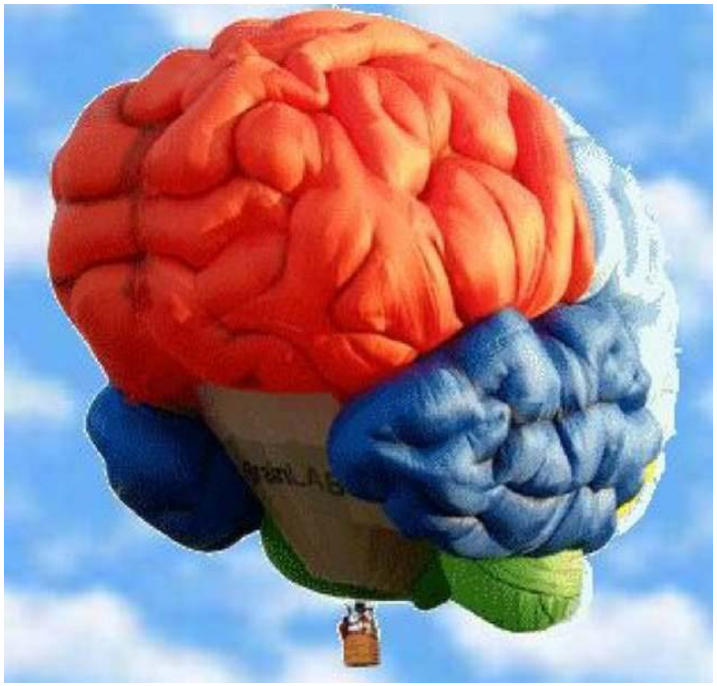


CEREBRAL OXYGENATION IN SUBARACHNOID HEMORRHAGE: QUALI MONITORAGGI?

Anesthesia and Critical Care Medicine Department of Surgical Specialties, Radiological Sciences and Public Health, University of Brescia;
Department of Anesthesia, Critical Care and Emergency, Spedali Civili University Hospital, Brescia, Italy

Dr. FRANK RASULO

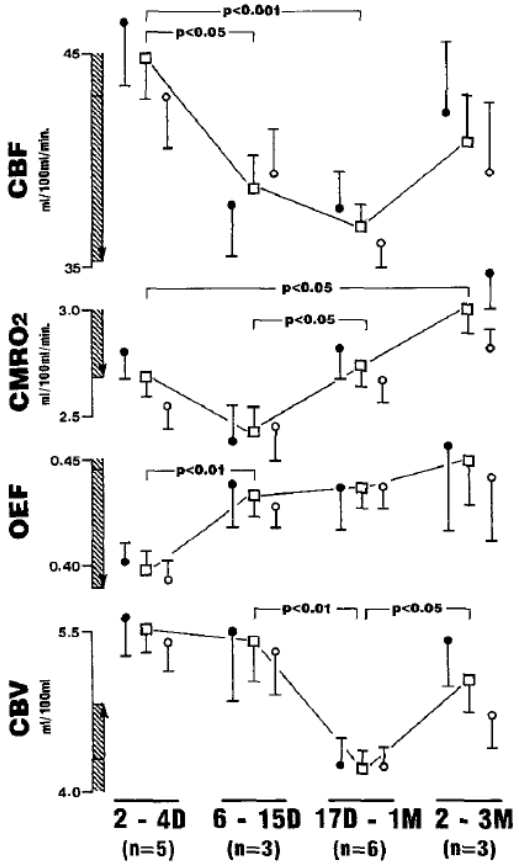
NO CONFLICTS

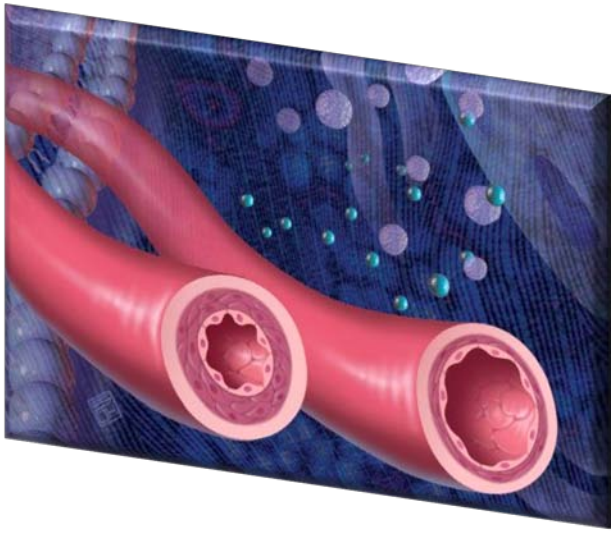
Sequential Changes of Cerebral Blood Flow After Aneurysmal Subarachnoid Haemorrhage

Acta Neurochir (Wien) (1990) 105: 98–106

M. Matsuda, A. Shiino, and J. Handa

Acta Neurochirurgica





PRIMARY METABOLIC DEPRESSION

SAH
without Vasospasm

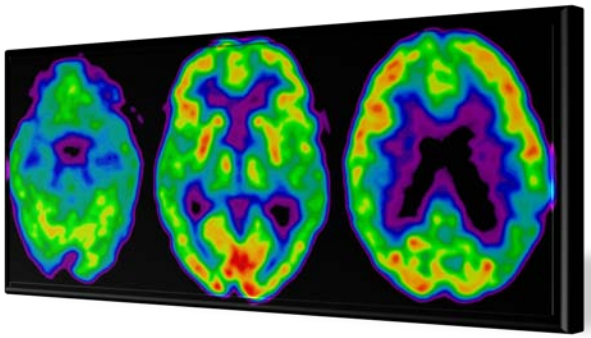
CBF
CMRO2
CBV

OEF **NORMAL**

Kent D Yundt et al .(1997)

Clinical and experimental studies have shown hypo-metabolism after SAH with no changes in CBF.

Hayashi et al 2000
Prunell GF et al 2004



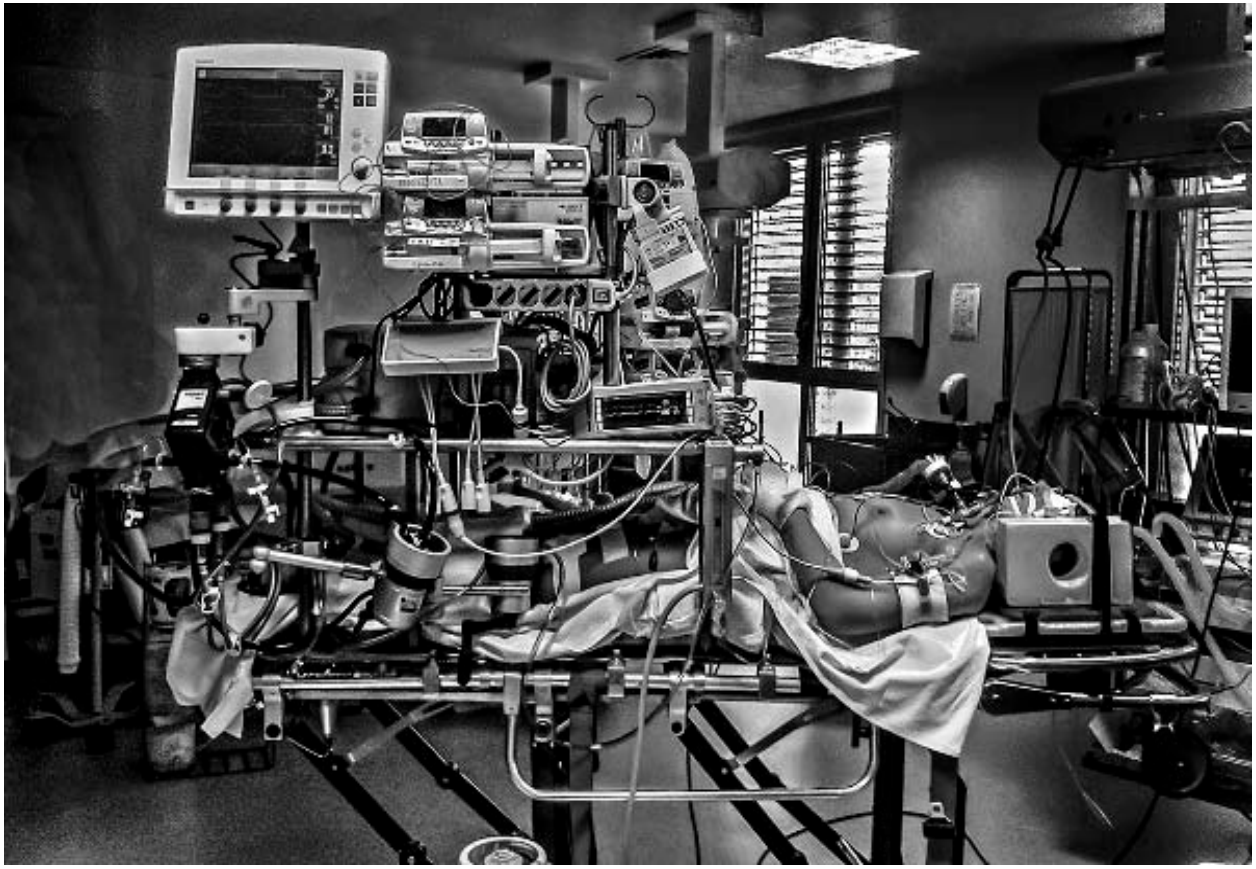
Spreading depression and blood itself probably play important roles in causing changes in metabolism.

Beaulieu et al 2000

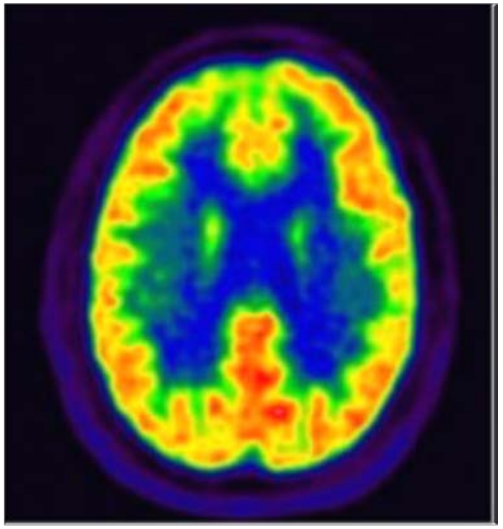
CEREBRAL OXYGENATION

	INVASIVE	NON-INVASIVE
DIRECT measurement	$PbrO_2$	
INDIRECT measurement	Microdialysis SjO_2 (global) rCBF (TD) Laser-Doppler	NIRS PET MRS CTP } Imaging

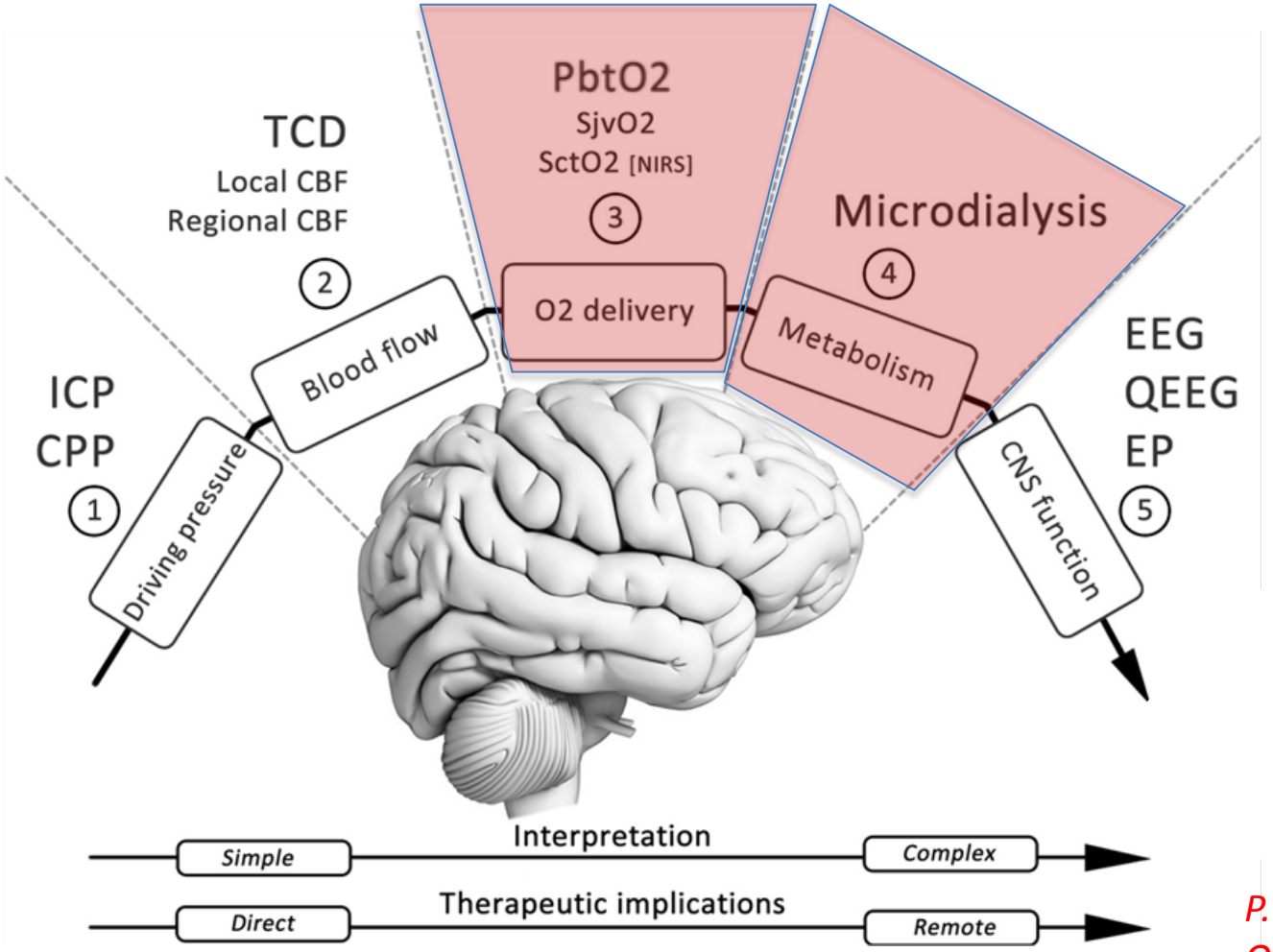
Cerebral O_2 Monitoring in SAH



SNAP-SHOT



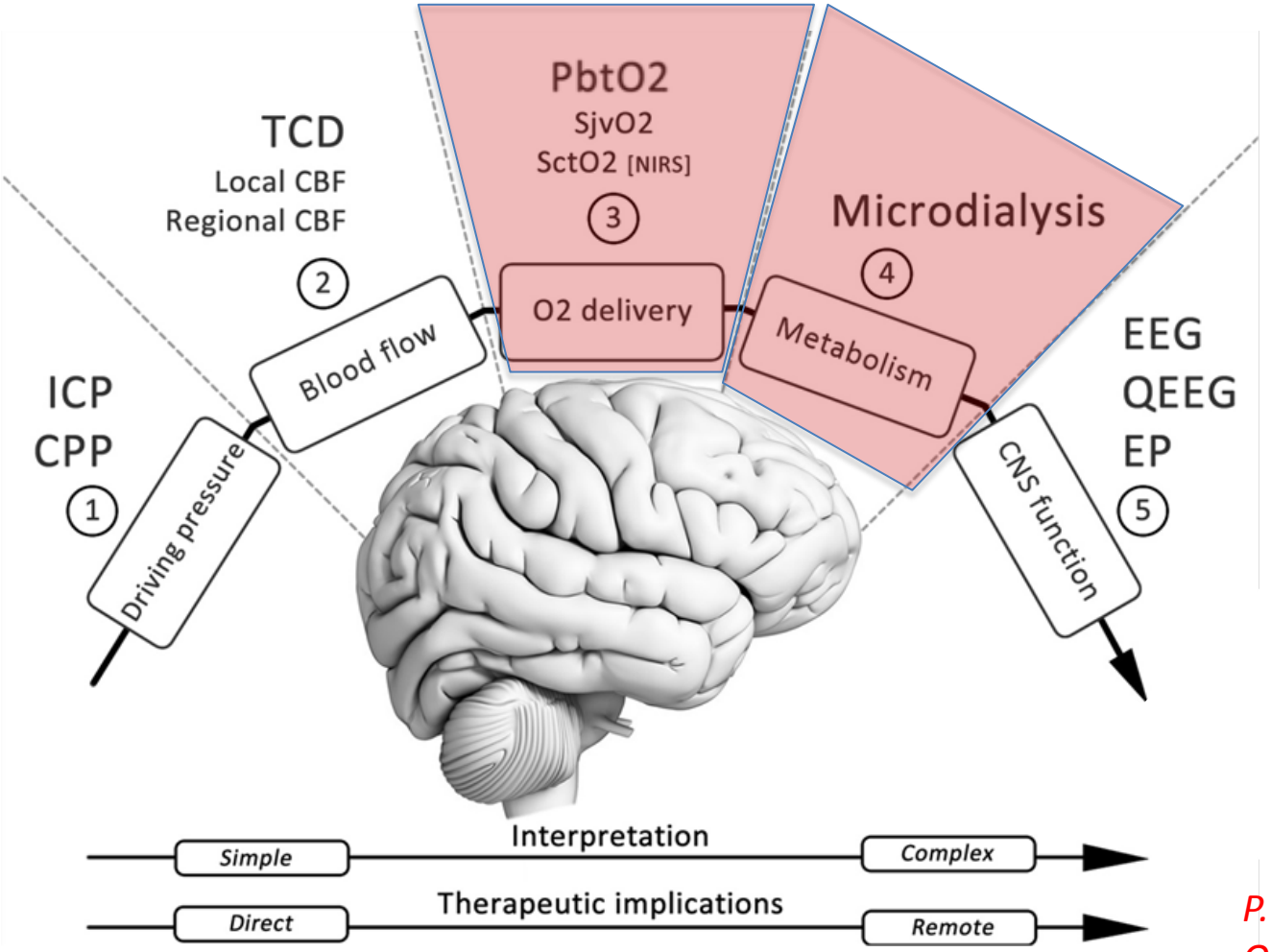
O_2 DELIVERY - METABOLISM



P. Pandin et al.
OJA, 2014

SjO_2

O_2 DELIVERY – $PbtO_2$



P. Pandin et al.
OJA, 2014

European Stroke Organization Guidelines for the Management of Intracranial Aneurysms and Subarachnoid Haemorrhage

Cerebrovascular Diseases
Cerebrovasc Dis 2013;35:93–112

Thorsten Steiner[a] Seppo Juvela[d] Andreas Unterberg[b] Carla Jung[b]
Michael Forsting[c] Gabriel Rinkel[e]

- EEG, $PbtO_2$ monitoring, and CMD may all be useful physiological monitors for DCI detection. Data from probes should be interpreted in light of its limited field of view and location in relation to pathology. The relative value of these monitors individually versus as part of a multi-modality monitoring strategy is not known (Low quality evidence—weak recommendation).

