

1 ST RESIDENTIAL
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 $\begin{array}{c} \mathbf{E} \mathbf{uropean} \ \mathbf{Society} \ \mathbf{of} \ \mathbf{N} \mathbf{e} \mathbf{urosonology} \\ \mathbf{and} \ \mathbf{Cerebral} \ \mathbf{H} \mathbf{e} \mathbf{modynamics} \end{array}$

Bertinoro September 7 -13, 2008

CD monitoring in ICU



NEURO INTENSIVE CARE UNIT- Dep. of Anesthesiology & Intensive Care, Spedali Civili University Hospital of Brescia, ITALY

Monitoring

- Neurophysiologic
- Cerebral Hemodynamic
- Neurochemical & Metabolic



Monitoring CBF (bedside)





Snapshot



Continuous

- Needs validation
- Invasive





Transcranial Doppler Monitoring in the Neuro Critical Care Unit

- ☐ Safe
- Cheap
- Repeatable
- Bedside
- ☐ Good learning curve
- Relyable





TCD in the ICU



- > INTRACRANIAL HYPERTENSION
- > CEREBRAL CIRCULATORY ARREST
- **AUTOREGULATION**
- > STENOSIS

- >SUBARACHNOID HEMORRHAGE
- > HEAD TRAUMA
- > STROKE
- > BRAIN DEATH



VASOSPASM & ANGIOGRAPHY







ANGIOGRAPHY



ANGIOGRAPHY

Neuroradiology

Radiolog

Robert A. Willinsky, MD, FRCPC Steve M. Taylor, BA Karel terBrugge, MD, FRCPC Richard I. Farb, MD, FRCPC George Tomlinson, PhD Walter Montanera, MD, FRCPC Neurologic Complications of Cerebral Angiography: Prospective Analysis of 2,899 Procedures and Review of the Literature¹

39 (1.3%) neurologic complications in 2,899 procedures;

20 (0.7%) transient

5 (0.2%) reversible

14 (0.5%) permanent (strokes)

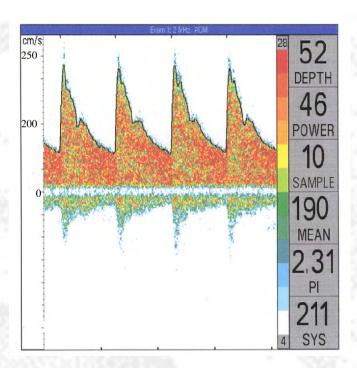
NEUROLOGIC COMPLICATIONS OF CEREBRAL ANGIOGRAPHY

A retrospective study of complication rate and patient risk factors AM Leffers, A Wagner. Acta Radiologica 41 (2000) 204-210

Complication rates for neurologic deficits associated with cerebral angiography in recent studies

Studies	n	All	Persistent
Grzyska et al. (ref. 4)	1095	0.54%	0.45%
Heiserman et al. 1994 (ref. 5)	1000	1%	0.5%
Waugh et al. 1992 (ref. 12)	2075	0.6%	0.3%
Present study	483	2.3%	0.4%







Comparison of TCD with angiography for detection of vasospasm

An inverse relationship between vessel diameter and TCD velocities

Aaslid et al.

FVm > 120 cm/s = mild vasospasm

FVm > 200 cm/s = severe vasospasm

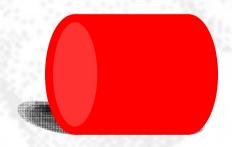
Correlation between FVm and angiographic lumen diameter of MCA

FVm < 120 cm.sec - < 25% narrowing

FVm 120-200 cm.sec - **25-50%** narrowing

FVm < 200 cm.sec - > 50% narrowing

Aaslid et al Sloan et al Vora et al Lindegaard et al







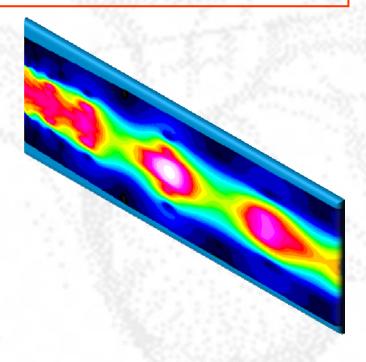


Assessment: Transcranial Doppler ultrasonography

Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology

M. A. Sloan, et al: NEUROLOGY 2004;62:1468-1481

"to review the use of TCD for diagnosis"





