilar trunk supply)
Cerebral Blood Flow in Summary...

Cerebral Blood Flow **Infant**
25mL.100g⁻¹.min⁻¹ (20-40)

Cerebral Blood Flow **Child**
100mL.100g⁻¹.min⁻¹ (75-110)

Cerebral Blood Flow
50mL.100g⁻¹.min⁻¹ (40-70)
750mL.min⁻¹ [10-15% Qc]

**Neural dysfunction threshold**
25mL.100g⁻¹.min⁻¹

**Electrogenesis suppression threshold**
10-15mL.100g⁻¹.min⁻¹

**Irreversible neuronal death threshold**
10mL.100g⁻¹.min⁻¹

- Temporo-sylvian cortex
  80mL.100g⁻¹.min⁻¹

- Fronto-latero-dorsal cortex
  65mL.100g⁻¹.min⁻¹

- White matter
  20-30mL.100g⁻¹.min⁻¹

- Thalamus
  85mL.100g⁻¹.min⁻¹
Two perconceived ideas:
- No energetic reserve **YES**
- Relative constant blood flow **NO**
Cerebral Blood Flow...

CBF = CmrO₂ / (CaO₂ - CvO₂)

CBF = CPP / CVR

Ohm’s law: CBF = ΔP / R

ΔP (CPP) = MAP – CVP

R = arteriolar low vascular résistances

50 mL.100g⁻¹.min⁻¹
Cerebellar CBF values determined by FAIR ASST were $43.8 \pm 5.1 \text{mL/100 g/min}$ for GM and $27.6 \pm 4.5 \text{mL/100 g/min}$ for WM. Quantitative perfusion studies indicated that CBF in cerebellum GM is 1.6 times greater than that in cerebellum WM.
Vascularisation Cérébrale
Neural dysfunction thresholds...

**EEG**
- BSR=0
- BSR>90

**Thresholds**
- Normal neuronal function
- Progressive depression neuronal function
- Abolished neuronal function
- Cancelled autoregulation
- BBB Opening
- Neuronal death

**Cerebral Blood Flow (mL.100g⁻¹.l⁻¹)**
- Normal Brain
  - Normal neuronal function
- Pellucida zone
  - Reversible neuronal lesion
  - Only electro-chemical dysfunction
- Penumbra
  - Simple neuronal necrosis
- Infarctus
  - Non-reversible neuronal lesion

**Ischemia duration (heures)**
- Viable tissue
- Definitive lesion
Cerebral Blood Flow Regulation

Humoral and chemical
($H^+$, $K^+$, $Ca^{2+}$, adenosine, osmolarity...)

Automatic, sympathetic nerve system (auto-regulation)

Nervous extrinsic
(superior cervical ganglion, sphenopalatine & otical ganglions, peptidergic fibers...)
Cerebral Blood Flow Regulation

Cerebral vascular autoregulation

CO2 Vasoreactivity

Metabolic coupling
Cerebral Blood Flow Control
Cerebral Autoregulation

CPP = MAP – CerebralVP
CPP = MAP – ICP
CPP = MAP – CVP [CVP>ICP]
CPP_{adult} = 80-100 mmHg
CPP_{pediа} = 65-85 mmHg

CBF (mL.100g^{-1}.min^{-1})

MAP (mmHg)

CPP (mmHg)
Pressure regulation

CEREBROVASCULAR RESERVE

Graph showing the relationship between Cerebral Blood Flow (ml/100g/min) and Arterial Blood Pressure (mmHg). The graph illustrates the concept of cerebrovascular reserve, where at certain blood pressures, the cerebral blood volume compartment dilates to maintain blood flow under varying pressures.
« Non-pressure » regulation
CBF oxygen reactivity

Haggendal, Acta Physiol Scand 1965

![Graph showing CBF oxygen reactivity](image)
Cerebral Blood Flow Control
Cerebral Autoregulation

Physiological mechanisms
- Myogenic (rapide but inaccurate)
  - Metabolic (NO, adenosine, prostaglandines – accurate but delayed)
  - Neurogenic (accurate but delayed)