Consensus Sum
Multidisciplinar
Monitoring in N

Peter Le Roux · David K. Menon · Giuseppe
Gretchen M. Brophy · Michael N. Diringer ·
Neeraj Badjatia · Julian Böesel · Randall Ch
Marek Czosnyka · Michael De Georgia · An
David Horowitz · Peter Hutchinson · Monish
Andrew Naidech · Mauro Oddo · DaiWai Ol
Corinna Puppo · Richard Riker · Claudia R

odality

ocrit Care
ber 2014

Recommendations

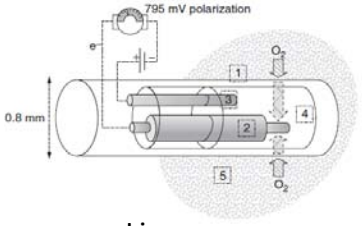
1. We recommend systemic pulse oximetry in all patients and end-tidal capnography in mechanically ventilated patients, supported by arterial blood gases measurement. (Strong recommendation, high quality of evidence.)
2. We recommend monitoring brain oxygen in patients with or at risk of cerebral ischemia and/or hypoxia, using brain tissue ($PbtO_2$) or/and jugular venous bulb oximetry ($SjvO_2$)—the choice of which depends on patient pathology. (Strong recommendation, low quality of evidence.)
3. We recommend that the location of the $PbtO_2$ probe and side of jugular venous oximetry depend on the diagnosis, the type and location of brain lesions, and technical feasibility. (Strong recommendation, low quality of evidence.)
4. While persistently low $PbtO_2$ and/or repeated episodes of jugular venous desaturation are strong predictors of mortality and unfavorable outcome, we recommend that brain oxygen monitors be used with clinical indicators and other monitoring modalities for accurate prognostication. (Strong recommendation, low quality of evidence.)
5. We suggest the use of brain oxygen monitoring to assist titration of medical and surgical therapies to guide ICP/CPP therapy, identify refractory intracranial hypertension and treatment thresholds, help manage delayed cerebral ischemia, and select patients for second-tier therapy. (Weak recommendation, low quality of evidence.)

Systematic and Comprehensive Literature Review of Publications on Direct Cerebral Oxygenation Monitoring

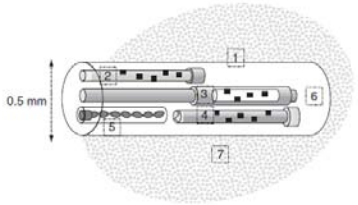
Erhard W. Lang*,1 and Matthias Jaeger2 *The Open Critical Care Medicine Journal*, 2013, *6*, 1-24

DIRECT INVASIVE PbtO2 MONITORING DEVICES

Device	Manufacturer		Sensor type
Licox -	GMS-Integra, (Kiel- Mielkendorf, Germany)	-	Polarographic ("Clark") cell
Neurotrend -	Codman, Johnson & Johnson (Raynham, MA, USA)	-	Optical sensors
Neurovent-PTO -	Raumedic (Münchberg, Germany)		Luminescense quenching
MPBS -	Oxford Optronix (Oxford, UK)		
Foxy, AL-300 -	Ocean Optics (Dunedin, FL, USA)		
PO2-100DW -	Inter Medical Co. Ltd. (Nagoya, Japan)	-	Clark type electrode

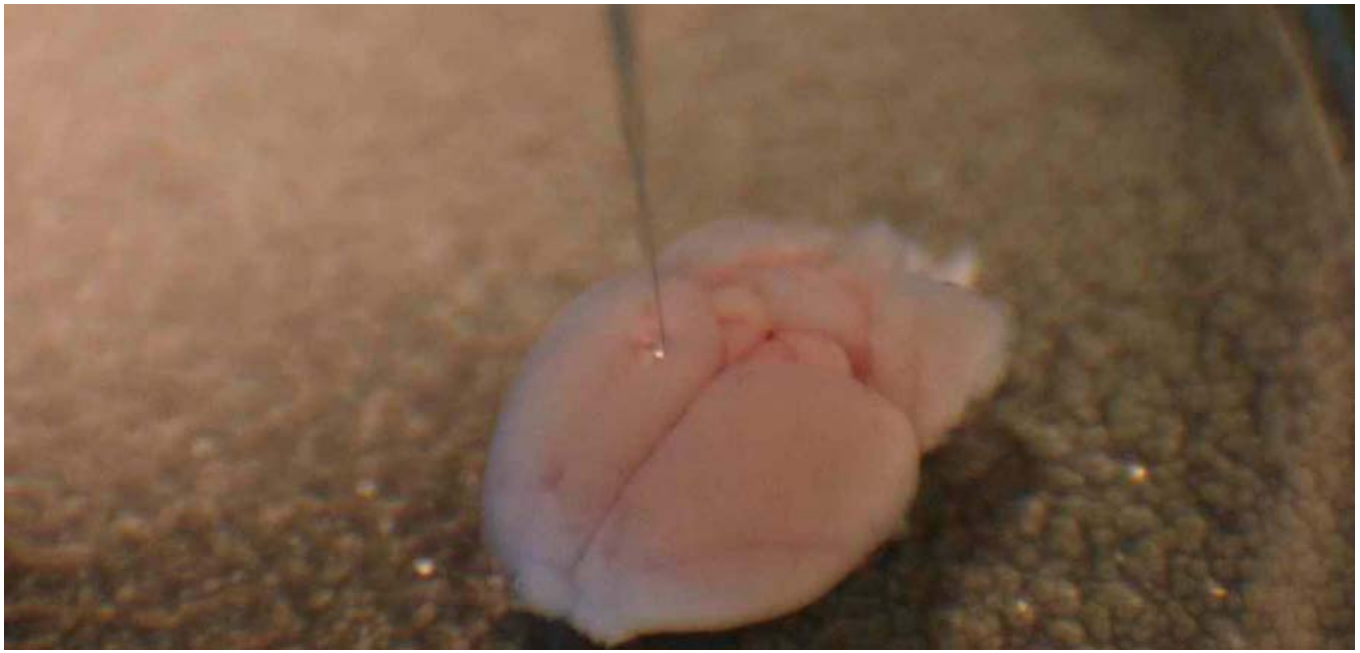


Licox sensor



Neurotrend sensor

$PbtO_2$

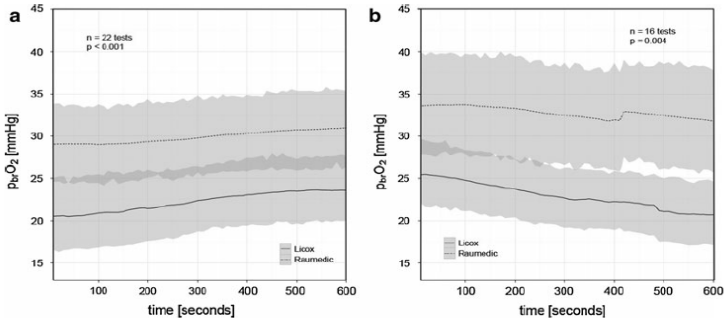


8-12 µm

Probes cannot be used interchangeably in patients after SAH

Cerebral tissue oxygenation measured by two different probes: challenges and interpretation

Julius Dengler
Intensive Care Med (2011) 37:1809–1815



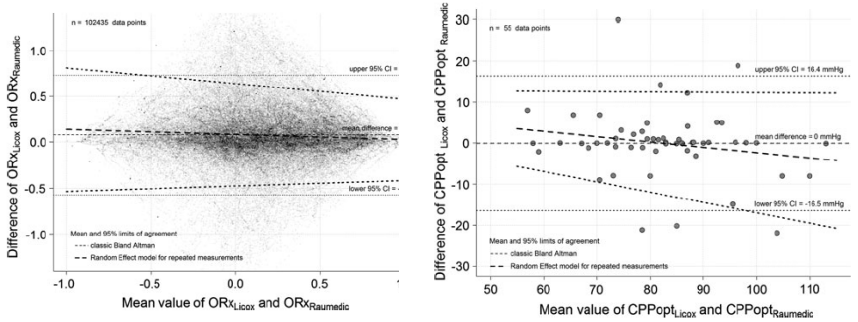
Licox - Raumedic

LX and NV probes measure different PbrO2 values in routine monitoring in patients after SAH and TBI. Our data therefore do not support the view that both probes can be used interchangeably.

The Oxygen Reactivity Index and Its Relation to Sensor Technology in Patients with Severe Brain Lesions

Julius Dengler · Christin Frenzel · Peter Vajkoczy · Peter Horn · Stefan Wolf

neurocritical care society Neurocrit Care (2013) 19:74–78



The main result is that Licox and Raumedic showed consistent differences in ORx and CPP_{opt}. Therefore, ORx values of both probes cannot be interchanged and should not be viewed as equivalent. This should be taken into

DIFFERENT THRESHOLD VALUES BETWEEN ***SAME*** DEVICES

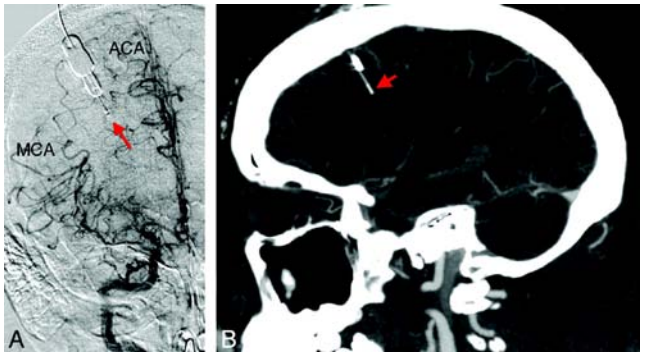
Sensor	Authors	Year	Proposed threshold [mm Hg (kPa)]	How the threshold was determined
Paratrend 7	Zauner and colleagues[102] Doppenberg and colleagues[19]	1997, 1998	25 (3.3)	Pb_{O_2} of 26 mm Hg ≈ CBF (xenon CT) < 18 ml per 100 g per min; all patients with Pb_{O_2} <25 mm Hg had a poor outcome
Paratrend 7	Doppenberg and colleagues[18]	1998	Between 19 and 23 (2.5 and 3)	Combined above data with a feline MCA occlusion study and outcome
Neurotrend	Menon and colleagues[66]	2004	10 (1.3)	Significantly greater diffusion gradients for oxygen ($Pv_{O_2} - Pb_{O_2}$) if Pb_{O_2} ≤10 mm Hg
Neurotrend	Johnston and colleagues[50]	2005	<14 (1.9)	Significant linear relationship between Pb_{O_2} and PET OEF (r^2=0.21, P<0.05); mean normal OEF=40% associated with Pb_{O_2}=14 mm Hg
Licox	Kiening and colleagues[53]	1996	8.5 (1.1)	Regression analysis: Sj_{O_2} threshold of 50% correlated with Pb_{O_2} of 8.5 mm Hg
Licox	van Santbrink and colleagues[98]	1996	Between 10 and 15 (1.3 and 2)	Significant difference in 6 month outcome at threshold ≤5 mm Hg (P=0.04), suggested maintenance of Pb_{O_2} between 10 and 15 mm Hg
Licox	Valadka and colleagues[96]	1998	20 (2.7) [6 (0.8)]	Tobit regression analysis relating the time below thresholds of Pb_{O_2} with likelihood of death. Much greater likelihood of death, the longer the Pb_{O_2} <20 mm Hg or any time of Pb_{O_2} <6 mm Hg
Licox	van den Brink and colleagues[97]	2000	<5 (0.6) for 30 min <10 (1.3) for 1 h 45 min <15 (2) for 4 h	The relative risk of death was graded. Hypoxic thresholds are expressed as the depth and duration of hypoxia imparting a 50% risk of death

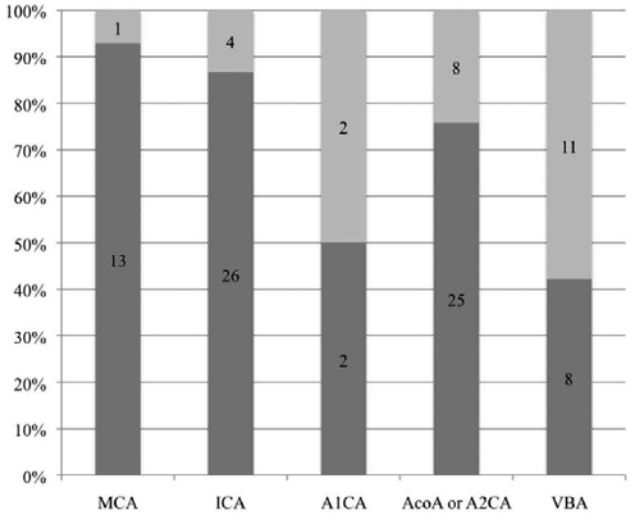
$PbtO_2$ *where ?*

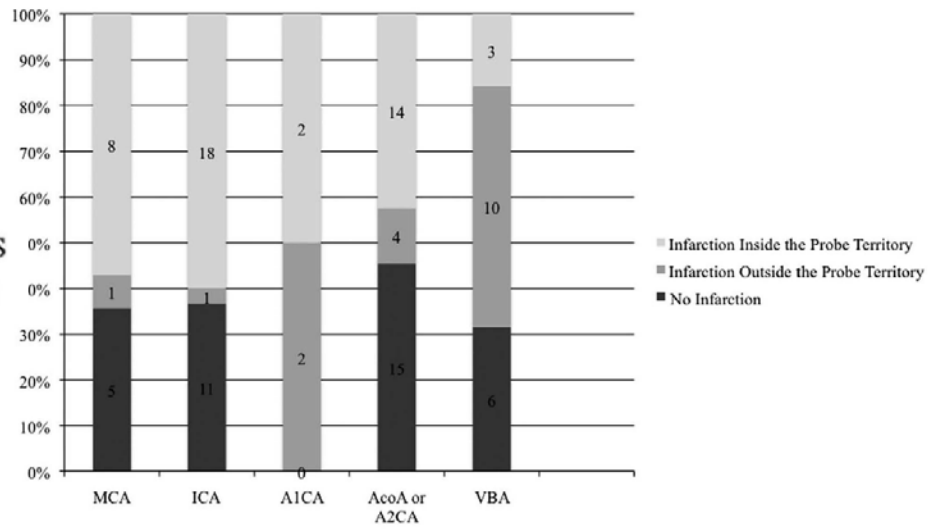
Occurrence of Vasospasm and Infarction in Relation to a Focal Monitoring Sensor in Patients after SAH: Placing a Bet when Placing a Probe?

PLOS ONE May 2013 | Volume 8 | Issue 5

Christian T. Ulrich[1]*, Christian Fung[1], Hartmut Vatter[2], Matthias Setzer[2], Erdem Gueresir[2], Volker Seifert[2], Juergen Beck[1], Andreas Raabe[1]







The probability that a single focal probe will be situated in the territory of severe CVS and infarction varies over a wide range.

Focal ptiO2 or MD measurements are useful for MCA and ICA aneurysms, but may have a high (50%) failure rate in patients with VBA and ACA aneurysms. More reliable CVS or infarction detection was observed in MCA and ICA.

Brain Tissue Oxygen Monitoring: Physiologic Principles and Clinical Application

Operative Techniques in Neurosurgery, Vol 7, No 1 (March), 2004: pp 2-9

Venu M. Nemani, MD, and Geoffrey T. Manley, MD, PhD

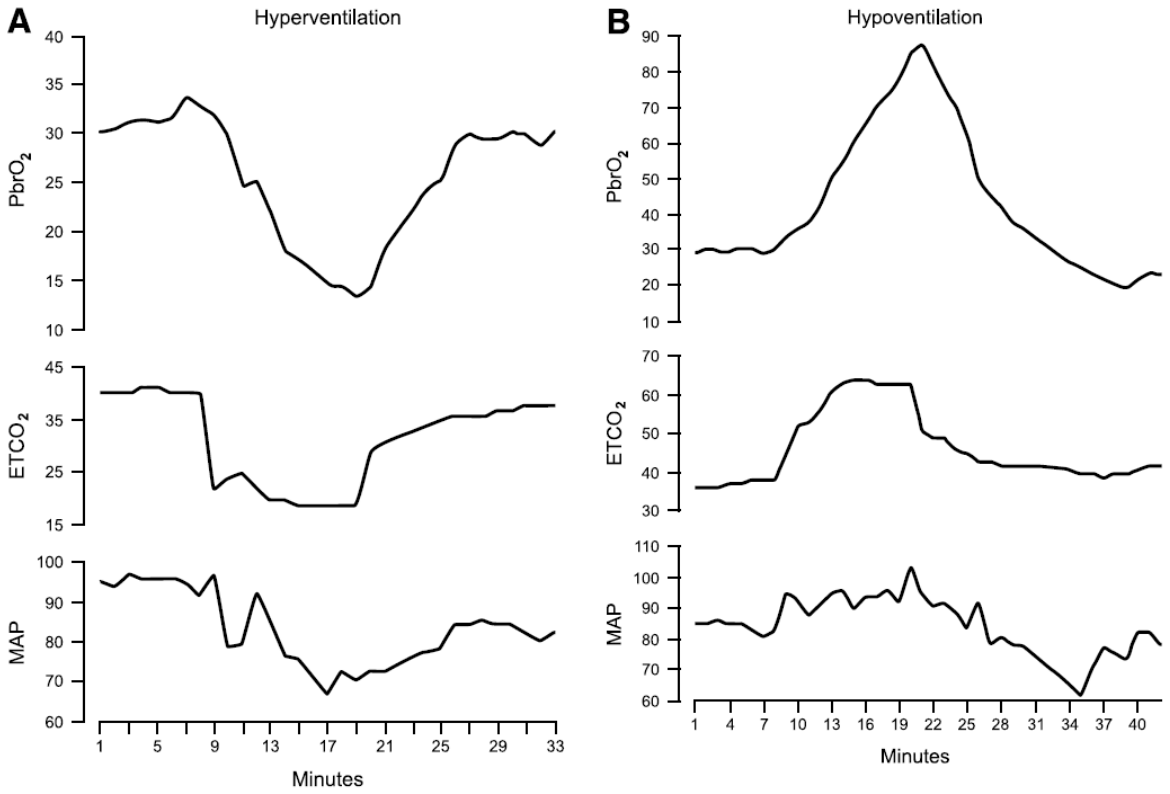


Fig 2. Characteristic effects of hyperventilation and hypoventilation on $PbrO_2$, end-tidal CO_2 ($ETCO_2$), and mean arterial pressure (MAP) from a representative experiment. (A) Hyperventilation for 10 minutes simultaneous decreased $PbrO_2$ and $ETCO_2$. A notable decrease in MAP was also observed. (B) Hypoventilation for 10 minutes increased $PbrO_2$ and $ETCO_2$ with no significant change in MAP. All values are in mmHg.

$PbtO_2$ FiO_2, CBF

MATHEMATICAL MODEL FAILS TO PREDICT HYPEROXIA INDUCED $PbtO_2$ VARIATIONS. PRELIMINARY FINDINGS.

Rita Bertuetti[1], Frank Rasulo[1], Andrea Lavinio[1,2], Alan Girardini[1], Paola Gazzoli[1], Nicola Latronico[1]

[1]Neuro Critical Care Unit, Institute of Anesthesia and Intensive Care, Spedali Civili University Hospital of Brescia, Italy.

[2]Neuro Critical Care Unit, Institute of Anaesthesia, Addenbrooke's University Hospital of Cambridge UK.

Objective:

to investigate relationships between cerebral tissue oxygen tension ($PbtO_2$) and arterial oxygen partial pressure (PaO_2) during cerebral blood flow (CBF) and metabolism steady state, also to understand whether cerebral oxygen tension depends on oxygen delivery or directly on PaO_2 during hyperoxia

Material and methods:

the authors created a mathematical model of $PbtO_2$ response to hyperoxia plotting PaO_2 as an independent variable (x), $PbtO_2$ as a dependent variable (y), and haemoglobin concentration, CBF and cerebral metabolic rate of oxygen ($CMRO_2$) as parametric variables (Fig.1). Four patients suffering severe head injury or subarachnoid hemorrhage (Glasgow Coma Scale ≤ 8) were enrolled in this study. Each patient received $PbtO_2$, ICP, MAP and CPP continuous monitoring. For assessment of autoregulation ORx ($PbtO_2$ pressure reactivity index) and PRx (pressure reactivity index) were calculated. A hyperoxic stimulation test able to provide comparable data with the predictive value of the model was elaborated: inspiratory oxygen fraction (FiO_2) was gradually increased by 20% every five minutes from a baseline value of 40% to a maximum of 100%. At the forth minute after the new FiO_2 set up an arterial blood sample was taken.