Vasospasm after Spontaneous SAH

INDICATION	SENSITIVITY (%)	SPECIFICITY (%)	REFERENCE STANDARD		
Vasospasm after Spontaneous Subarachnoid Hemorrhage			Conventional angiography		
Intracranial ICA	25-30	83-91			
MCA	39-94	70-100			
ACA	13-71	65-100			
VA	44-100	82-88			
ВА	77-100	42-79			
PCA	48-60	78-87			

Copyright 2004 American Academy of Neurology







Courtesy of Andrea Rigamonti

Vasospasm after Spontaneous SAH

INDICATION	SENSITIVITY (%)	SPECIFICITY (%)	REFERENCE STANDARD
Vasospasm after Spontaneous Subarachnoid Hemorrhage	69	83	Conventional angiography
Intracranial ICA	100	97	PCA SIGN SIGN SIGN SIGN SIGN SIGN SIGN SIGN
MCA	100	93	Vertebral Temporal Window
ACA	71	85	Transforaminal Window

Copyright 2004 American Academy of Neurology



TCD and post-traumatic SAH

Predicting Outcome after Traumatic Brain Injury:
Development and International Validation of Prognostic
Scores Based on Admission Characteristics
Ewout W. Steyerberg et al., PLoS Medicine August 2008 | Volume 5 | Issue 8 | e165



Head injury patients with SAH have a worse prognosis than patients without SAH or Vasospasm.



TCD and post-traumatic SAH

CT evidence of SAH following closed head injury occurs in up to 63% of patients

tSAH → VSP→ focal DID
63% 15%



INDICATION	SENSITIVITY (%)	SPECIFICITY (%)	REFERENCE STANDARD
Vasospasm after Traumatic Subarachnoid Hemorrhage			Conventional angiography

Data on sensitivity, specificity and predictive value of TCD for VSP after tSAH are needed.



TCD & Delayed Ischemic Deficits (DID)

Ability of TCD to predict onset of DID's following SAH

DID's occured in 20–30% of SAH pts. within 3–14 days after bleed. Vasospasm-related DIDs were the major cause of bad outcome.

Kassell NF et al.

8 of 21 patients with SAH developed DID. Good correlation between TCD and DID. Sekhar et al.



TCD monitoring and Clinical Decision Making After Subarachnoid Hemorrhage

MJ. McGirt et al. J Stroke Cerbrovasc Disease. March 2003

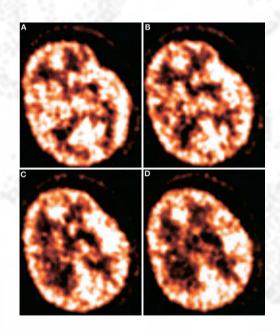
"TCD-defined vasospasm preceded the neurological deficit in 64%, therefore earlier intervention might reduce the incidence of vasospasm-related stroke."

What about the other 36%?



Positron Emission Tomographic Cerebral Perfusion Disturbances And Transcranial Doppler Findings Among Patients With Neurological Deterioration After Subarachnoid Hemorrhage

Pawan S. Minhas et al. *Neurosurgery* 52: 1017-1024, 2003



Regions of cerebral ischemia are not always associated with territories of maximal vasospasm.



TCD Delayed Ischemic Deficits (DID)

The accuracy of TCD to detect vasospasm in patients with aneurysmal subarachnoid hemorrhage

L.Mascia et al. Intensive Care Medicine 2003

MFv threshold > 160 cm/s

Angiog	graphic vasosp	oasm				
Test	Sensitivity	Specificity	PPV	NPV	LR+	LR-
TCD	1.00	0.75	0.72	1.00	4	0
Clinica	ıl vasospasm					
Test	Sensitivity	Specificity	PPV	NPV	LR+	LR-
TCD	1.00	1.00	1.00	1.00	0	0



Vasospasm Probability Index: a combination of transcranial doppler velocities, CBF, and clinical risk factors to predict cerebral vasospasm after aneurysmal SAH.

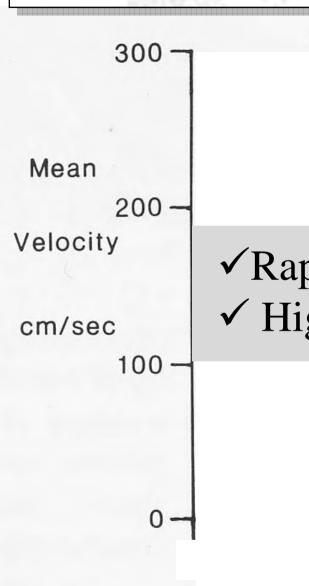
Gonzalez NR et al.. J Neurosurg. 2007 Dec;107(6):1101-12.