Physiopathology
- Arterial hypertension (reversible right shift)

Therapeutic application
- Controlled hypertension (Rosner J et al Trauma 1990, J Neurosurg 1995)

Anesthesia
- Halogenated: dose-dependante alteration
  - Intravenous: conservation
  - Concept of MAP maintenance
Cerebral Blood Flow Control
CO2 vasoreactivity

CBF changes 2 to 3% for each mmHg of PaCO2

Physiological mechanism - maximum 6-8 hours
- Preliminary extracel. & CSF H+ $\searrow$ Secondary HCO3$^-$ reaction

Physiopathology
- Ischemia threshold \( \text{PaCO}_2 \) 30mmHg?
(PaCO2 20mmHg = flat / suppressed EEG)

Therapeutic applications
Rapid & simple for decreasing CBF in neuroanesthesia et neuroICU
THE EFFECTS OF ALTERED ARTERIAL TENSIONS OF CARBON DIOXIDE AND OXYGEN ON CEREBRAL BLOOD FLOW AND CEREBRAL OXYGEN CONSUMPTION OF NORMAL YOUNG MEN

By Seymour S. Kety and Carl F. Schmidt

(From the Department of Pharmacology, University of Pennsylvania, Philadelphia)

(Received for publication December 4, 1947)
CO2 vasoreactivity

A. NORMAL

CEREBRAL ARTERIOLE

ARTERIAL
pH 7.40
PCO₂ 40
HCO₃⁻ 24

EXTRACELLULAR
pH 7.28
PCO₂ 45
HCO₃⁻ 18

B. ACUTE HYPERVENTILATION

ARTERIAL
pH 7.57
PCO₂ 25
HCO₃⁻ 22

EXTRACELLULAR
pH 7.47
PCO₂ 30
HCO₃⁻ 16

C. 6-12 HOURS OF HYPERVENTILATION

ARTERIAL
pH 7.53
PCO₂ 25
HCO₃⁻ 20

EXTRACELLULAR
pH 7.32
PCO₂ 30
HCO₃⁻ 13

D. ACUTE NORMALIZATION OF CO₂

ARTERIAL
pH 7.36
PCO₂ 40
HCO₃⁻ 22

EXTRACELLULAR
pH 7.21
PCO₂ 45
HCO₃⁻ 14
Oxygen/CO2 vasoreactivity relationship
Cerebral Blood Flow Control
In practice

- CBF (mL.100g⁻¹.min⁻¹)
- PAM (mmHg)
- PPC (mmHg)
- PaCO₂ (mmHg)
- PaO₂ (mmHg)

CPP (PAM) = PaO₂ - PaCO₂

Normal vasodilation and vasoconstriction

Ø arteriolar
Vasodilation Vasoconstriction

PaCO₂ 2 (mmHg) range:
- Normal: 10 - 85 mmHg
- Adulte: 160 mmHg

PaO₂ 2 (mmHg) range:
- Normal: 20 - 125 mmHg
- Adulte: 130 mmHg

PAM (mmHg) range:
- Normal: 60 - 160 mmHg
- Adulte: 50 - 130 mmHg

PPC (mmHg) range:
- Normal: 50 - 130 mmHg
- Adulte: 130 - 160 mmHg
Cerebral Blood Flow Control
Metabolic coupling

Physiological mechanism
- $\Delta 1^\circ C = \Delta 5-7\%$ DSC

Physiopathology
- Hyperthermia
- Seizure & convulsions
  (coupling rupture by intercurrent vasodilation,
   $\uparrow$ CBF et ICP)

Therapeutic applications
- Mild hypothermia (34-35$^\circ$)
  (coupling maintenance or recovery,
   $\downarrow$ CBF et ICP)

Anesthesia
- Barbiturates
- Others
  (coupling maintenance, $\downarrow$ metabolism
   & $\downarrow$ CBF et ICP)
Cerebral Blood Flow Regulation