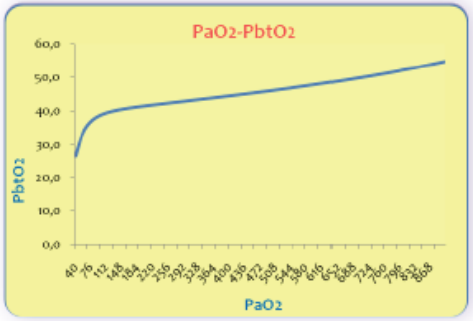


Fig. 1. Theoretical prototype created by assigning virtual values to the parametrical variables: CBF=40 ml/100 g/min; Hb=14 g/dl; CMRO2= 2 ml/100 g/min.

Results:

hyperoxic stimulation tests were performed. For each test, the relationship between PaO_2 and $PbtO_2$ showed to be positive and strong: mean linear correlation coefficient R =0.958 (±0.058) and mean R^2=0.923(±0.107).

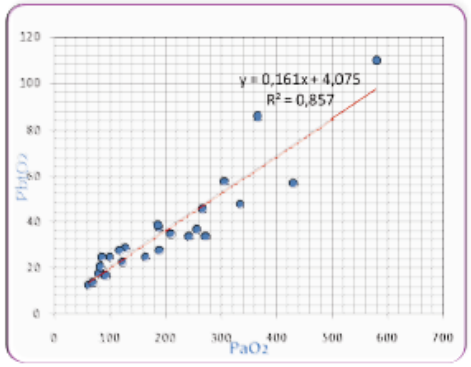


Fig. 2. Plotting of PaO_2 values with the corresponding $PbtO_2$ measured by means of hyperoxic stimulation tests. The calculated regression line shows a linear pattern, differently from what expected by the mathematical model.

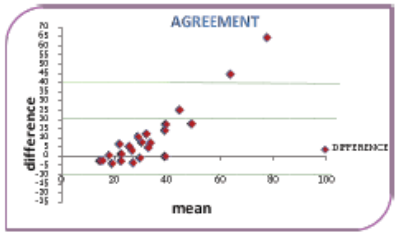


Fig. 3. Graphical representation of Bland & Altman test comparing $PbtO_2$ observed measures and model expected values. Green dotted lines represent, from upper to lower, upper limit of concordance between the two compared values, the bias, and the lower limit of concordance. As the mean increases the difference between the numbers obtained through the two methods increases too.

Conclusions:

relationship between PaO_2 and $PbtO_2$ resulted to be significantly strong, and therefore proving the mathematical model to be wrong (Fig. 2). During hyperoxia $PbtO_2$ showed to be directly dependent on PaO_2 rather than oxygen delivery, so the correlation between $PbtO_2$ and CBF previously proved in literature is not to be such under conditions of hyperoxygenation, the result being an overestimation of the CBF which may limit the prognostic influence of $PbtO_2$ under such conditions.

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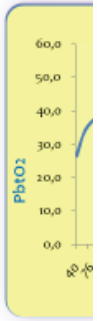

Fig. 1. Theoretic variables: CBF=

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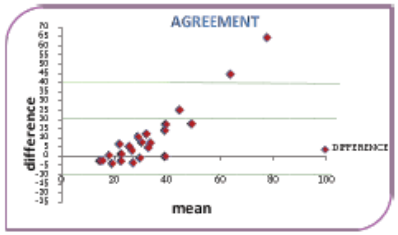
Fig. 2. Plotting hyperoxic stim differently fro



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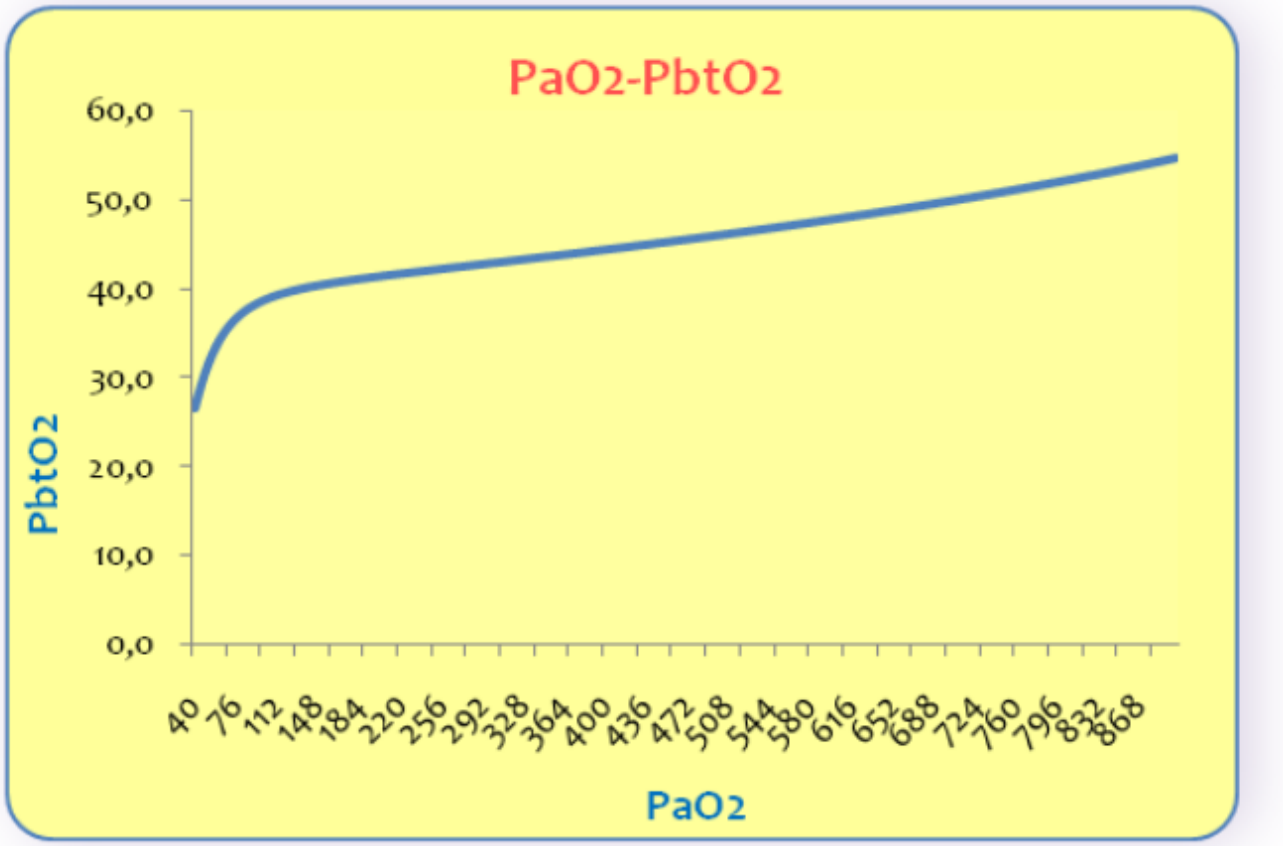

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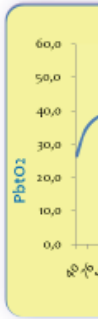

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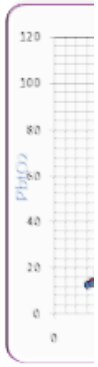

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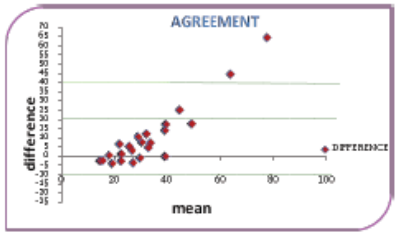

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2). During hyperoxia $PbtO_2$ showed to be directly dependent on PaO_2 rather t
CBF previously proved in literature is not to be such under conditions of hyper
which may limit the prognostic influence of $PbtO_2$ under such conditions.

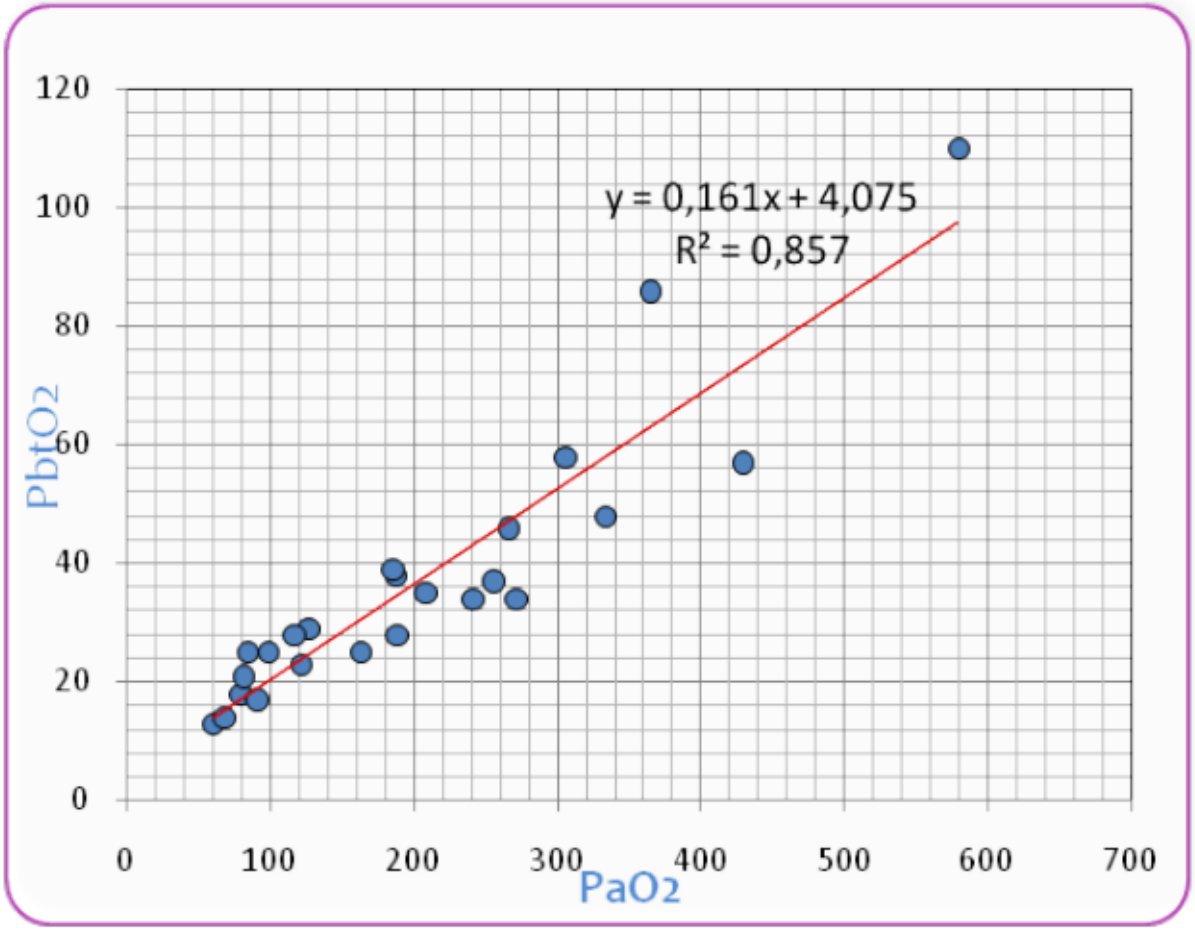


Fig. 2. Plotting of PaO_2 values with the corresponding $PbtO_2$ measured by means of hyperoxic stimulation tests. The calculated regression line shows a linear pattern, differently from what expected by the mathematical model.

$PbtO_2$ FiO_2, CBF

MATHEMATICAL MODEL FAILS TO PREDICT HYPEROXIA INDUCED $PbtO_2$ VARIATIONS. PRELIMINARY FINDINGS.

Rita Bertuetti[1], Frank Rasulo[1], Andrea Lavinio[1,2], Alan Girardini[1], Paola Gazzoli[1], Nicola Latronico[1]

[1]Neuro Critical Care Unit, Institute of Anesthesia and Intensive Care, Spedali Civili University Hospital of Brescia, Italy.

[2]Neuro Critical Care Unit, Institute of Anaesthesia, Addenbrooke's University Hospital of Cambridge UK.

Objective:

to investigate relationships between cerebral tissue oxygen tension ($PbtO_2$) and arterial oxygen partial pressure (PaO_2) during cerebral blood flow (CBF) and metabolism steady state, also to understand whether cerebral oxygen tension depends on oxygen delivery or directly on PaO_2 during hyperoxia

Material and methods:

the authors created a mathematical model of $PbtO_2$ response to hyperoxia plotting PaO_2 as an independent variable (x), $PbtO_2$ as a dependent variable (y), and haemoglobin concentration, CBF and cerebral metabolic rate of oxygen ($CMRO_2$) as parametric variables (Fig.1). Four patients suffering severe head injury or subarachnoid hemorrhage (Glasgow Coma Scale ≤ 8) were enrolled in this study. Each patient received $PbtO_2$, ICP, MAP and CPP continuous monitoring. For assessment of autoregulation ORx ($PbtO_2$ pressure reactivity index) and PRx (pressure reactivity index) were calculated. A hyperoxic stimulation test able to provide comparable data with the predictive value of the model was elaborated: inspiratory oxygen fraction (FiO_2) was gradually increased by 20% every five minutes from a baseline value of 40% to a maximum of 100%. At the forth minute after the new FiO_2 set up an arterial blood sample was taken.

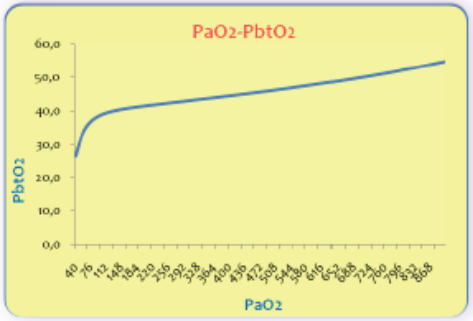
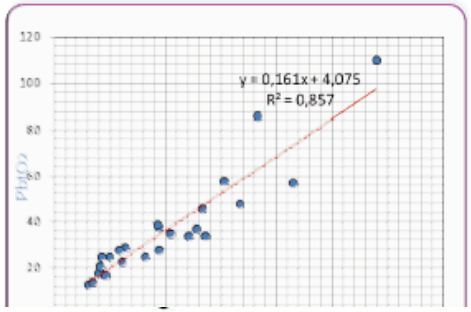


Fig. 1. Theoretical prototype created by assigning virtual values to the parametrical variables: CBF=40 ml/100 g/min; Hb=14 g/dl; CMRO2= 2 ml/100 g/min.

Results:

hyperoxic stimulation tests were performed. For each test, the relationship between PaO_2 and $PbtO_2$ showed to be positive and strong: mean linear correlation coefficient R =0.958 (±0.058) and mean R^2=0.923(±0.107).



Conclusions:

relationship between PaO_2 and $PbtO_2$ resulted to be significantly strong, and therefore proving the mathematical model to be wrong (Fig. 2). During hyperoxia $PbtO_2$ showed to be directly dependent on PaO_2 rather than oxygen delivery, so the correlation between $PbtO_2$ and CBF previously proved in literature is not to be such under conditions of hyperoxygenation, the result being an overestimation of the CBF which may limit the prognostic influence of $PbtO_2$ under such conditions.

The physiology behind direct brain oxygen monitors and practical aspects of their use

Childs Nerv Syst (2010) 26:419–430

Eileen Maloney-Wilensky · Peter Le Roux

Table 1 Interventions that can be used to correct $PbtO_2$ values

$P_{bt}O_2$ Low (<20mmHG)		
Increased Demand	⬆ ICP	Treat ICP - diuretics, CSF drainage, sedation (barbiturates, Propofol), craniotomy
	Pain	Give pain medication
	Shivering	Stop shivering- Demerol, Thorazine, paralytic
	Agitation	Give sedation
	Seizures	Give Benzodiazapine & adjunct anticonvulsant
	Fever	Treat fever- Tylenol, NSAID, cooling devices
Decreased Delivery	Hypotension ⬇ (CPP)	Isotonic fluids (NS or hypertonic saline), vasopressors
	Hypovolemia	Isotonic fluids (NS or hypertonic saline), blood replacement
	Anemia	Blood replacement
	Hypoxia	Increase FIO2, PEEP, pulmonary toilet

$P_{bt}O_2$ high (>50mmHG)		
Increased delivery	Hyperdynamic (hyperemic)	Hyperventilation?
Decreased demand	Hypothermia	Normothermia
	Sedatives Anesthesia Paralysis	Decrease sedation, anesthesia, or paralysis as needed but treatment may not be necessary

The physiology behind direct brain oxygen monitors and practical aspects of their use

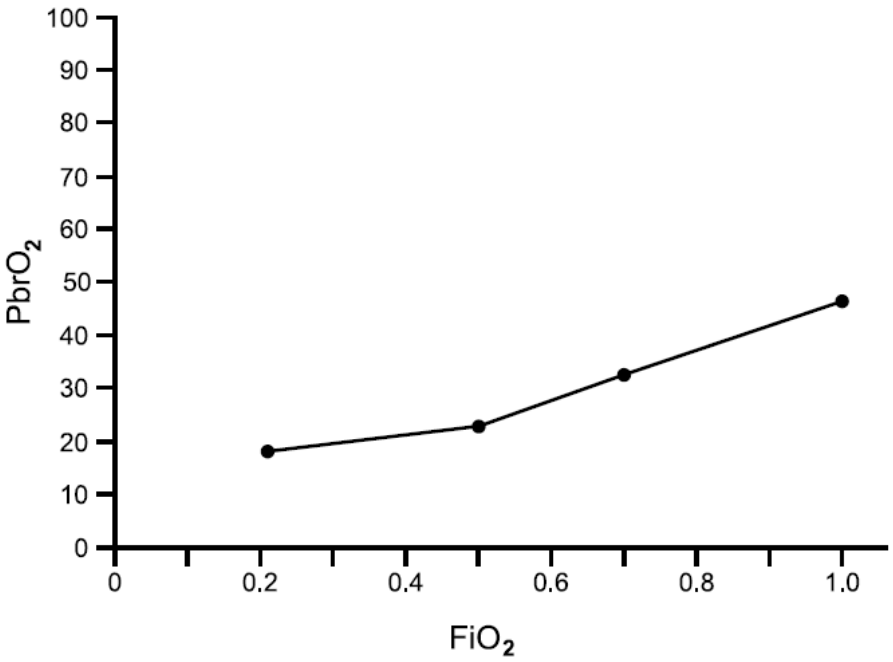
Childs Nerv Syst (2010) 26:419–430

Eileen Maloney-Wilensky · Peter Le Roux

Table 2 Therapies used in our ICU to treat compromised brain oxygen

Frequently used therapy	Less frequently used therapy
Adjust ventilator parameters to increase PaO_2	Ventriculostomy
Increase FiO_2 (e.g. 50 to 60%)	Continuous or intermittent CSF drainage
Increase PEEP	Blood transfusion
Transient Normobaric Hyperoxia 100% FiO_2	Neuromuscular paralysis
Augment CPP	Pancuronium, vecuronium
Colloid bolus	Adjust ventilator rate
Neosynephrine, dopamine	Increase to lower $PaCO_2$ (ICP)
Pharmacologic analgesia and sedation	Decrease to increase $EtCO_2$, $paCO_2$
Propofol, versed, ativan	Pulmonary toillette and suction
Fentanyl, morphine	Penthothal (barbiturate burst suppression)
Head position or avoid turning, certain positions	Labetalol
ICP control	
Sedation, mannitol, IV lidocaine, HTS	
Insure temperature <38°C	
DC (or other cranial surgery)	

$PbtO_2$ & FiO2



Give Yourself a

BOOST!