The index (combination of predictive factors associated with the development of vasospasm) may become a valuable tool for the clinician to evaluate the individual probability of cerebral vasospasm after aneurysmal SAH.

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MCA – \checkmark Fv <121 - >182 cm/s (low risk for VSP – high risk for VSP)
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- ✓ Rapid rise in Fvm in a few days
- ✓ High Lindegaard ratio

TCD and Multimodal Monitoring

Journal of NeuroEngineering and Rehabilitation

Methodology

Relationship between oxygen supply and cerebral blood flow assessed by transcranial Doppler and near – infrared spectroscopy in healthy subjects during breath – holding Filippo Molinari*¹, William Liboni², Gianfranco Grippi² and Emanuela Negri²



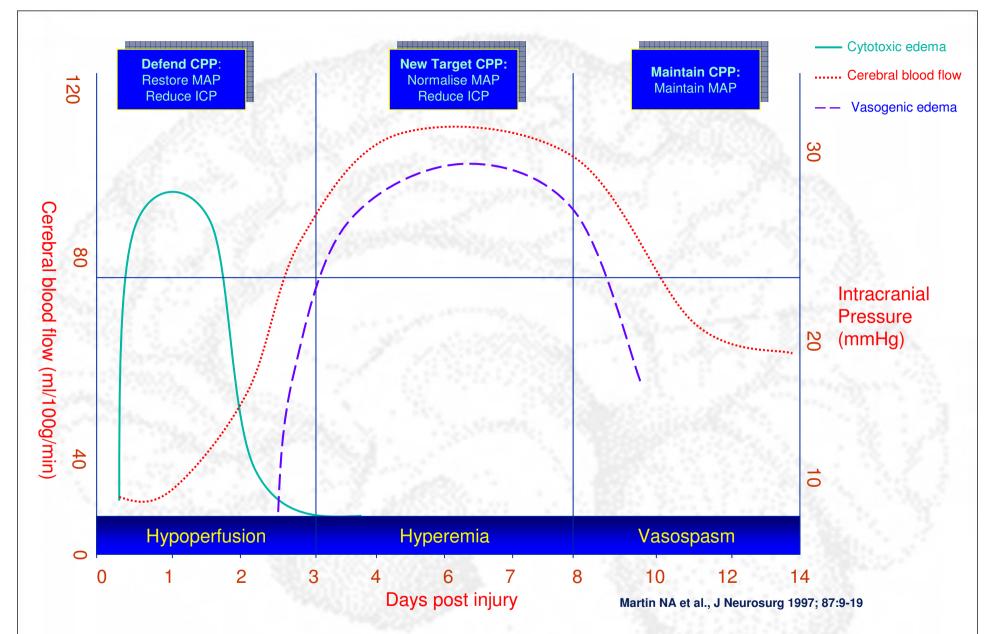
Brain Tissue pO_2 in Relation to Cerebral Perfusion Pressure, TCD Findings and TCD-CO $_2$ -Reactivity After Severe Head Injury

Acta Neurochir (Wien) (1996) 138: 425-434

J. Dings, J. Meixensberger, J. Amschler, B. Hamelbeck, and K. Roosen







FV decreases during the first 48 h after head trauma and subsequently increases between 48–120 h. The ability of TCD to distinguish between the two processes facilitates clinical management.

TCD in Traumatic Brain Injury

Correlation with cerebral perfusion

Persistently low MCA FVm is associated with poor neur. outcome

Serial transcranial Doppler measurements in traumatic brain injury with special focus on the early posttraumatic period. Santbrink H van et al , Acta Neurochir 2002 (Wien) 144:1141–1149

Intracranial blood flow velocity after head injury: relationship to severity of injury, time, neurological status and outcome. Chan KH et al., J Neurol Neurosurg Psychiatry 1992;55:787–791

Evaluating the Outcome of Severe Head Injury With Transcranial Doppler Ultrasonography José A. Moreno et al., Neurosurgical Focus 1999 MCA FVm < 30 - 35 cm/sec

The most reliable indicators of low CPP are low FVd with high PI.



TCD and Intracranial Pressure (ICP)

Correlation with ICP

The gold standard for the measurement of ICP it's invasive measurement.

However there are various situations where a non invasive measurement may be useful...