**Triple control:**
- Autorégulation
- CO2 vasoreactivity
- Metabolic coupling

**Distributive large caliber proximal arteries**

**Arteriolo-capillary Smaller caliber Distal arteries To be seen...**
**Intrinsic property** of smooth muscle to respond to changes in mechanical load or intravascular pressure

*Bayliss, J Physiol 1902*

Exists in arteries and arterioles **denuded of endothelium** and in sympathetically denervated animals

*Busija, Rev Physiol Biochem Pharmacol 1984*

**Ca\(^{2+}\)** and **Cl\(^{-}\) channels** / **K\(^{+}\)\(\text{Ca}\) channels

*Osol, Am J Physiol Heart Circ Physiol 2002*
Endothelial biochemical regulation

- Nitric Oxide
- PGI₂
- Endothelium-Derived Hyperpolarizing Factor
Large and small pial arteries

**SCG** = superior cervical ganglia  
**SPG** = sphenopalatine ganglion  
**OG** = otic ganglion  
**TG** = trigeminal ganglion

Rostral vs. caudal increase

Reactivity to NE is greater in ACA compared to MCA (cats)
Metabolic regulation

High levels of CBF
Cochlear nucleus, mammillary body, cortex

Low levels of CBF
Hypothalamus, cerebellum, medulla

Edvinsson, 1993
Metabolic regulation
Dependence to the metabolic threshold

Cerebral blood flow during exercise: mechanisms of regulation

20% of maximal oxygen uptake
40% of maximal oxygen uptake
Other influences on Cerebral Blood Flow

Partial pression of oxygen – PaO2

Temperature

Hematocrit
Multimodal regulation / reactivity

- **Myogenic Regulation**
- **Metabolic Regulation**
- **Endothelium**
- **Neural Regulation**
  - Extrinsic
  - Intrinsic
- **Extrinsic**
  - Carotid sinus, aortic arch (Baroreceptors)
- **Intrinsic**
  - Brain Stem Chemoreceptors
- **MAP**
  - MAP-based Regulation
- **NO**
- **L-arg**
- **Adenosine**
- **Chemical Regulation**
  - Brain metabolism
  - Brain activity
  - PaCO₂
  - Perivascular pH
  - Adenosine
- **H⁺**
- **K⁺**
- **VI P**
- **Brain activity**
Multimodal regulation / reactivity

- PaCO₂
- ABP
- CPP
- Intracranial compliance
- CBF
- CBV
- CSF balance
- Sympathetic
- Metabolic demand
- O₂ supply
- O₂ flow
- Venous flow

ASSESS
Original dilution method Kety & Schmidt (1945)
Inhalation – dilution of N20
Arterio-venous difference during a 30min long period

Scintigraphy Xe 133
Especially regarding the regional cerebral blood flow

Positron Emission Tomography
$O_{15}$
Global regional & vascular territories parameters:

- **Maximum time** \((T_{\text{max}}, \text{scdes vs minutes})\)
- **Peak to time** \((\text{scdes vs minutes})\)
- **Mean transient time to peak** \((\text{scdes vs minutes})\)
- **Cerebral Blood Volume** \((\text{ml}.100\text{g}^{-1})\)
- **Cerebral Blood Flow** \((\text{Cerebral Blood Flow, ml.min}^{-1}.100\text{g}^{-1})\)
But in the real live, at the patient’s bedside...
Cerebral blood flow assessment at the patient’s bedside...

Tissular thermo-dilution (thermo-dilution probe)

- Minimal invasive
- Continuous / Monitoring
- Regional Cerebral Blood Flow

Neurosonology

- Transcranial Doppler
- Transcranial Duplex
- Transcranial Triplex
- Tissular microDoppler

Non invasive

- Continuous vs discontinuous
- Time-to-time exam vs monitoring
- Regional Cerebral Blood Flow

Tissular oximetry

- Cerebral oximetry (rSO2, SctO2)

Non invasive

- Regional Cerebral Blood Flow
In 1956, Clark described the principles of an electrode that could measure oxygen tension (polarography) in blood or tissue.