95% Pure Oxygen

$PbtO_2$ & FiO2

Benefits of increased PaO2 to the brain

An increase in PbtO2 is associated with improved brain metabolism, measured with cerebral microdialysis

Normobaric hyperoxia-induced improvement in cerebral metabolism and reduction in intracranial pressure inpatients with severe head injury: a prospective historical cohortmatched study. Tolias CM et al. J Neurosurg (2004) 101:435–444

high-flow oxygen therapy reduces infarct volumes in animal stroke models, and improves clinical deficits in patients with acute stroke

Singhal Ab et al. Normobaric hyperoxia reduces MRI diffusion abnormalities and infarct size in experimental stroke. Neurology (2002) 58:945–952

Singhal AB, et al A pilot study of normobaric oxygen therapy in acute ischemic stroke. Stroke (2005)36:797–802

Increasing FiO2 in patients with brain injury increased O2 delivery to the brain and decreased the level of lactate levels as measured by microdialysis .

Bergsneider M, Hovda DA, Shalmon E, et al: Cerebral hyperglycolysis following severe traumatic brain injury in humans: A positron emission tomography study. J Neurosurg 86:241-251, 1997

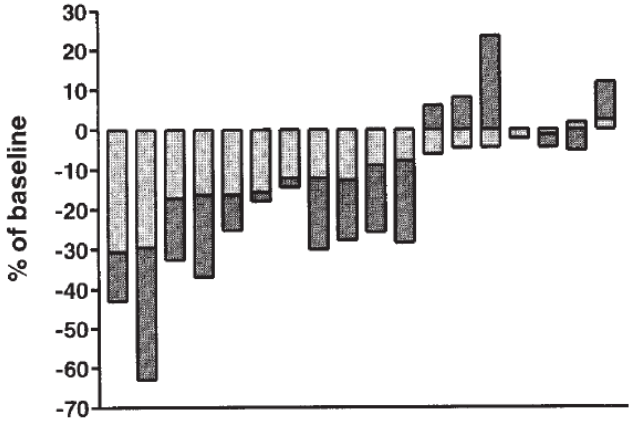
Increased inspired oxygen concentration as a factor in improved brain tissue Oxygenation and tissue lactate levels after severe human head injury.

Menzel M et al: J Neurosurg 91:1-10, 1999

Lack of improvement in cerebral metabolism after hyperoxia in severe head injury: a microdialysis study

J Neurosurg 98:952–958, 2003

SANDRA MAGNONI, M.D., LAURA GHISONI, M.D., MARCO LOCATELLI, M.D., MARIANGELA CAIMI, M.D., ANGELO COLOMBO, M.D., VALERIO VALERIANI, M.D., AND NINO STOCCHETTI, M.D.



Conclusions

In this study we confirm that increasing FiO_2 to 100% causes lactate in brain tissue after TBI to decrease slightly. Lactate falls similarly in adipose tissue, indicating a systemic effect of hyperoxia. We found no improvement in the lactate/pyruvate ratio, however, and a nonsignificant reduction of cerebral O_2 extraction. These data indicate an overall depression of cerebral glucose metabolism rather than improved oxidative function. In the absence of more convincing data on its benefits, and considering the potential harmful effects on the respiratory apparatus of hyperoxia, we conclude that it cannot be recommended for improving brain metabolism after TBI.

TABLE 5

*Comparison of the main features of hyperoxia testing in our study and in Menzel, et al.**

Study Feature	Menzel, et al.	Present Study
methods		
infusion rate	2 μl/min	0.3 μl/min
glucose recovery	35%†	80%‡
lactate recovery	42%†	80%‡
dialysate sampling int	30 mins	30 mins
clinical data		
no. of patients	12	8
age (yrs)§	35.5 ± 16.8	41.1 ± 21.2
baseline gluc (mmol/L)§	0.56 ± 0.39	2.28 ± 1.35
baseline lact (mmol/L)§	1.3 ± 0.9	3.2 ± 2.8
hyperoxia		
int btwn TBI & test (hrs)§	12 ± 7	44 ± 18
no. of tests	12	18
duration of test	6 hrs (3 hrs FiO_2 60% + 3 hrs FiO_2 100%)	3 hrs FiO_2 100%

Effect of normobaric hyperoxia on cerebral oxygenation, metabolism and oxidative stress in patients with subarachnoid hemorrhage caused by intracranial aneurysm rupture.

Solodov AA, et al. Anesteziol Reanimatol. 2013 Jul-Aug;(4):66-71

Conclusions:

В статье описываются особенности комбинированного метода пластики дефекта основания черепа у больных назальной ликвореей с локализацией в клиновидной пазухе. Всего таким способом было прооперировано 15 пациентов с локализацией ликворной фистулы в клиновидной пазухе, у 8 из которых верифицировано менингоцеле. Основными преимуществами способа являются: хорошая визуализация всех отделов клиновидной пазухи, надежная пластика ликворной фистулы, функциональность, сохранение анатомической целостности полости носа и клиновидной пазухи.

Ключевые слова: назальная ликворея, местные питаемые лоскуты, дефект основания черепа, клиновидная пазуха.

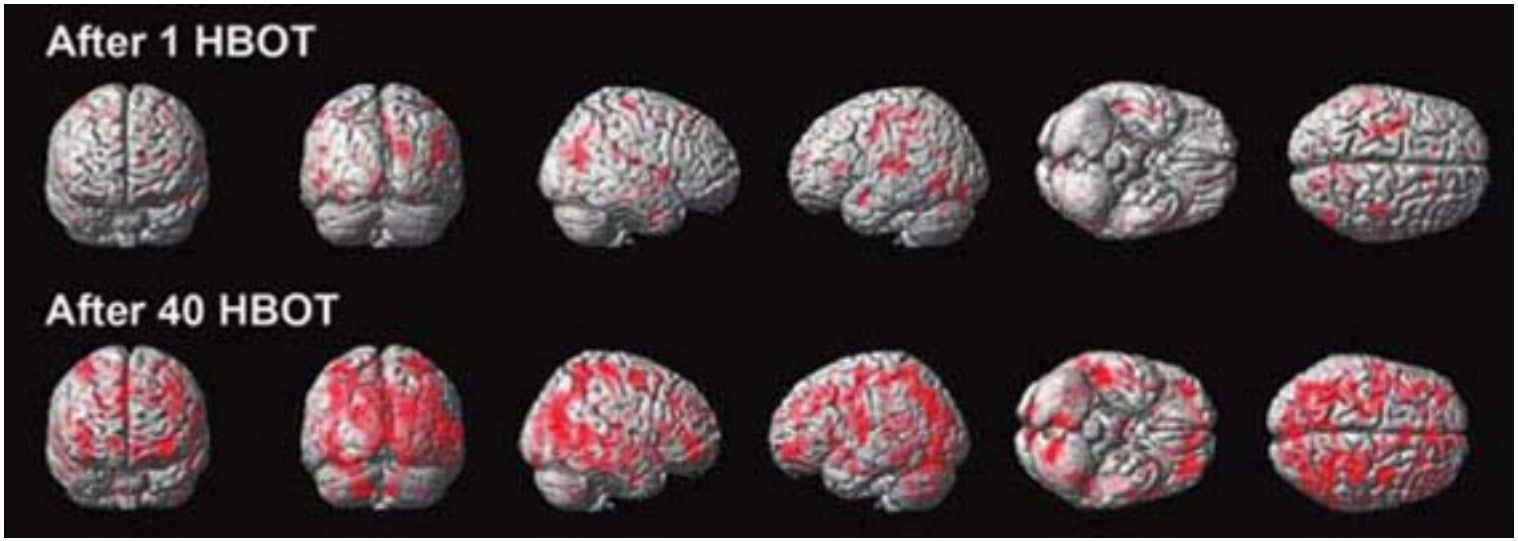
Библиография: 8 источников.

Increase of FiO2 from 0.3 to 0.5 and 1.0 was accompanied with brain oxygen tension (PbrO2) increase and cerebral extraction ratio for oxygen (O2ER) decrease. Application of normobaric hyperoxia had no effect on ICP, cerebral perfusion pressure, arterial blood pressure and cerebral metabolism.

$PbtO_2$ & Hyper-FiO2

Effect of hyperbaric oxygen therapy on cerebral vasospasm: a vascular morphometric study in subarachnoid hemorrhage

Özgür Çelika et al. International Journal of Neuroscience Volume 2014: 124(8)



CEREBRAL OXYGENATION

Intraoperative brain oxygenation monitoring and vasospasm in aneurysmal subarachnoid hemorrhage.
Cerejo A et al. Neurol Res 2012; 34(2): 181-6.

28 aSAH patients

Post-operative TCD vasospasm developed in 13 patients, all of them with basal values inferior to 10 mmHg.

PbtO(2) basal value was significantly lower in cases that developed TCD vasospasm.

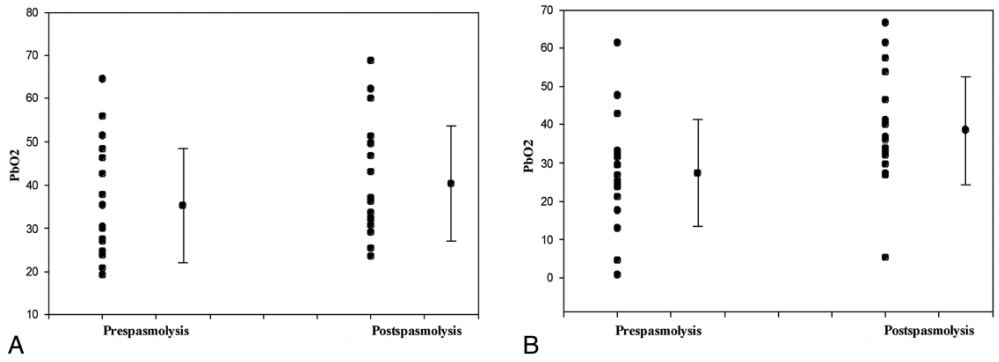
The finding of low intraoperative basal PbtO(2) values may be an indicator for a high risk of occurrence of post-operative TCD vasospasm in cases of aneurysmatic SAH.

Brain Tissue Oxygen Monitoring to Assess Reperfusion after Intra-Arterial Treatment of Aneurysmal Subarachnoid Hemorrhage—Induced Cerebral Vasospasm: A Retrospective Study

AJNR Am J Neuroradiol 33:1411–15 | August 2012 |

Mild-to-moderate and moderate-to-severe group physiologic parameters before and after spasmolytic therapy along with percentage improvement in PbO_2 after spasmolytic therapy

Vasospasm Severity	Timing	PbO_2[a] (mm Hg ± SE)	CPP[b] (mm Hg ± SE)	ICP[b] (mm Hg ± SE)	SaO_2[b] (mm Hg ± SE)	FIO_2[b] (mm Hg ± SE)	% PbO_2 Improvement
Mild-mod	Prespasmolysis	35.2 ± 3.1	110.9 ± 3.5	5.4 ± 2.2	99.6 ± 0.3	55.7 ± 3.5	14
	Postspasmolysis	40.3 ± 3.1	107.9 ± 4.0	4.6 ± 1.0	99.5 ± 0.3	55.5 ± 4.1	
Mod-sev	Prespasmolysis	27.3 ± 3.1	116.7 ± 3.8	5.8 ± 1.3	99.8 ± 0.2	57.5 ± 6.1	40
	Postspasmolysis	38.4 ± 3.2	113.9 ± 4.4	7.8 ± 1.9	99.2 ± 0.5	57.0 ± 6.1	



100% of instances the mean PbO2 increased after spasmolysis and correlated with improvement in angiographic VS .

CPP, ICP, SaO2, and FIO2, did not show any statistically significant difference before and after spasmolysis.

The utility of pbtO2 for optimizing Triple-H therapy in SAH patients

Relative importance of hypertension compared with hypervolemia for increasing cerebral oxygenation in patients with cerebral vasospasm after subarachnoid hemorrhage.
Raabe A et al. J Neurosurg 2005;

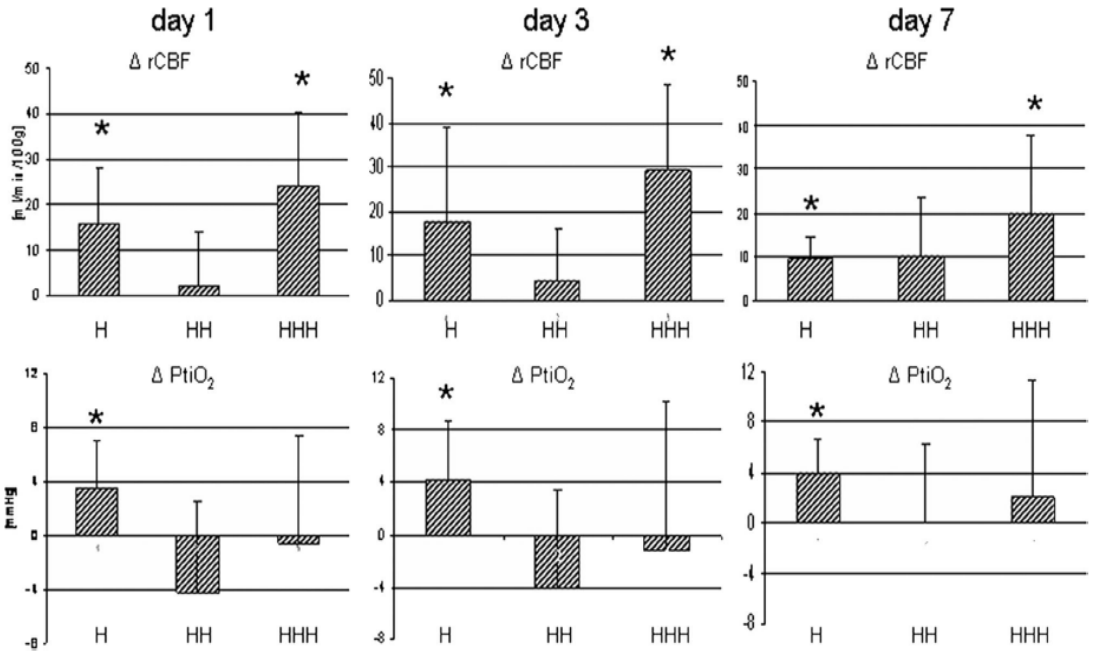
- 55 periods of moderate hypertension - pbtO2 increases 50 cases (90%), Complications in 3 patients (8%).
- 25 periods of hypervolemia, pbtO2 increases during three intervals (12%), Complications in 9 patients (53%).
- 10 periods of hypervolemic hypertension, pbtO2 increases during 6 of the intervals (60%), Complications in 5 patients (50%).

In poor-grade aSAH patients, moderate hypertension in a normovolemic, hemodiluted patient is an effective method of improving cerebral oxygenation and is associated with a lower complication rate compared wit hypervolemia

The utility of pbtO2 for optimizing Triple-H therapy in SAH patients

Effects of hypervolemia and hypertension on regional cerebral blood flow, intracranial pressure, and brain tissue oxygenation after subarachnoid hemorrhage.

Muench E, Horn P, Bauhuf C, *et al*. Crit Care Med 2007; 35(8): 1844-51



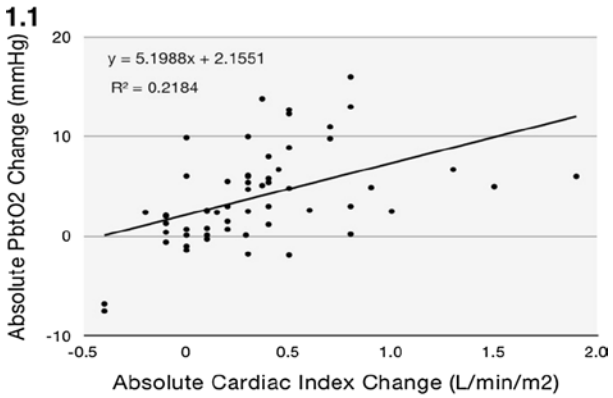
Vasopressor-induced elevation of MAP caused an increase of CPP and Pti02 in SAH patients.

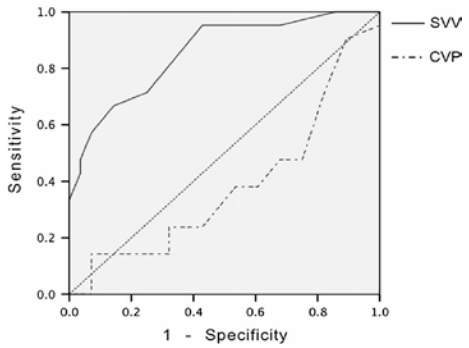
While volume expansion results in an increase CBF, hypervolemia reverses the hypertension-induced benefit on PtiO2.

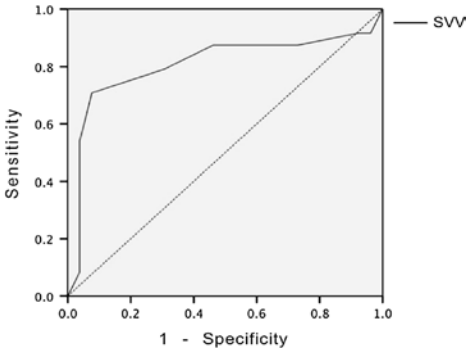
Fluid Responsiveness and Brain Tissue Oxygen Augmentation After Subarachnoid Hemorrhage

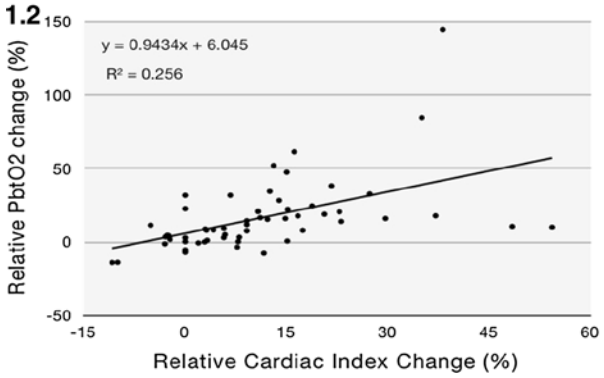
Neurocrit Care (2014) 20:247–254

Pedro Kurtz · Raimund Helbok · Sang-bae Ko · Jan Claassen · J. Michael Schmidt · Luis Fernandez · R. Morgan Stuart · E. Sander Connolly · Neeraj Badjatia · Stephan A. Mayer · Kiwon Lee



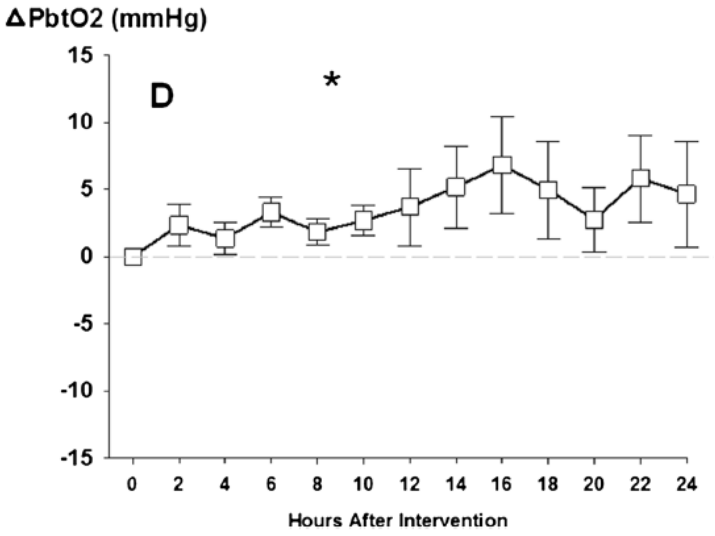


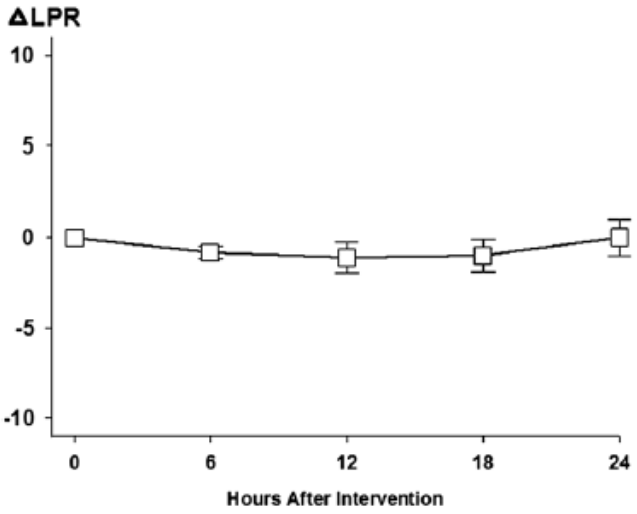




Augmentation of CI can improve cerebral oxygenation after SAH.