- mild and moderate head injury
- Ischemic & hemorrhagic stroke, vasospasm
- meningo-encephalytis
- faulty ICP cathters
- outside the ICU (ER, OR, etc.)





The earliest sign of increased ICP is increased pulsatility.





There is a strong correlation between PI and ICP (ICP values> 20 mmHg), and between PI and CPP (CPP values< 70 mmHg).

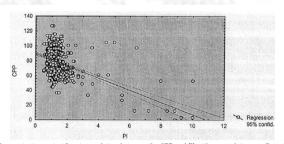
Voulgaris et al.



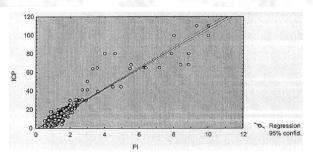
TRANSCRANIAL DOPPLER SONOGRAPHY PULSATILITY INDEX (PI) REFLECTS INTRACRANIAL PRESSURE (ICP)

Johan Bellner, M.D.et al. Surg Neurol 2004;62:45-51

First **prospective** study to investigate the relationship between ICP and TCD derived PI.



Graph demonstrating a significant correlation between the CPP and PI with a correlation coefficient of -0.493 (p < 0.0001) and a correlation formula of: CPP = $89.646 - 8.258 \times PI$. The correlation between CPP and PI is mainly when PI >3. The dotted lines are the 95% confidence interval for the regression line, which can be significantly affected by outliers when PI is large.



Graph demonstrating a significant correlation between the ICP and the PI with a correlation coefficient of 0.938~(p<0.0001) and a correlation formula of: ICP = $10.927 \times PI-1.284$. The dotted lines are the 95% confidence interval for the regression line, which can be significantly affected by outliers when PI is large.



Admission TCD and TBI

Admission FVd < 25 cm/s and PI > 1.3 is associated with a poor outcome.

Trabold et al.

Admission velocities, FVm< 30 cm/s, were related to GCS, and correctly predicted early outcome.

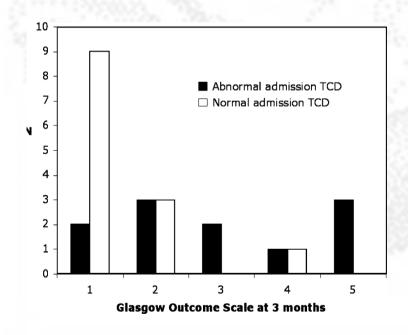
Chan KH et al.



Admission TCD and TBI

Transcranial Doppler ultrasound goal-directed therapy for the early management Catherine Ract Sophie Le Moigno of severe traumatic brain injury Nicolas Bruder Bernard Vigué

Intensive Care Med (2007) 33:645-651



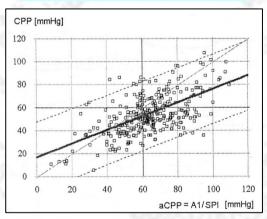
3-month GOS was significantly poorer in group 1 than in group 2.



TCD, CPP and ICP

Aaslid et al

$$eCPP = AP1 \times \frac{FVm}{FV1} + 15$$

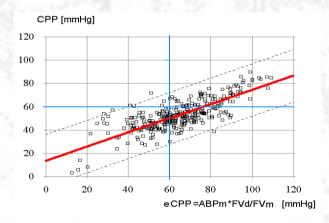


≤ 27 mmHg **⇒** 95%

≤ 10 mmHg **⇒** 52%

Czosnyka et al.

$$nCPP = MAP \times \frac{FVd}{FVm} + 14$$



≤ 21 mmHg ⇒ 95% delle stime

≤ 10 mmHg → 81% delle stime

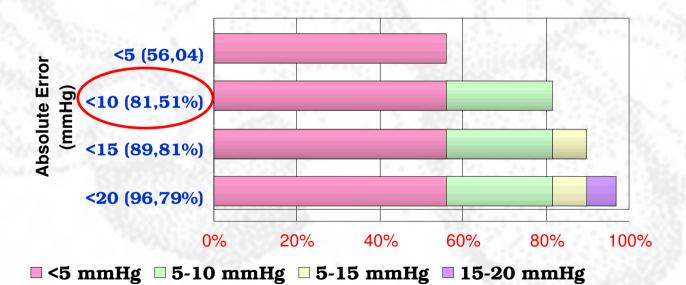


Influence of CVA on the non-invasive estimation of CPP through TCD.