7-15mm³
The Bowman perfusion monitor connected to the Qflow probe to monitor continuously the rCBF in absolute units (ml.100g⁻¹.min⁻¹) and temperature.

Insertion 2.5cm below the dura in the white matter and CT-Scan checked.
The Bowman perfusion monitor connected to the Qflow probe to monitor continuously the rCBF in absolute units ($\text{ml.100g}^{-1}.\text{min}^{-1}$) and temperature.

Insertion 2.5cm below the dura in the white matter and CT-Scan checked.
Regional tissular CBF – Early validation

Continuous monitoring of regional cerebral blood flow: experimental and clinical validation of a novel thermal diffusion microprobe

Peter Vajkoczy, M.D., Harry Roth, M.D., Peter Horn, M.D., Thomas Lucke, M.D., Claudius Thome, M.D., Ulrich Hubner, Gregory T. Martin, Ph.D., Christina Zappetal, M.D., Ernst Klar, M.D., Lothar Schilling, M.D., and Peter Schmiedek, M.D.


n=16 adult farm-bred sheep
Mécanisme de l'Amnésie induite par l'Anesthésie

Multimodality:
- Spherical rCBF measurement at the catheter tip
- Recommendation = close to ICP or as much as possible (systematically) combined to PbtO₂/ICP catheter
Regional tissular CBF – integration in practice

Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury

Clinical article

Guy Rosenthal, M.D.,1,3 Rene O. Sanchez-Mejia, M.D.,1 Nicolas Phan, M.D.,1 J. Claude Hemphill III, M.D.,3 Christine Martin, R.N., M.S., C.N.S.,1 and Geoffrey T. Manley, M.D., Ph.D.1

Hyperventilation Challenge
Cerebral CO2 vasoreactivity preservation

MAP Challenge
Cerebral autoregulation preservation
Local/Regional CBF vs Transcranial Doppler

A comparison study of cerebral autoregulation assessed with transcranial Doppler and cortical laser Doppler flowmetry

Christian Zweifel, Marek Czosnyka, Andrea Lavinio, Gianluca Castellani, Dong-Joo Kim, Emmanuel Carrera, John D. Pickard, Peter J. Kirkpatrick and Peter Smielewski

Neurolo Res 2010; 32: 425-428
N=29 adult patients with severe TBI

- Continuous ICBF and sequential TCD assessment

Cortical autoregulation (ICBF) was worse than autoregulation assessed in the MCA (TCD) during rising ICP and falling CPP

- CPP > 60mmHg, cortical assessed autoregulation (ICBF) is similar to autoregulation assessed in the MCA (TCD)
The ASNM and ASN strongly support:
- Acquisition and interpretation of intraoperative TCD ultrasonograms performed by qualified individuals
- Service providers define their diagnostic criteria and develop ongoing self-validation programs of these performance criteria in their practices (Class III evidence, Type C recommendation)

TCD monitoring is an established monitoring modality:
- Assessment of cerebral vasomotor reactivity and autoregulation
- Documentation of the circle of Willis functional status
- Identification of relative cerebral hypo- and hyper-perfusion
- Detection of cerebral emboli (Class II and III evidence, Type B recommendation)
Structured systematized multiplane approach (temporal window):

5 axial US views vs planes
1 coronal US view vs plane (basilar pre-brainstem)
Neurosonology / Auto-regulation

![Graph showing the relationship between MCA MV and MAP (mmHg) with data points and a trend line.](Image)
Cerebral oximetry: the reality in some numbers...