High dose erythropoietin increases brain tissue oxygen tension in severe vasospasm after subarachnoid hemorrhage.
Helbok R *et al*. BMC Neurol 2012; 12: 32.





EPO increases PbtO2 in poor grade SAH patients with severe cerebral vasospasm.
No clear effect on metabolism or outcome.

The Effect of Packed Red Blood Cell Transfusion on Cerebral Oxygenation and Metabolism After Subarachnoid Hemorrhage

Pedro Kurtz[1] · Raimund Helbok[2] · Jan Claassen[3] · J. Michael Schmidt[4] · Luis Fernandez[4] · R. Morgan Stuart[5] · E. Sander Connolly[5] · Kiwon Lee[6] · Stephan A. Mayer[7] · Neeraj Badjatia[8]

Neurocrit Care (2016) 24:118–121

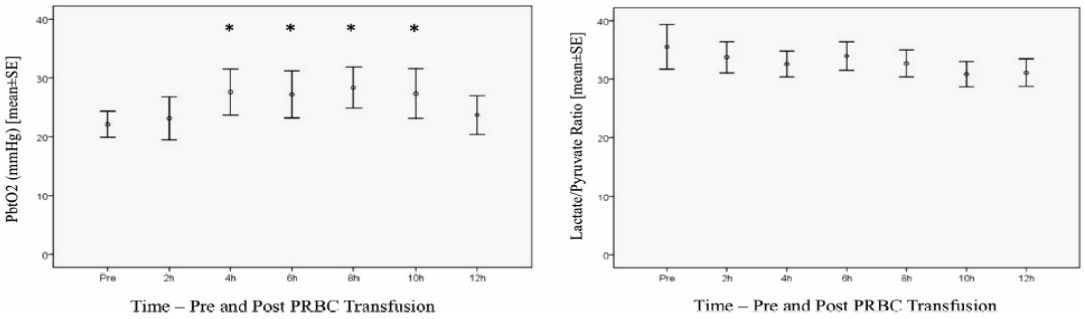


Fig. 1 Evolution of $PbtO_2$ and LPR, at baseline and for 12 h post-transfusion. The data are presented as the mean ± the standard error of the mean (SEM); $*P < 0.05$

Table 1 Physiological parameters

Variable	Baseline	Post-transfusion
Hemoglobin (g/dL)	8.1 (1.1)	10.3 (0.9)
Cerebral perfusion pressure (mmHg)	85.9 (17.3)	98.4 (19.6)
Systemic glucose (mg/dL)	131.2 (40.4)	145.8 (32.8)
End-tidal CO_2 (mmHg)	30.6 (5.6)	30.2 (4.9)
Oxygen saturation (%)	99.2 (1.2)	99.4 (1.2)
$PbtO_2$ (mmHg)	20.1 (13.7)	24.9 (15.2)
LPR	36.6 (15.9)	35.5 (16.4)
Lactate (mmol/L)	3.8 (1.9)	4.1 (2.2)
Pyruvate (mmol/L)	119.0 (65.9)	123.8 (56.0)
Glucose (mmol/L)	1.3 (1.3)	1.2 (0.7)

Continuous Monitoring of Cerebrovascular Autoregulation After Subarachnoid Hemorrhage by Brain Tissue Oxygen Pressure Reactivity and Its Relation to Delayed Cerebral Infarction

(*Stroke*. 2007; 38:981-986.)

Matthias Jaeger, MD; Martin U. Schuhmann, MD, PhD; Martin Soehle, MD; Christoph Nagel, MD; Jürgen Meixensberger, MD, PhD

Index of PtiO2 Pressure Reactivity For Determining Autoregulation

The index of PtiO2 pressure reactivity (ORx) was calculated as the moving linear (Pearson's) correlation coefficient between values of CPP and PtiO2 from the previous 60 minutes of monitoring.

Variable	Noninfarction Group (n=47)	Infarction Group (n=20)	*P*
CPP, mm Hg	81.1±12.1	82.8±11.4	0.43
ICP, mm Hg	12.2±3.9	14.4±5.2	0.10
$PtiO_2$, mm Hg	23.9±5.8	20.8±5.0	0.06
ORx	0.23±0.14	0.43±0.09	0.0000002

TABLE 3. Likelihood of Delayed Infarction Among Suggested Thresholds of ORx From Days 5 and 6

Variable	Noninfarction Group (n=42)	Infarction Group (n=19)	Percentage With Delayed Infarction
ORx <0.25	21	2	9
0.25 <ORx <0.40	14	6	30
ORx >0.40	7	11	61

In summary, continuous monitoring of ORx allows detection of impaired autoregulation after SAH. Persistent autoregulatory failure is independently associated with the occurrence of delayed cerebral infarction and seems to be an important cofactor in addition to vasospasm itself.

High cerebral perfusion pressure improves low values of local brain tissue O2 tension (PtiO2) in focal lesions.
Stocchetti et al. Acta Neurochir Suppl. 1998; 71:162-5

In ischemic areas PtiO2 is dependent on CPP suggesting both a derangement of pressure autoregulation and high regional cerebrovascular resistences (CVRs).

Low PtiO2 was associated with normal CPP, thus indicating that CPP could be an inadequate estimate of rCBF in focal ischemic areas.

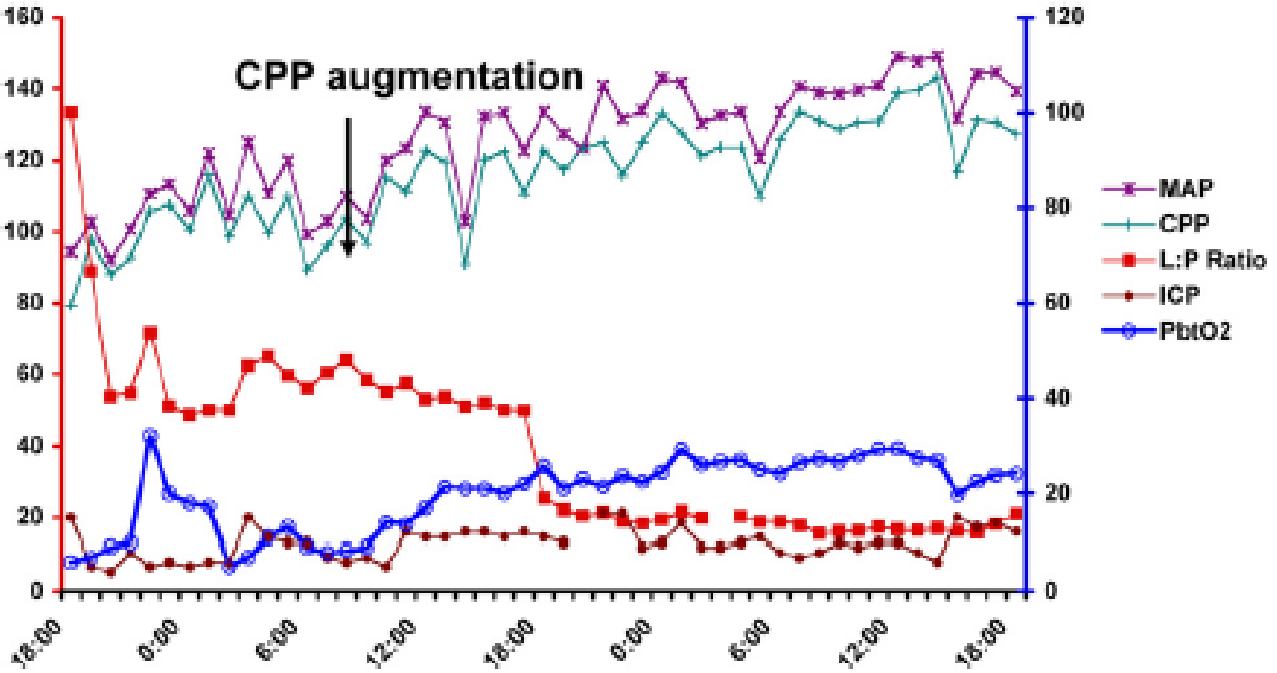
Arterial hypertension, capable of increasing CPP above normal values, appeared useful in normalizing tissue oxygenation in ischemic areas.

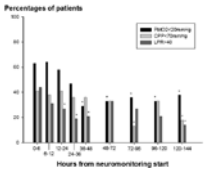
Multimodal Monitoring in Subarachnoid Hemorrhage

Danielle K. Sandsmark, MD, PhD; Monisha A. Kumar, MD; Soojin Park, MD; Joshua M. Levine, MD

Table. Cerebral Physiological Monitors Used for SAH

Physiological Parameter	
Intracranial pressure	
Cerebral blood flow	
Cerebral oxygenation	
Cerebral metabolism	

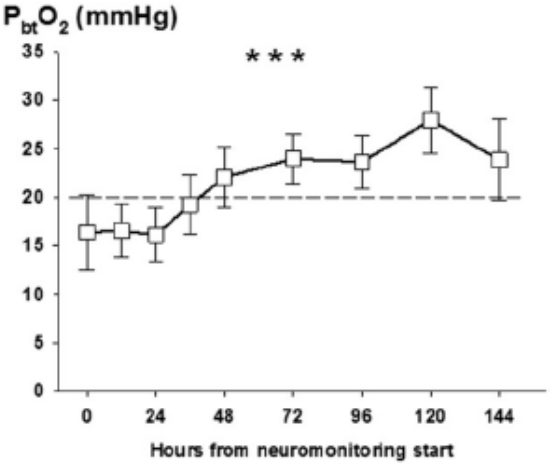


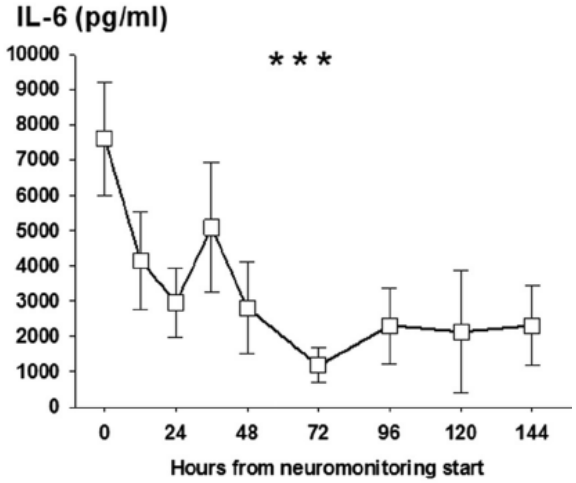


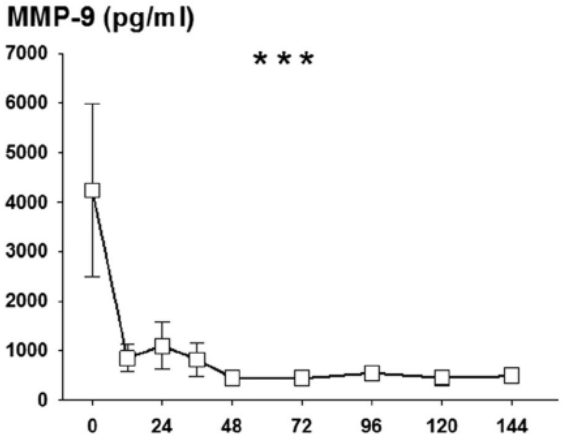
Early brain injury after aneurysmal subarachnoid hemorrhage: a multimodal neuromonitoring study

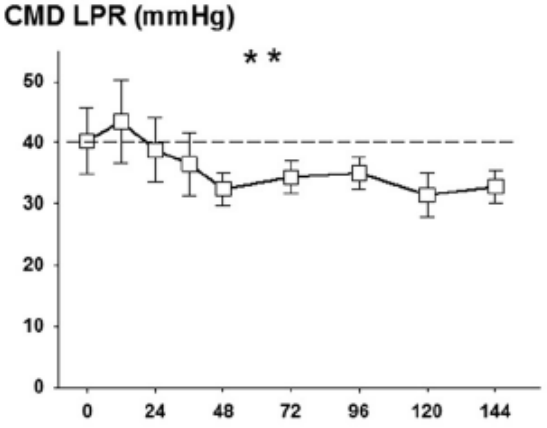
Helbok *et al. Critical Care* (2015) 19:75

A higher pro-inflammatory response was associated with the development of DCI, whereas admission disease severity and early brain tissue hypoxia were associated with higher CMD-MMP-9 and (CMD)-IL-6 levels and a poor functional outcome.









$PbtO_2$ & MMM

Detection of Cerebral Compromise With Multimodality Monitoring in Patients With Subarachnoid Hemorrhage

Chen HI, Stiefel MF, Oddo M, et al. Neurosurgery. 2011;69:53–63.

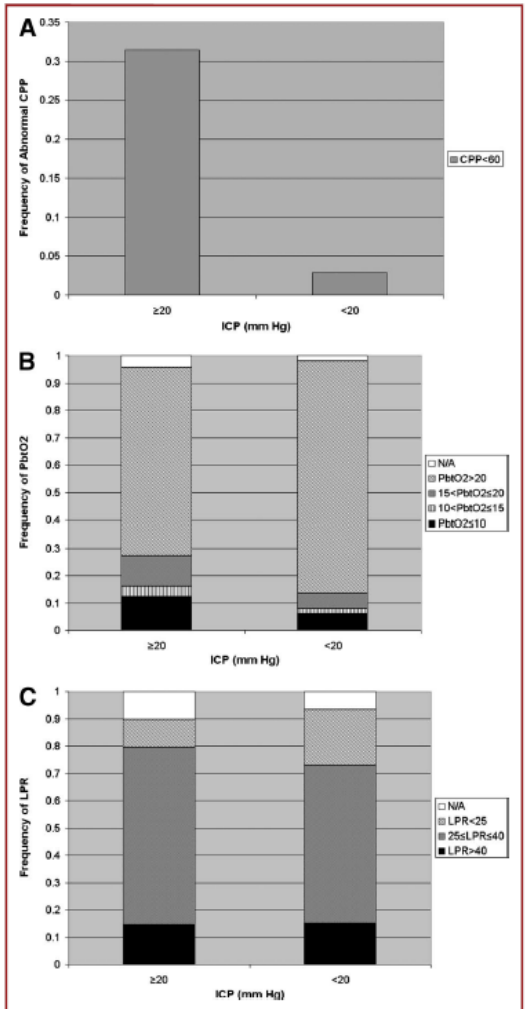


FIGURE 1. *Distribution of physiological parameters for abnormal and normal intracranial pressure (ICP). The frequency of abnormal cerebral perfusion pressure (CPP) (A), brain tissue oxygen ($P_{BT}O_2$) (B), and lactate/pyruvate ratio (LPR) (C) when ICP is increased and normal is depicted. N/A, samples for which complete data are not available.*

TABLE 2. Comparison of Physiological Parameters for Abnormal and Normal Intracranial Pressure[a]

	ICP ≥20 mm Hg	ICP <20 mm Hg	*P* Value
No.	235	1948	
CPP, mm Hg	74 ± 27	96 ± 26	<.001
$P_{bt}O_2$, mm Hg	22 ± 12	29 ± 12	<.001
LPR	35 ± 18	37 ± 38	.30

TABLE 3. Comparison of Physiological Parameters for Abnormal and Normal Cerebral Perfusion Pressure[a]

	CPP <60 mm Hg	CPP ≥60 mm Hg	*P* Value
No.	133	2046	
ICP, mm Hg	30 ± 23	10 ± 7	<.001
$P_{bt}O_2$, mm Hg	17 ± 13	29 ± 12	<.001
LPR	56 ± 47	35 ± 32	<.001

Cerebral hypoxia (PtiO2 < 20 mm Hg) and cerebral energy dysfunction (LPR > 40) may occur despite normal levels of ICP and CPP in the poorgrade SAH population

$PbtO_2$ & Outcome

Brain oxygen tension and outcome in patients with aneurysmal subarachnoid hemorrhage

J. Neurosurg. / Volume 109 / December 2008

ROHAN RAMAKRISHNA, M.D.,[1] MICHAEL STIEFEL, M.D.,[1] JOSHUA UDOTEUK, B.S.,[1] ALEJANDRO SPIOTTA, M.D.,[1] JOSHUA M. LEVINE, M.D.,[1–3] W. ANDREW KOFKE, M.D.,[1,3] ERIC ZAGER, M.D.,[1] WEI YANG, M.S.,[4] AND PETER LEROUX, M.D.[1]

Brain oxygen tension in SAH

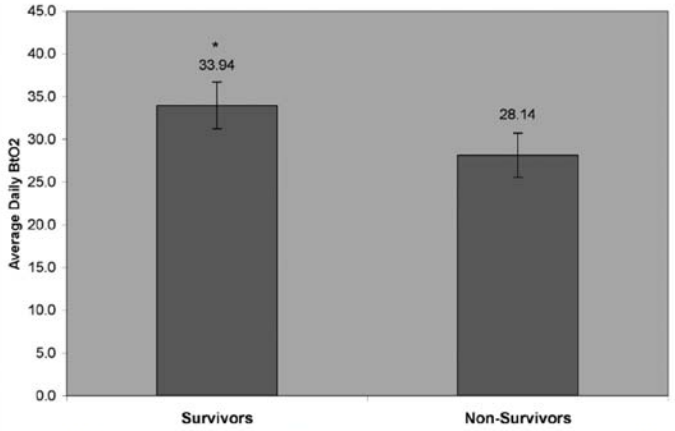


FIG. 1. Histogram illustrating mean daily $PbtO_2$ (BtO2) values in survivors and nonsurvivors. *p = 0.05.

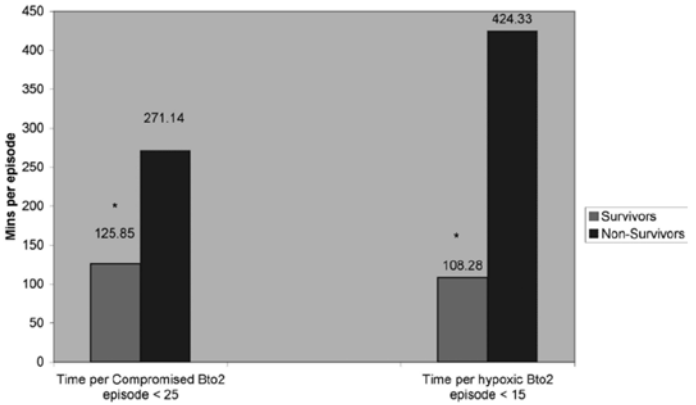


FIG. 2. Histogram illustrating relationship between survival after SAH and mean time of compromised cerebral oxygenation (< 25 mm Hg) and cerebral hypoxia (< 15 mm Hg). *p < 0.05.

TABLE 4

*Brain oxygen tension values stratified according to mean CPP**

Mean CPP (mm Hg)	Mean $PbtO_2$		Mean Min $PbtO_2$	
	Survivors	Nonsurvivors	Survivors	Nonsurvivors
<70 (0 survivors & 5 nonsurvivors)	NA	13.17 ± 6.60	NA	3.95 ± 3.40
70–80 (4 survivors & 4 nonsurvivors)	33.12 ± 12.28	30.03 ± 8.00	18.57 ± 7.83	19.22 ± 7.93
80–90 (6 survivors & 7 nonsurvivors)	36.05 ± 4.80	33.17 ± 3.79	25.04 ± 3.52	21.12 ± 3.76
>90 (11 survivors & 9 nonsurvivors)	33.09 ± 2.39	31.72 ± 2.23	19.56 ± 1.46	18.72 ± 1.90

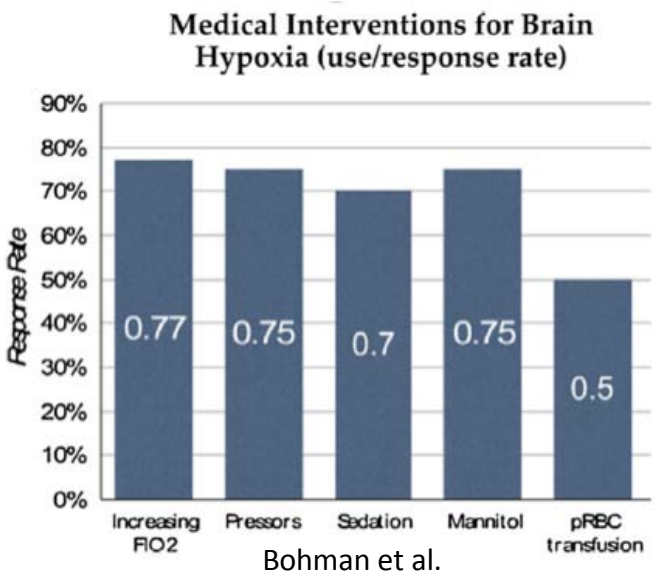
Patients who die after aneurysmal SAH tend to have lower mean PbtO2 levels and a greater duration of compromised PbtO2 during their hospital course than survivors of SAH.

$PbtO_2$ & Outcome

Regional Brain Monitoring in the Neurocritical Care Unit

Neurocrit Care (2015) 22:348–359

Jennifer Frontera[1] · Wendy Ziai[2] · Kristine O'Phelan[3] · Peter D. Leroux[4] · Peter J. Kirkpatrick[5] · Michael N. Diringer[6] · Jose I. Suarez[7] · the Second Neurocritical Care Research Conference Investigators



Bohman et al.

Medical interventions other than those to treat ICP and CPP can improve PbtO2.

Successful medical treatment of brain hypoxia was associated with decreased mortality.

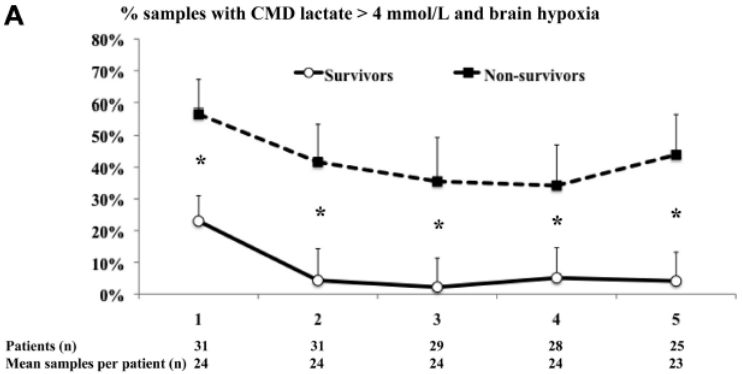
$PbtO_2$ & Outcome

Brain Lactate Metabolism in Humans With Subarachnoid Hemorrhage

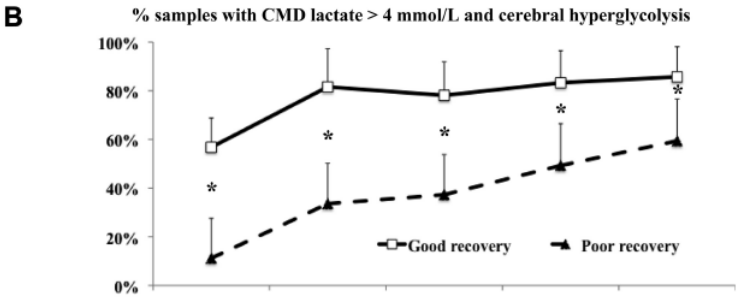
Mauro Oddo, MD et al.

(*Stroke*. 2012;43:1418-1421.)

A



B



Elevated Brain Lactate Pattern	Odds Ratio	Confidence Interval	*P*
Elevated CMD–lactate >4 mmol/L, hyperglycolytic	1.49	1.08–2.05	0.016
Elevated CMD–lactate >4 mmol/L, hypoxic	0.78	0.59–1.03	0.08

CMD indicates cerebral microdialysis.

Table 1. Associations of Brain Lactate Metabolism With Outcome

	Mortality			6-Mo Outcome Among Survivors		
Variable	Survivors N = 19/31 (61%)	Nonsurvivors N = 12/31 (39%)	*P* Value	Good Outcome N = 12/19 (63%)	Poor Outcome N = 7/19 (37%)	*P* Value
CMD–lactate >4 mmol/L	29 (8%–60%)	68 (59%–100%)	0.02	29 (11%–65%)	24 (2%–66%)	0.45
Hypoxic	9 (3%–17%)	28 (9%–95%)	0.002	11 (4%–17%)	4 (1%–53%)	0.46
Hyperglycolytic	88 (27%–99%)	13 (1%–87%)	0.07	97 (87%–100%)	30 (10%–74%)	0.007
N of valid samples	158 (100–166)	100 (54–137)	0.06	155 (87–165)	153 (48–188)	0.89
Duration of brain monitoring, d	7 (7–7)	5 (4–7)	0.13	7 (7–7)	7 (6–7)	0.77

Hypoxic lactate production was higher among non-survivors than survivors (figure A)
Hyperglycolytic lactate was associated with better long-term recovery (figure B)

$PbtO_2$ & Outcome

Poor-grade aneurysmal subarachnoid hemorrhage: relationship of cerebral metabolism to outcome

ASITA SARRAFZADEH, M.D., DANIEL HAUX, M.D., INGEBORG KÜCHLER, PH.D., WOLFGANG R. LANKSCH, M.D., PH.D., AND ANDREAS W. UNTERBERG, M.D., PH.D.

J Neurosurg 100:400–406, 2004

Ischemic neurochemical patterns:

lactate/pyruvate ratios

cerebral lactate concentrations

↑

DCI

Cerebral glucose levels

PbtO2 (<20 mmHg)

↓

Unfavorable outcome.

detected before the occurrence of delayed cerebral ischemia

Monitoring brain tissue oxymetry: Will it change management of critically ill neurologic patients?

Journal of the Neurological Sciences 261 (2007) 1–9

Anna Teresa Mazzeo [a,*], Ross Bullock [b,1]

Table 2
Proposed clinical indications of brain tissue oxygen monitoring

1. Understanding pathophysiology of neuro-injury
2. Recognition of impending ischemia
3. Guiding management and providing feedback to intervention
4. Targeting therapy towards improved cerebral oxygenation
5. Autoregulation assessment
6. Predicting prognosis

Table 1
Brain oxygen pressure and outcome versus other monitored parameters

	Good outcome	Moderate/ severe disability	Death/vegetative
	$PtiO_2$>35 mmHg	$PtiO_2$=26–35 mmHg	$PtiO_2$=25 mmHg
Brain pO_2 (mmHg)	39±4	31±5	19±8
Brain pCO_2 (mmHg)	50±8	47±2	64±21
Brain pH	7.14±0.12	7.11±0.12	6.85±0.41
Dialysate glucose (μmol/l)	986±321	891±350	639±223
Dialysate lactate (μmol/l)	1031±417	1180±524	1642±682
CBF (ml/100 g/min)	34.5±14	22±4	16±8

Perfusion rate for microdialysis was 2 μl/min (modified from Zauner et al. [17]).

$PbtO_2$ guided therapy

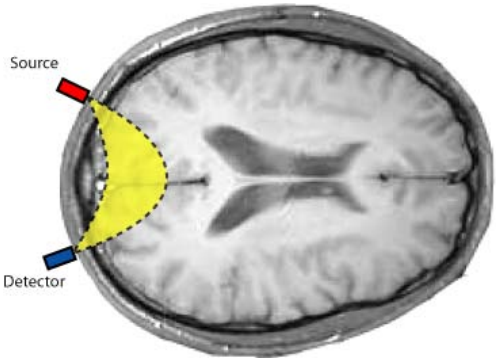
$P_{ti}O_2$ < 20	
O_2 Challenge (Increase FiO_2 transiently to 100%)	
$P_{ti}O_2$ increases	$P_{ti}O_2$ does not change
Preserved system function	System malfunction or probe place into hemorrhagic areas or infarcted tissue
Keep PaO_2 = 80 – 100 mmHg and $PaCO_2$ = 35 – 45 mmHg	Follow-up computed tomography scan may be necessary to confirm probe location
Avoid normabaric hyperoxia (i.e. PaO_2 > 150mmHg)	Replace $P_{ti}O_2$ catheter
$P_{ti}O_2$ remained < 20	
Cerebral Perfusion Pressure Optimization	
ICP < 20mmHg	ICP ≥ 20mmHg
Monitor Fluid Status (e.g. PiCCO Plus) Maintain euvolemia and increase CPP	Treat intracranial hypertension Keep ICP < 20mmHg
MAP Challenge	**Consider re-image** (non-contrast CT head) **CSF drainage** **Surgical drainage** of occupying lesions
Increase MAP by 10mmHg	**Head of bed elevation** (between 30° and 45°) **Normoventilation** ($PaCO_2$: 35-40mmHg) **Normothermia** (< 37.5°C) **Sedation and analgesia** (RASS -5) **Hypertonic agents** Mannitol 1g/Kg or hypertonic saline (e.g. 23.5% saline, 2ml/Kg)
ICP remains stable or decreases / ICP increases	
Autoregulation preserved / Autoregulation impaired	**Refractory Cases:** - **Early decompressive craniectomy + duroplasty** (< 48h of SAH) - **Mild hypothermia** (between 32°C and 34°C) - **Barbiturate**
Increase CPP up to SBP = 200mmHg / Keep CPP = 60 – 70 mmHg	
$P_{ti}O_2$ remained < 20	
Decrease Cerebral Metabolic Rate of Oxygen ($CMRO_2$)	
Treat pain, agitation, fever, shivering, seizures	
$P_{ti}O_2$ remained < 20	
Increase Oxygen Delivery	
Hgb > 9mg/dL	Hgb < 9mg/dL
Increase cardiac output artificially (e.g. dobutamine, milrinone)	Blood transfusion

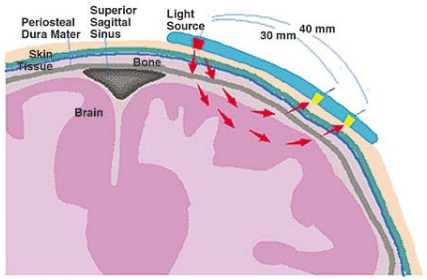
de Oliveira Manoel et al. Critical Care (2016)

NIRS Near-InfraRed Spetroscopy

Non-invasive monitor of cerebral and myocardial oxygen sufficiency and circulatory parameters.
Jobsis FF. Science ;1977, 198:1264-7.

Regional cerebrovascular oxygen saturation measured by optical spectroscopy in humans. McCormick PW. Stroke; 1991, 22:596-602.

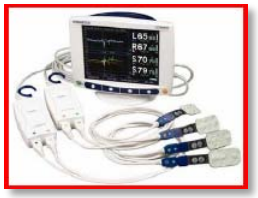




It is a noninvasive technology using near-infrared spectroscopy (NIRS) to monitor regional cerebral tissue oxygen saturation (rSO2).

Currently five FDA cleared devices:

- Somanetics–INVOS



- CASMED–Fore-sight

- Ornim–Cerox

- Nonin-Equanox

- Masimo O_3

Transcranial Cerebral Oximetry Related to Transcranial Doppler After Aneurysmal Subarachnoid Haemorrhage

Acta Neurochir 1998

A. Ekelund, P. Kongstad, H. Säveland, B. Romner, P. Reinstrup, K.-A. Kristiansson, and L. Brandt

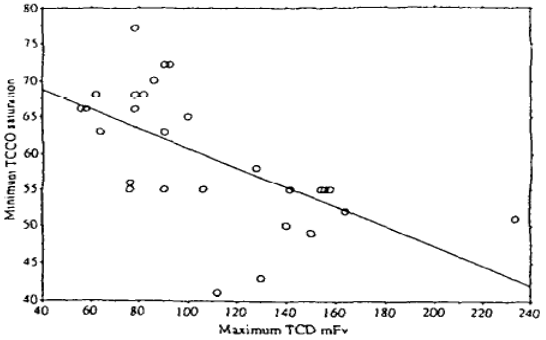


Fig. 1. The correlation between maximum TCD mFV and minimum TCCO saturation in all series, $r = -0.62$, $p < 0.01$

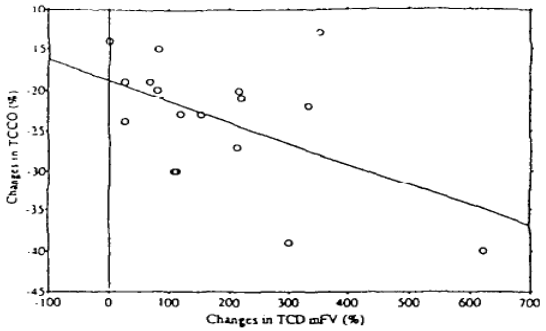


Fig. 2. Changes in TCCO correlated with changes in TCD mFV in the MCA in patients with saturation 63% or less, $r = -0.52$, $p = 0.03$

TCCO and TCD together may develop into methods for detecting reduced cerebral circulation in clinical practice. The clinical benefits of noninvasive methods are obvious, especially the possibility for repeated measurements, prompt access and for the patient, a comfortable bedside examination. However, the clinical *neurological* bedside examination is still the gold standard for correct diagnosis of *symptomatic* vasospasm.

Bedside assessment of cerebral vasospasms after subarachnoid hemorrhage by near infrared time-resolved spectroscopy.
Yokose N. Adv Exp Med Biol. 2010;662:505-11

7 - aSAH patients (WFNS grade V).

SO(2) and TCD performed repeatedly .

In 3 patients, rSO(2) abruptly decreased 5 and 9 after SAH. DCA revealed severe vasospasms in these patients.

TCD detected vasospasm in 2 of 3 cases and failed to do so in one.

TRS-rSO(2) could detect vasospasms after SAH by evaluating the *cortical blood oxygenation*.

Continuous Measurement of Cerebral Oxygenation with Near-Infrared Spectroscopy after Spontaneous Subarachnoid Hemorrhage

Homajoun Maslehaty,

International Scholarly Research Network
ISRN Neurology
Volume 2012, Article ID 907187, 7 pages

Case 2. A 42-year-old male patient presented with SAH H&H grade 2 and Fisher grade 3 due to a ruptured aneurysm of the ACoA (Figure 4(a)).

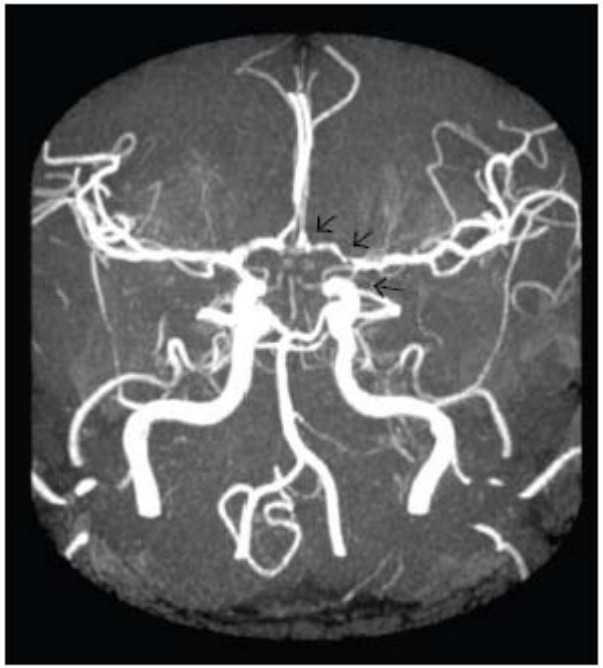
Following embolization the patient suffered from headaches, but he was alert without neurological deficits at all times.

NIRS showed normal and stable rSO2 values (Figure 5).

TCD showed elevated blood flow velocities of the left ACA and MCA up to 220 cm/second.

MRA showed left ICA and ACA spasm (Figure 4(b)), the clinical condition of the patient remained stable without deterioration.

The patient was discharged without neurological deficits.



(b)

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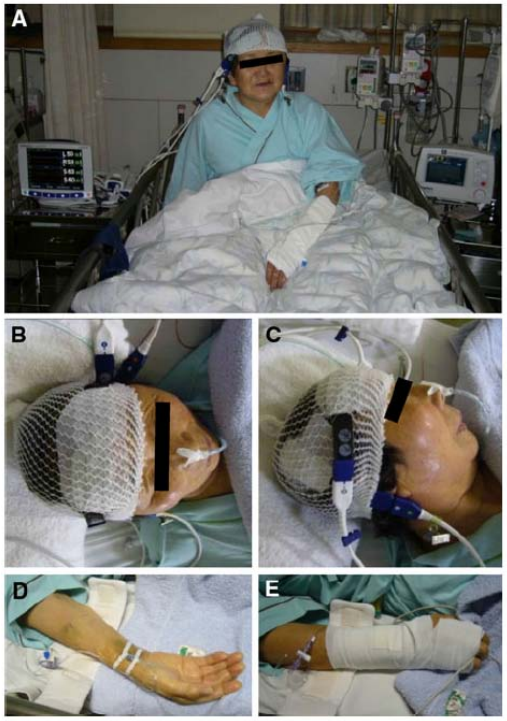
Spedali Civili, University Hospital of Brescia, Italy

Continuous Cardiac Output and Near-Infrared Spectroscopy Monitoring to Assist in Management of Symptomatic Cerebral Vasospasm After Subarachnoid Hemorrhage

Tatsushi Mutoh · Tatsuya Ishikawa · Akifumi Suzuki · Nobuyuki Yasui

neurocritical care society
Neurocrit Care (2010) 13:331–338
DOI 10.1007/s12028-010-9383-9



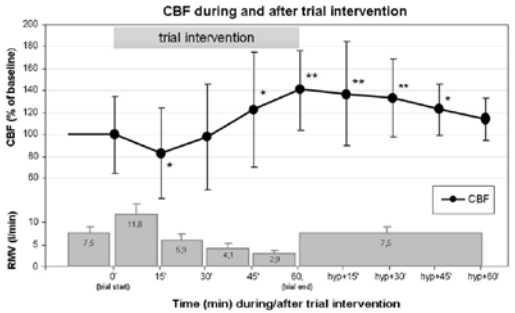
Artery-based pulse contour cardiac output (APCO) NIRS rSO2 monitoring for reversing vasospasm with Dobutamine-induced hyperdynamic therapy.