Normal SctO₂ between 60 and 75% (algorithmic reasoning & contextualizing)

Baseline variation as high as 10%, may be 20%
(individual and disease-specific thresholds)
Thavasothy M et al. A comparison of cerebral oxygenation as measured by the NIRO 300 and the INVOS 5100 near-infrared spectrophotometers. Anesthesia 2002; 57: 999-1006

Reduction of 13% from baseline reflects a threshold of cerebral ischemia

SctO₂ of 35% for longer than 2-3h: permanent neurological deficits
Clinical change in SctO2 / rSO2

- Absolute values < 50% or a drop > 20% from baseline
- Absolute values of 40% and a drop > 20% are associated in neurological dysfunctions

Edmonds HL, Jr., J Intervent Cardiol 1998;11:197-204.
Cerebral Oximetry Monitoring to Maintain Normal Cerebral Oxygen Saturation during High-risk Cardiac Surgery

A Randomized Controlled Feasibility Trial

Alain Deschamps, Ph.D., M.D., Richard Hall, M.D., Hilary Grocott, M.D., C. David Mazer, M.D., Peter T. Choi, M.D., Alexis F. Turgeon, M.D., M.Sc., Etienne de Medicis, M.D., Jean S. Bussières, M.D., Christopher Hudson, M.D., Sumner Syed, M.D., Doug Seal, M.D., Stuart Herd, M.D., Jean Lambert, Ph.D., André Denault, M.D., Ph.D., for the Canadian Perioperative Anesthesia Clinical Trials Group

Anesthesiology 2016; 124: 826-836

Threshold 10%
Guidelines for the use of cerebral oximetry by near-infrared spectroscopy in cardiovascular anesthesia: a report by the cerebrospinal Division of the Academic Committee of the Japanese Society of Cardiovascular Anesthesiologists (JSCVA)

Kenji Yoshitani1,2,3 - Masahiko Kawaguchi1,2 - Kazuyoshi Ishida1,3 - Kengo Maekawa4 - Hiroshi Miyawaki5 - Satoshi Tanaka6 - Hiroyuki Uchino7 - Manabu Kakinohana8 - Yasuhiro Koide9 - Mikiya Yokota10,11 - Hirotsugu Okamoto11 - Minoru Nomura11

**Cardiopulmonary bypass**

- Cerebral saturation
  - Bilateral reduction of 20%
  - Verify head position
  - Central, optic, and superior vena cava catheter inspection
  - Rule out mechanical and superior vena cava obstruction
  - Reposition or remove catheter or cannula

- MAP
  - MAP < 50 mmHg
    - If MAP normal
      - $\text{SaO}_2$ if $\text{SaO}_2$ normal
      - If $\text{PaCO}_2$ normal
        - Correct hyperventilation (Adjust ventilation rate: $\text{PaCO}_2 > 35$ mmHg)
        - Consider red blood cell transfusion
      - $\text{Hb} < 7-8$ g/dL
        - $\text{SvO}_2 < 65$
          - Hemodynamic and TEE evaluation
          - Convolutions, hypothermia
            - Convulsions, hypothermia
              - Increased CMB
              - Cerebral imaging (CT scan/MBI)
    - If MAP normal
      - Increased ICP

- Carotid cross-clamping
  - MAP > 60 mmHg
  - $\text{rSO}_2$ on the side of surgery
    - Relative 20% reduction from baseline Absolute value ≤ 50%
    - Consider shunt placement
      - If $\text{SaO}_2$ normal
        - Lower than normal
          - Treat and find etiology (Raise oxygen concentration)
    - If $\text{Hb} > 7-8$ g/dL
      - Correct hyperventilation
        - Adjust ventilation rate $\rightarrow$ $\text{PaCO}_2 < 35$ mmHg
    - If $\text{CMRO}_2$ increased
      - Convulsions, hypothermia
        - Convulsions, hypothermia
          - Increased CMB
          - Cerebral imaging (CT scan/MBI)
Cerebral Blood Flow vs EEG / SctO2 (rcSO2)

Cerebral Oximetry
- SctO2 NIRS
  - Normal: <10%
  - Ischemia: 13-15%
  - Lesion Infarctus: >15%

Thresholds
- Normal neuronal function: BSR=0
- Progressive depression neuronal function: BSR=60
- Abolished neuronal function: BSR=35
- Neuronal death: BSR=30