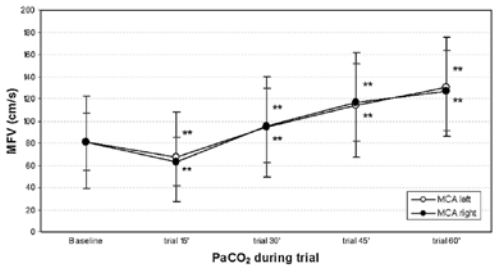
Integrative monitoring with APCO and NIRS may provide continuous, realtime, and clinically relevant information useful for evaluating the effectiveness of medical treatment of vasospasm with DOB.

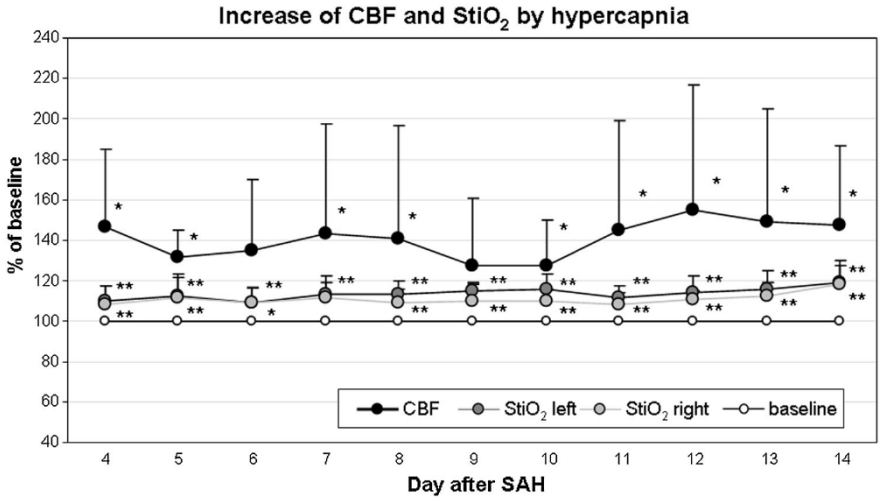
Controlled Hypercapnia Enhances Cerebral Blood Flow and Brain Tissue Oxygenation After Aneurysmal Subarachnoid Hemorrhage: Results of a Phase 1 Study

Thomas Westermaier[1] · Christian Stetter[1] · Ekkehard Kunze[1] · Nadine Willner[1] · Judith Holzmeier[1] · Judith Weiland[1] · Stefan Koehler[1] · Christopher Lotz[2] · Christian Kilgenstein[2] · Ralf-Ingo Ernestus[1] · Norbert Roewer[2] · Ralf Michael Muellenbach[2]

neurocritical care society Neurocrit Care 17 February 2016







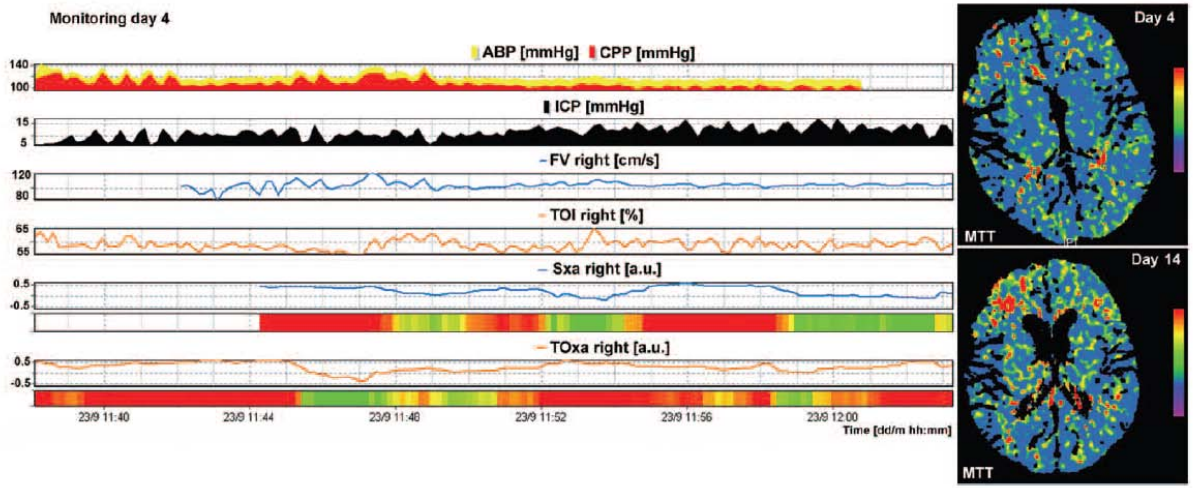
NIRS + Thermal Diffusion, TCD, ICP

Impairment of Cerebral Autoregulation Predicts Delayed Cerebral Ischemia After Subarachnoid Hemorrhage

A Prospective Observational Study

Karol P. Budohoski

Stroke. 2012;43



Assessment of autoregulation using TCD or NIRS can be used to gage the risk of DCI.

Conclusions—Disturbed autoregulation in the first 5 days after SAH significantly increases the risk of DCI. Autoregulatory disturbances can be detected using near-infrared spectroscopy and transcranial Doppler technologies.

Brain tissue oxygen evaluation by wireless near-infrared spectroscopy *Che-Chuan Wang, MD*

JOURNAL OF SURGICAL RESEARCH 200 (2016) 669–675

NIRS ???

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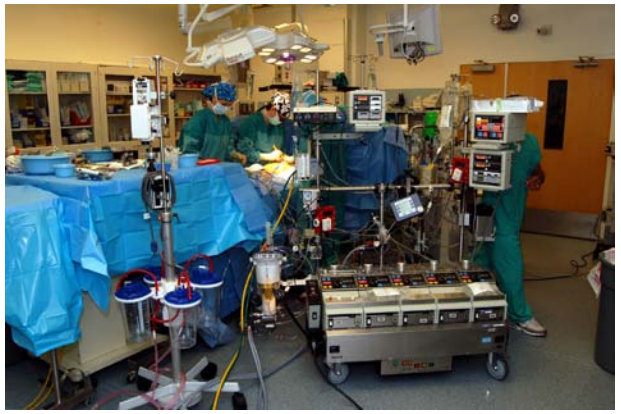
Cerebral Near-Infrared Spectroscopy (NIRS) Monitoring and Neurologic Outcomes in Adult Cardiac Surgery Patients and Neurologic Outcomes: A Systematic Review

Anesth Analg. 2013 March ; 116(3)

Fei Zheng, MD[*], Rosanne Sheinberg, MD[*], May Sann Yee, MD[*], Masa Ono, MD, PhD[†], Yueyging Zheng, MD[‡], and Charles W. Hogue, MD[*]

Conclusions—Reductions in $rScO_2$ during cardiac surgery may identify CPB cannula malposition, particularly during aortic surgery. Only low-level evidence links low $rScO_2$ during cardiac surgery to postoperative neurologic complications, and data are insufficient to conclude that interventions to improve $rScO_2$ desaturation prevent stroke or POCD.



Should Cerebral Near-infrared Spectroscopy be Standard of Care in Adult Cardiac Surgery?

Priscilla J.W. Bevan, MBChB * Heart, Lung and Circulation (2015) **24**, 544–550

Studies into the clinical efficacy of NIRS monitoring have thus far failed to definitively show that interventions to correct cerebral desaturations improve neurological outcomes.

ORIGINAL ARTICLE

Assessment of cerebral oxygenation in neurocritical care patients: comparison of a new four wavelengths forehead regional saturation in oxygen sensor (EQUANOX®) with brain tissue oxygenation. A prospective observational study

P. ESNAULT [1], H. BORET [1], A. MONTCRIOL [1], E. CARRE [2], B. PRUNET [1], J. BORDES [1]
P. SIMON [1], C. JOUBERT [3], A. DAGAIN [3], E. KAISER [1], E. MEAUDRE [1]

(Minerva Anestesiol 2015;81:876-84)

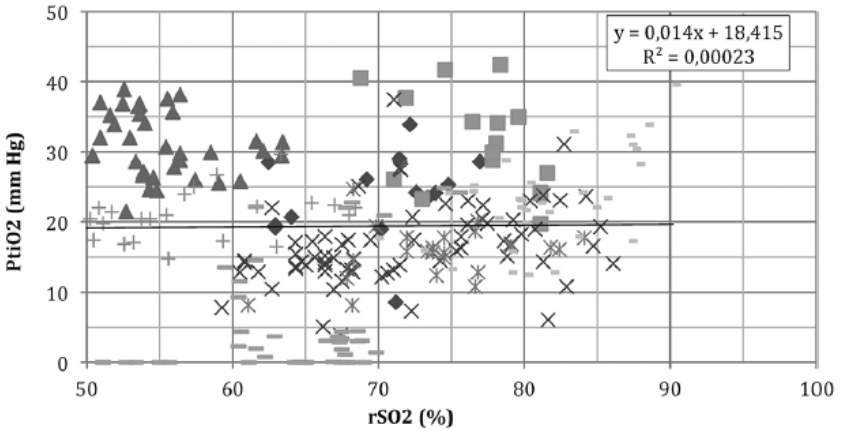


Figure 1.—Graphic showing the absence of correlation between $PbtO_2$ and rSO_2 (pooled values).

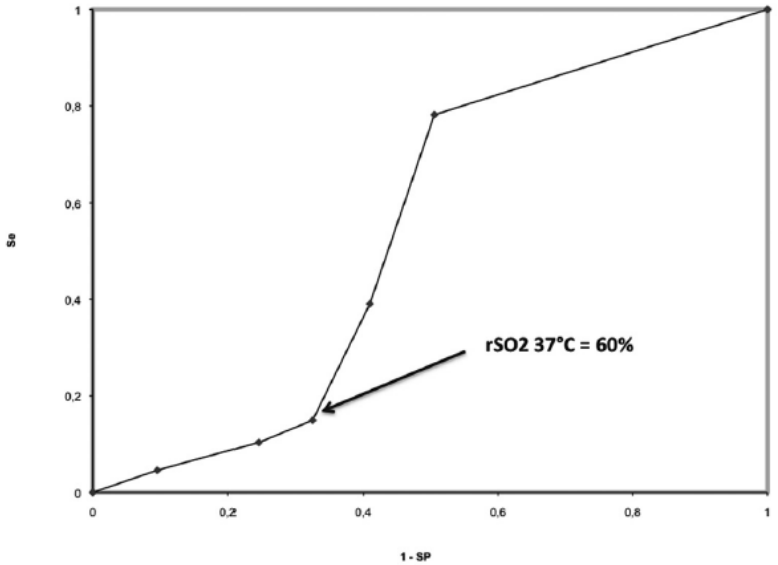


Figure 3.—ROC curve constructed with 5 rSO_2 thresholds (50%, 55%, 60%, 65% and 70%) to detect moderate cerebral hypoxia ($PbtO_2 \leq 15$). AUC=0.54.

Conclusions

these results emphasize the low ability of rSO_2 to detect cerebral hypoxia compared to $PbtO_2$. Even using a third generation NIRS monitoring, rSO_2 cannot be used a substitute for $PbtO_2$ after brain injury.

Continuous-wave near-infrared spectroscopy is not related to brain tissue oxygen tension

J Clin Monit Comput 20 August 2015

Thomas Kerz[1] · Christian Beyer[1] · Alexandra Huthmann[3] · Darius Kalasauskas[1] · Amr Nimer Amr[1] · Stephan Boor[2] · Stefan Welschehold[1]

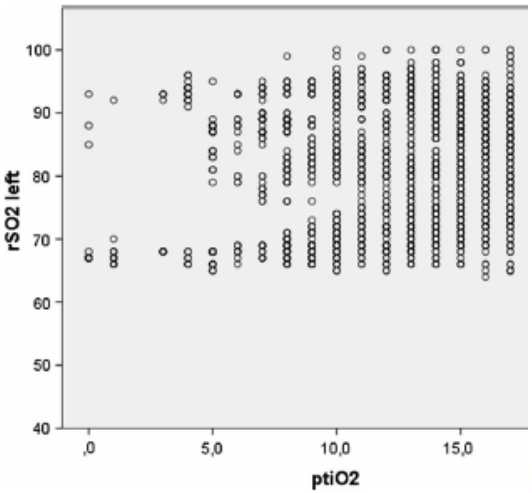


Fig. 5 Scatterplot for all data PtiO2 < 21 mmHg–rSO2 left

There was no correlation between rSO2 mea NIRS, and invasively measured PtiO2 values of 11 critically ill neurological patients. C unable to detect ischemic cerebral episodes PtiO2 value <15 mmHg. CW-NIRS should r the detection of cerebral ischemia.

1 TBI and 10 SAH

Measurement of rSO2 was no better than flipping a coin in the detection of cerebral ischemia.

NCS (Neurocritical Care)

- Significant differences between devices
- There is no established norm as to baseline cerebral saturations
- There is little evidence that the absence of desaturation indicates adequate cerebral blood flow
- NIRS is nonspecific in nature
- extracranial tissues affect NIRS
- too many false-positive readings

Any false negatives ????

Cerebral oximetry in dead subjects

Schwarz GJ. Neurosurg Anesthesiol. 1996

18 dead subjects

15 healthy

mean rSo2 in the dead subjects was 51.0 %

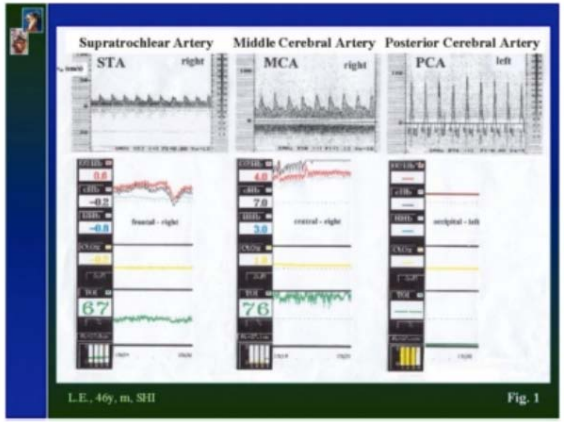
mean rSo, in the control group was 68.4 %

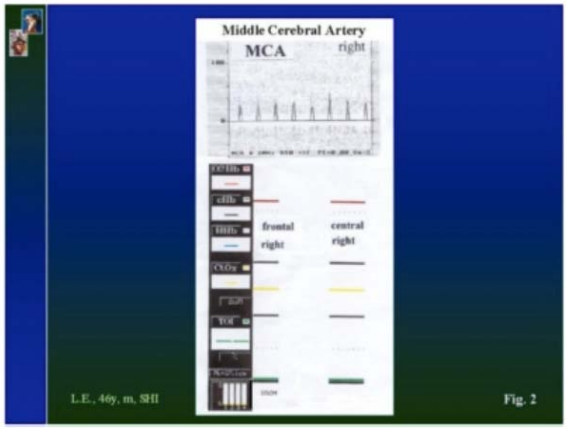
After removal of the brain at autopsy in five of the dead subjects, the rSo2 was 73.4%

Six of the18 dead had values above the lowest values found in the healthy adults (>=60%).

Near-Infrared Spectroscopy (NIRS): Validation and Technical Aspects in Documentation of Brain Death

G Litscher Internet Journal of Neuromonitoring. 2002 Volume 3 Number 1





Forensic Science International

Evaluation of post-mortem oxymetry with reference to the causes of death[1]

Forensic Science International
87 (1997) 201–210

Hitoshi Maeda*, Kazunori Fukita, Shigeki Oritani, Kaori Ishida, Bao-Li Zhu

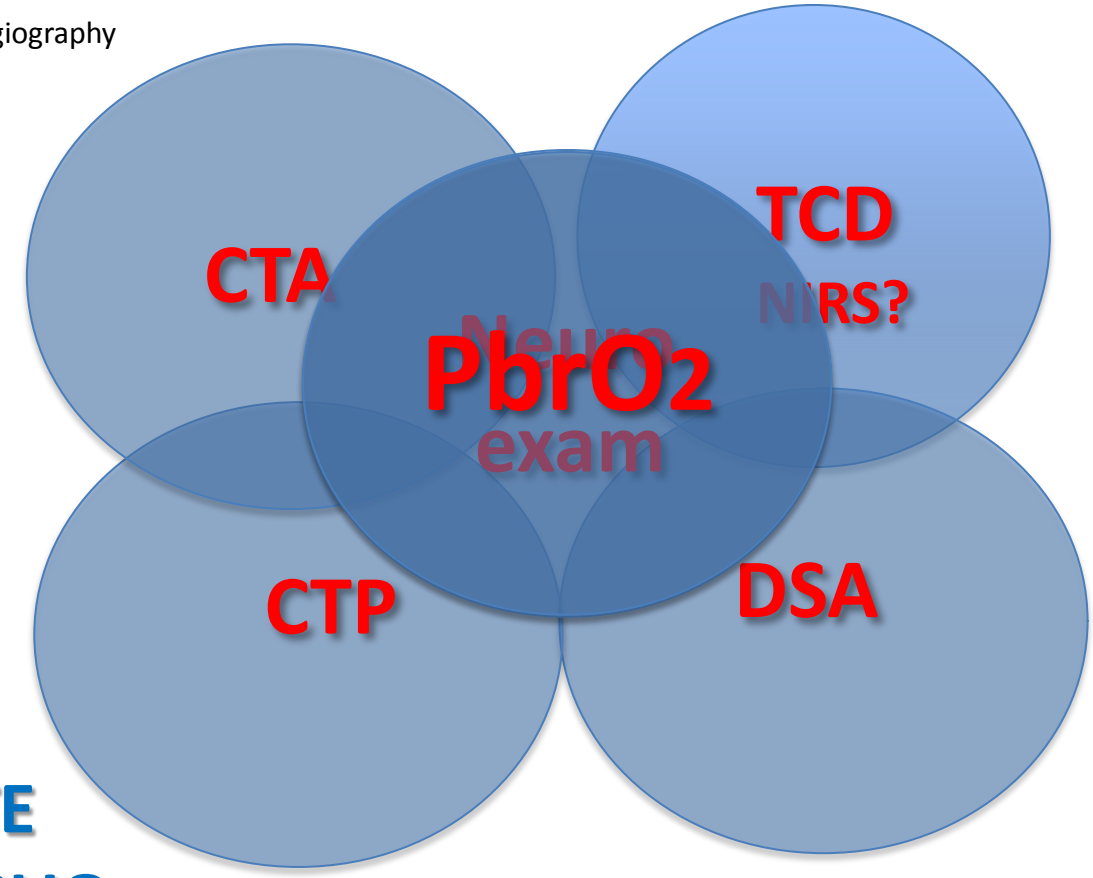
rSO2 saturation during 214 autopsies, values ranged from 0.3% to 95%

Cause of death	t-Hb *average (g/dl)	O_2–Hb (%)	Grouping according to O_2–Hb levels <10% (n=76/45)	10–50% (n=35/35)	>50% (n=10/13)
Blunt injuries (n=45/63)	1.6–29.9 *11.9	0.0–97.7	4/25	18/29	3/9
Stab/incised wounds (n=12/10)	6.6–22.9 *13.6	0.0–89.1	4/5	6/3	2/2
Gun shot head injuries (n=3/0)	7.0–20.2 *13.8	5.0–45.6	1/0	2/0	
Asphyxiation (n=22/6)	6.2–24.0 *17.3	0.0–50.5	18/5	3/1	1/0
Drowning (n=12/1)	3.8–27.1 *14.3	0.0–19.0	10/1	2/0	
Poisoning/drug shock (n=8/2)	8.8–25.4 *21.2	0.4–35.2	7/1	1/1	
Cold exposure (n=5/1)	6.0–22.9 *14.8	32.5–80.8		1/0	4/1
Other traumas[a] (n=3/1)	12.6–24.2 *19.3	2.8–5.2	3/1		
Natural diseases (n=11/9)	1.4–23.7 *12.5	0.1–93.5	9/7	2/1	0/1

CAN WE TAKE ANYTHING HOME ???

TCD - Transcranial Doppler
CTA - Angio CT
CTP - Perfusion CT
DSA - Digital Subtraction Angiography

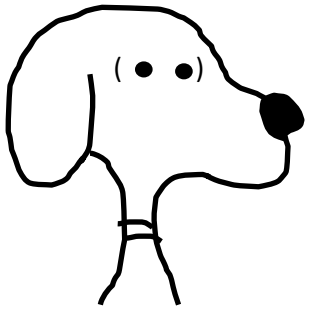
PAZIENTE SVEGLIO



PAZIENTE NON SVEGLIO

Grazie

Picasso



Christian Rasulo

Grazie

Dr. Frank Rasulo
ANESTHESIA, INTENSIVE CARE, PERIOPERATIVE CARE MEDICINE
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Original Investigation JAMA Neurology May 2016 Volume 73, Number 5

Pathophysiologic Mechanisms of Cerebral Ischemia and Diffusion Hypoxia in Traumatic Brain Injury

Tonny V. Veenith, FRCA; Eleanor L. Carter, FRCA; Thomas Geeraerts, PhD; Julia Grossac, MD; Virginia F. J. Newcombe, PhD; Joanne Outtrim, MSc; Gloria S. Gee, AS; Victoria Lupson, BSc; Rob Smith, PhD; Franklin I. Aigbirhio, PhD; Tim D. Fryer, PhD; Young T. Hong, PhD; David K. Menon, PhD; Jonathan P. Coles, PhD

Figure 1. Evidence of Cerebral Ischemia Using Oxygen 15–Labeled Positron Emission Tomography After Head Injury

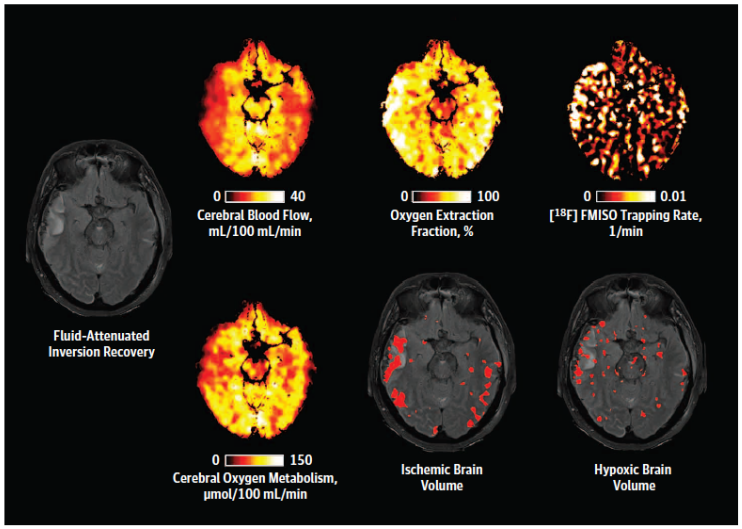
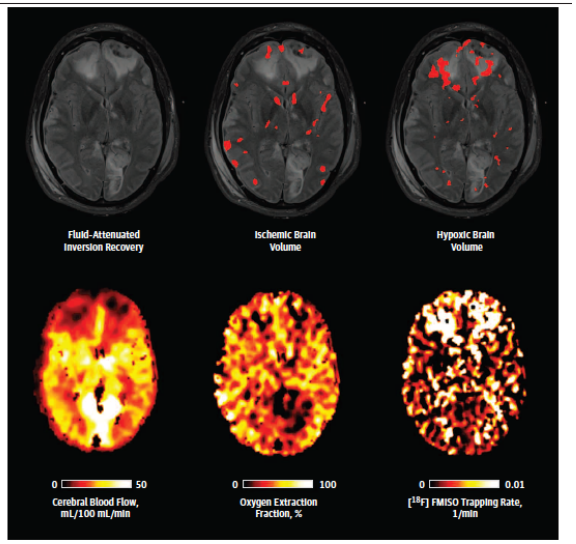


Figure 3. Evidence of Tissue Hypoxia Using Fluorine 18–Labeled Fluoromisonidazole ([18F]FMISO) Positron Emission Tomography



fluoromisonidazole ([18F]FMISO)

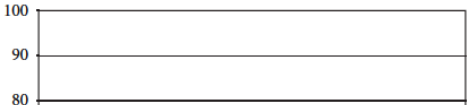
oxygen 15–labeled PET

CONCLUSIONS AND RELEVANCE Tissue hypoxia is not confined to regions with structural abnormality and can occur in the absence of conventional macrovascular ischemia.

Continuous Measurement of Cerebral Oxygenation with Near-Infrared Spectroscopy after Spontaneous Subarachnoid Hemorrhage

Homajoun Maslehaty,

International Scholarly Research Network
ISRN Neurology
Volume 2012, Article ID 907187, 7 pages



Case 1. A 70-year-old female presented with SAH H&H grade 5, Fisher grade 4 due to a ruptured left sided PCA aneurysm.

TCD showed elevated blood flow velocities (200 cm/sec) of both MCA and ACA arteries despite triple H therapy and nifedipine.

NIRS showed left-sided decrease of rSO2 below 40% on day 5 after onset (Figure 2).

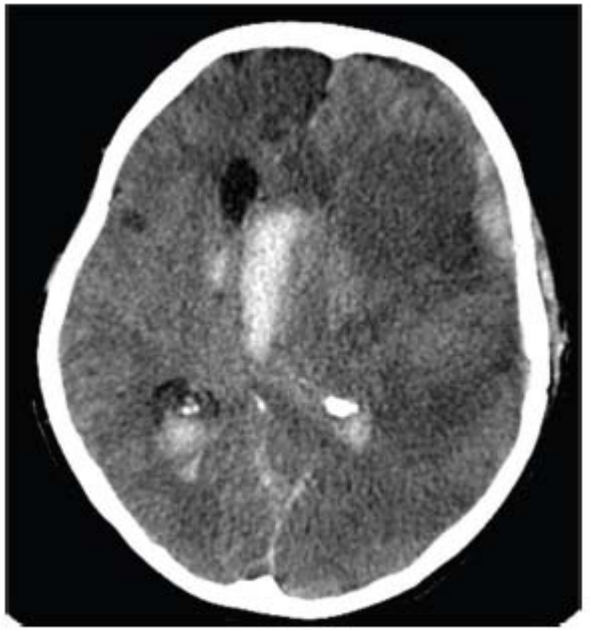
Left frontal applied ICP probe showed no significant changes at the same time (ICP 11mmHg, CPP 118 mmHg).

Subsequently performed native CT and PW-CT scans showed neither perfusion deficits nor ischemic stroke (Figures 3(a) and 3(b)).

Two days later, ICP increased slowly and reached the maximum of 39mmHg on day twelve after onset.

In parallel to this right-sided rSO2, values decreased as well.

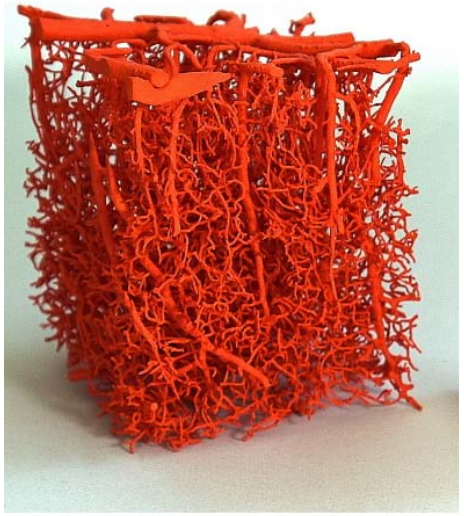
Newly performed CT scan showed a marked left hemispheric ischemic stroke with shift of the midline strictures and signs of brain herniation (Figure 3(c)).



(c)

In consideration of the poor clinical condition, the age, and occurrence of distinct ischemic stroke, we decided to limit the therapy. The patient died on day twelve after onset.

Basic mechanisms of diffusive and diffusion-related oxygen transport in biological systems: a review.
Groebe K, Thews G (1992) Adv Exp Med Biol 317:21–33



There is “acellular blood flow” in the brain.

Studies in the rat cortex show that up to 20% of capillaries may not contain erythrocytes.

Therefore non-Hgb O2 transport may be important, and since the driving force for O2 delivery to the cells (mitochondria) is the O2 tension gradient, it provides a rationale for a clinical use of therapy designed to improve brain oxygenation.

Occurrence of Vasospasm and Infarction in Relation to a Focal Monitoring Sensor in Patients after SAH: Placing a Bet when Placing a Probe?

PLOS ONE May 2013 | Volume 8 | Issue 5

Christian T. Ulrich[1]*, Christian Fung[1], Hartmut Vatter[2], Matthias Setzer[2], Erdem Gueresir[2], Volker Seifert[2], Juergen Beck[1], Andreas Raabe[1]

Table 2. Number of patients according to aneurysm type, location of the probe and occurrence of infarction.

Aneurysm location	Total aneurysms	CVS in probe area	CVS outside probe area	Infarct inside probe territory	Infarct outside probe territory	No infarct
MCA right	6	6	0	3	0	3
MCA left	8	7	1	5	1	2
ICA right	15	12	3	10	1	4
ICA left	15	14	1	8	0	7
A1CA right	3	1	2	1	2	0
A1CA left	1	1	0	1	0	0
AcoA, A2CA	33	25	8	14	4	15
VBA	19	8	11	3	10	6

The probability that a single focal probe will be situated in the territory of severe CVS and infarction varies over a wide range. More reliable CVS or infarction detection was observed in MCA and ICA.

In our opinion, focal ptiO2 or CBF or microdialysis measurements are useful for MCA and ICA aneurysms, but may have a high (25– 50%) failure rate in patients with VBA and ACA aneurysms.

Regional Brain Monitoring in the Neurocritical Care Unit

Neurocrit Care (2015) 22:348–359

Jennifer Frontera[1] · Wendy Ziai[2] · Kristine O'Phelan[3] · Peter D. Leroux[4] · Peter J. Kirkpatrick[5] · Michael N. Diringer[6] · Jose I. Suarez[7] · the Second Neurocritical Care Research Conference Investigators

Table 2 Local and systemic factors that influence brain oxygenation

Local factors
O_2 consumption by neurons and glia
O_2 diffusion conditions/gradients in tissue
Number of perfused capillaries per tissue volume
Length and diameter of perfused capillaries
Capillary perfusion rate and microflow pattern
Hemoglobin oxygen release in microcirculation
Systemic factors
Arterial blood pressure
ICP
PaO_2
$PaCO_2$
pH
Temperature
Blood hemoglobin content
Viscosity
Hematocrit

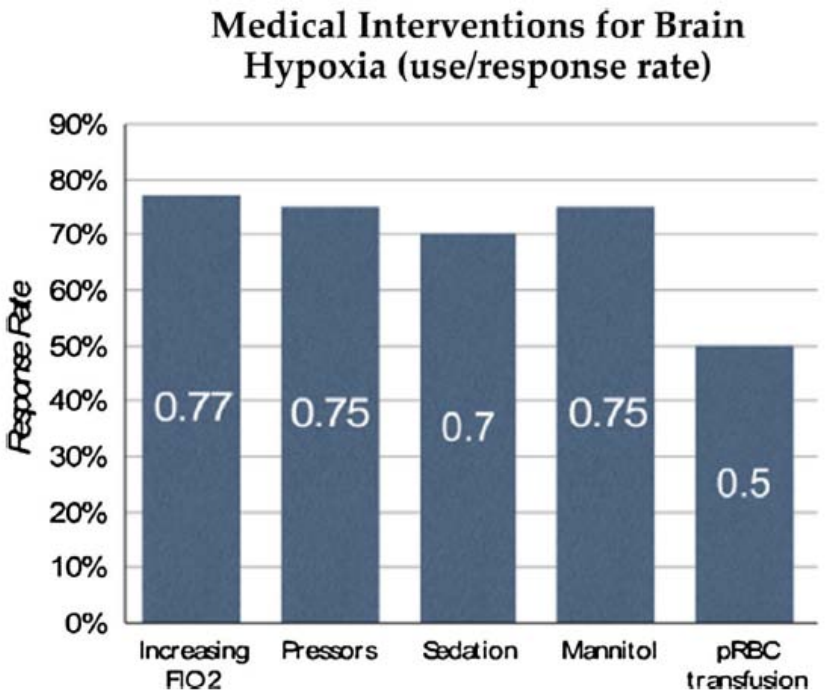


Fig. 1 Medical Interventions for Brain Hypoxia. Figure 2 of Bohman et al. [38]

Bedside Monitoring of Cerebral Blood Oxygenation and Hemodynamics after Aneurysmal Subarachnoid Hemorrhage by Quantitative Time-Resolved Near-Infrared Spectroscopy

WORLD NEUROSURGERY, DOI:10.1016/J.WNEU.2010.02.061

Noriaki Yokose[1], Kaoru Sakatani[1,2,3], Yoshihiro Murata[1], Takayuki Awano[1], Takahiro Igarashi[1], Sin Nakamura[1], Tatsuya Hoshino[1], Yoichi Katayama[1,3]

- CONCLUSION: TR-NIRS detected vasospasm by evaluating the CBO in the cortex and may be more sensitive than TCD, which assesses the blood flow velocity in the M1 portion. The cerebral oxygen metabolism in SAH might be reduced by brain damage due to aneurysmal rupture.

Brain tissue oxygen evaluation by wireless near-infrared spectroscopy

Che-Chuan Wang, MD

JOURNAL OF SURGICAL RESEARCH 200 (2016) 669–675

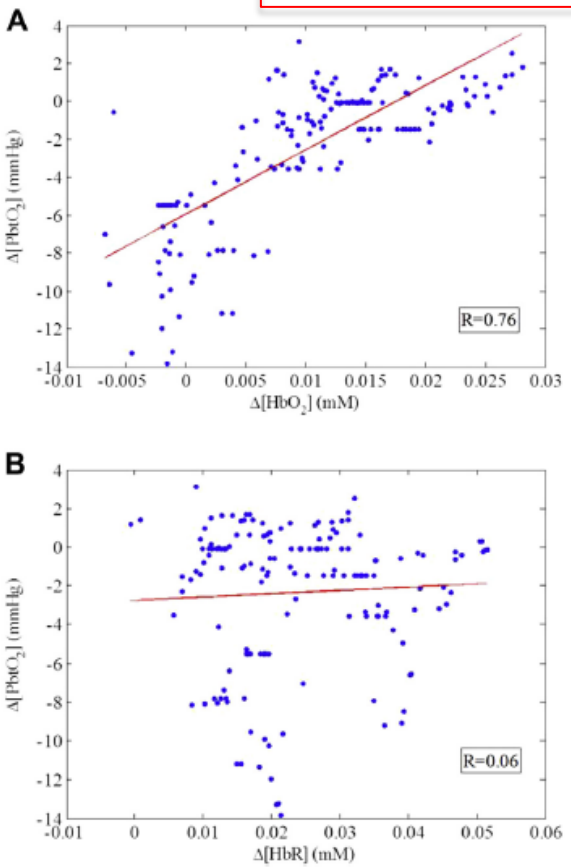
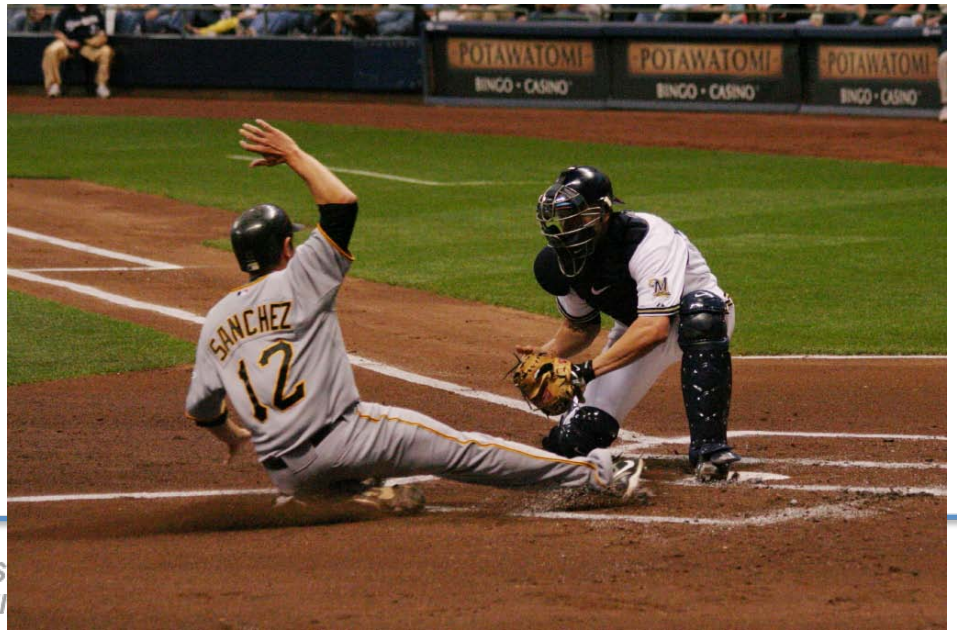


Fig. 4 – Correlations between changes in partial pressure of oxygen in brain tissue ($\Delta PbtO_2$) and (A) oxyhemoglobin (ΔHbO_2) and (B) deoxyhemoglobin after traumatic brain injury (TBI). (Color version of figure is available online.)

Changes in PbtO2 had a similar tendency with the hemoglobin parameters. There was significant correlation between changes in PbtO2 and HbO2 (correlation . 0.76) but not with changes in HbR (correlation . 0.06).

In different severities of brain injury, changes in HbO_2 are highly and positively correlated to changes in $PbtO_2$. The proposed wireless NIRS system can be used to noninvasively estimate cerebral hypoxia. As such, the relative concentration changes of HbO_2 may be used as the reference parameter to estimate the partial pressure of oxygen in the brain tissue.

Cerebral O_2 Monitoring in SAH



$PbtO_2$ & FiO2

Effect of hyperoxia on cerebral metabolic rate for oxygen measured using positron emission tomography in patients with acute severe head injury

J Neurosurg 106:526–529, 2007

MICHAEL N. DIRINGER, M.D., F.C.C.M.,[1] VENKATESH AIYAGARI, M.B.B.S., D.M.,[1] ALLYSON R. ZAZULIA, M.D.,[1,2] TOM O. VIDEEN, PH.D.,[1,2] AND WILLIAM J. POWERS, M.D.[1–3]

TABLE 1

*Physiological data in five patients with acute TBI before and after ventilation with 100% oxygen**

SvO_2 (%)	74.3 ± 8.2	82.4 ± 7.1
CvO_2 (vol %)	10.85 ± 2.9	11.43 ± 3.1

Although the number of patients we studied was very small, there was not even a hint of a consistent improvement in brain oxygen metabolism. Of course, we cannot rule out the existence of an individual patient who may respond differently; this is an issue that could be addressed with larger studies. Nevertheless, these results do not support the use of 100% oxygen in patients with TBI based on the rationale that it generally improves brain oxygen metabolism.