Cerebral Blood Flow (mL.100g⁻¹.l⁻¹)
- Normal Brain: Normal neuronal function
- Pellucida zone: Reversible neuronal lesion
  - Only electro-chemical dysfunction
- Penumbra: Simple neuronal necrosis
- Infarctus: Non-reversible neuronal lesion

Ischemia duration (hours)
- Viable tissue
- Definitive lesion
Cerebral oximetry: **the great controversy**

- **Broad validation and use for somatic monitoring of normal brain at risk of injury** as measure of **autoregulation**
  - Brady KM et al. Stroke 2007; 38: 2818-2825

- **Particular validation in neonates and children**

- **Very variable correlation** for SjvO2 and biomarkers (S100β) **with conflicting results**
  - Subbaswamy A et al. Neurocrit Care 2009; 10: 129-135

- **Poor alternative conclusive informations regarding acute pathological situations** (TBI, stroke, cerebral hemorrhage...)

- **May be adapted after acute phase in brain injured patients** (TBI, stroke, cerebral hemorrhage...)
  - Taussky Ph et al. Neurosurg Focus 2012; 32: E2
Cerebral oximetry: some persisting questions...

- Regarding carotid surgery (CEA):
  « NIRS-guided carotid endarterectomy always controversial and has to be validated against electrophysiology »

- Regarding cardiac surgery:
  « NIRS does not support to prevent significatively POCD & stroke – Probably no powerful enough to characterize a complex and multi-origin problem» - no official guidelines

- Possible perspectives:
  1. Generalization of a systematic use in cerebral at risk patients – Remains under discussion
     Smith M. Philos Transact A Math Phys Eng Sci 2011; 369: 4452-4469
  2. Generalization during sitted or semi-sitted (beach-chair) surgical position (20% of hypotension with ischemia-related cerebrovascular events / 80% of patients with significant hypo SctO2) – No strong evidence
  3. Functional dimension – Further development

- Regarding ICU: signification problem remains:
  « Problem of the NIRS monitoring signification in non-healthy brain remains (problem of reference for the patient)...»
  Li Z et al. Microvasc Res 2010; 80: 142-147
Assessment of risk factors for cerebral oxygen desaturation during neonatal and infant general anesthesia: an observational, prospective study

n = 44
Up to 3 months of age

Conclusions: Cerebral oxygen desaturation ≥20% from baseline occurred in almost one fifth of patients. Although different perioperative factors can predispose to cerebral oxygenation changes, arterial blood pressure seems to be the most important. Gestation as another possible risk factor needs further investigation.
Corrélation entre la rSO2 moyenne et la durée du séjour >10 jours

- Choisie car elle représente une typologie de patients très consommateurs de ressources

\[ y = 67.7 - 0.36 \text{ Mean } rSO_2 \]

\[ r^2 = 0.29, P < 0.05 \]
Cerebral oximetry: finally, today and after...

- However, the *physiological dimension* is becoming increasingly clear

- Probably *unable to answer questions that are often too complex*

- Review how we pose the problem: *a simple answer to a simple question*

- *Brain tolerance to hypotension* (intraoperative period)...

- *Brain tolerance to PaCO2 variations* (hypocapnia)...

- Bedside monitoring of *cerebral vasoreactivity* (triple control)
- Physiology of the cerebral hemodynamics: complexe anatomy with vascular specificities

- Cerebral hemodynamics: multiples regulations & reactivities
  Several mechanisms working simultaneously
  Predominance of Pressure (MAP) and CO2 (vasoreactivity)

- Assessment / Monitoring
  - Imaging (not for monitoring)
  - PtiO2 / Pb02
  - Neurosonology
  - Cerebral oximetry SctO2 / rS02
### Coming back home messages...

- Physiology of cerebral blood flow under the triple control
- Central compartment: main vessels and Circle of Willis
- Peripheral compartment: arterio-cappilary tissular compound

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Cerebral Blood Flow Monitoring
Neurosono (TCCS) + rSO2 / Sct02 (NIRS): THE solution to be developed regarding either intraoperative period or ICU