DePeri E, Rasulo F et al. Intensive Care Med (Sup.1) Sept 2005:103,

No. 389. 18th ESICM, Amsterdam

- ✓ 21 patients
- √ <mark>530 samples</mark>





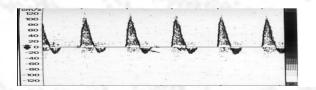
Monitoring of Increased Intracranial Pressure Resulting From Cerebral Edema With Transcranial Doppler Sonography in Patients With Middle Cerebral Artery Infarction TALIP ASIL et J Ultrasound Med 22:1049–1053, 2003

- ICP was higher on the third day than at admission in these patients.
- Increased ICP on TCD correlated with midline shifts on CT scans.
- Early outcomes of pts who had increased ICP on TCD were poorer.



TCD in Decompressive Craniectomy

Cerebral hemodynamic changes gauged by transcranial Doppler ultrasonography in patients with post-traumatic brain swelling treated by surgical decompression Edson Bor-Seng-Shu et al. J Neurosurg 104:93-100, 2006







TCD in Decompressive Craniectomy

➤ Male 44 yr, slurred speach, sudden hemiparesis

➤First TCD in ER =

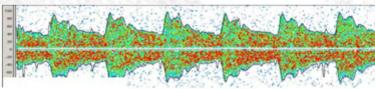
≻Angiography



- ► left MCA Ischemic Stroke
- **≻**Thrombolysis



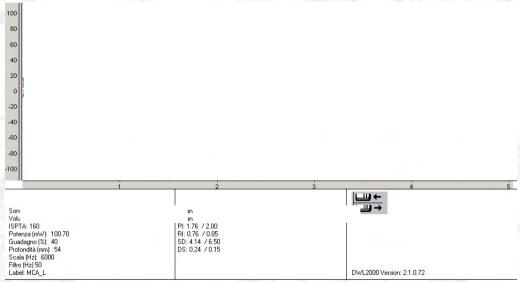
2° TCD





TCD in Decompressive Craniectomy

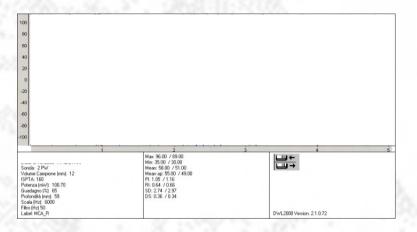




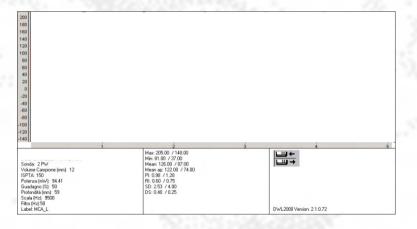


TCD in Decompressive Craniectomy



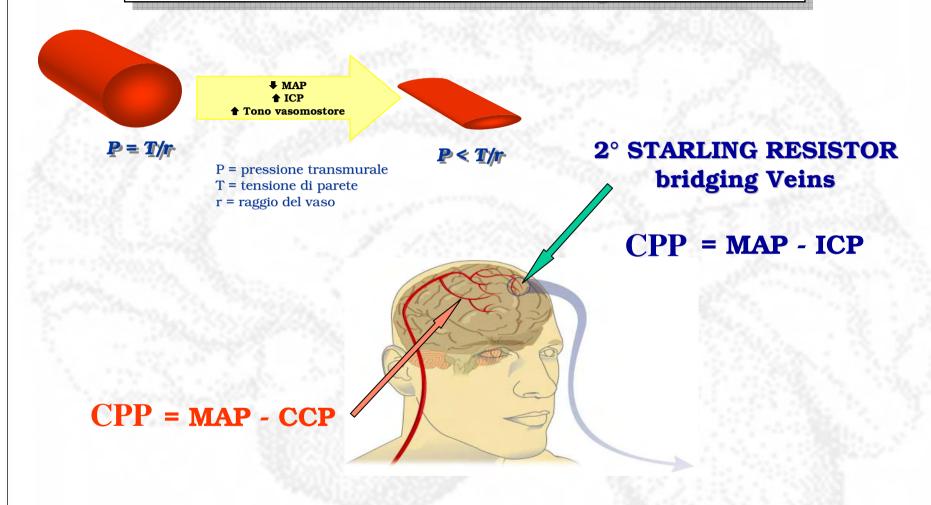








TCD & Critical Closing Pressure



1° STARLING RESISTOR Arteriolar resistance vessels



TCD and CCP

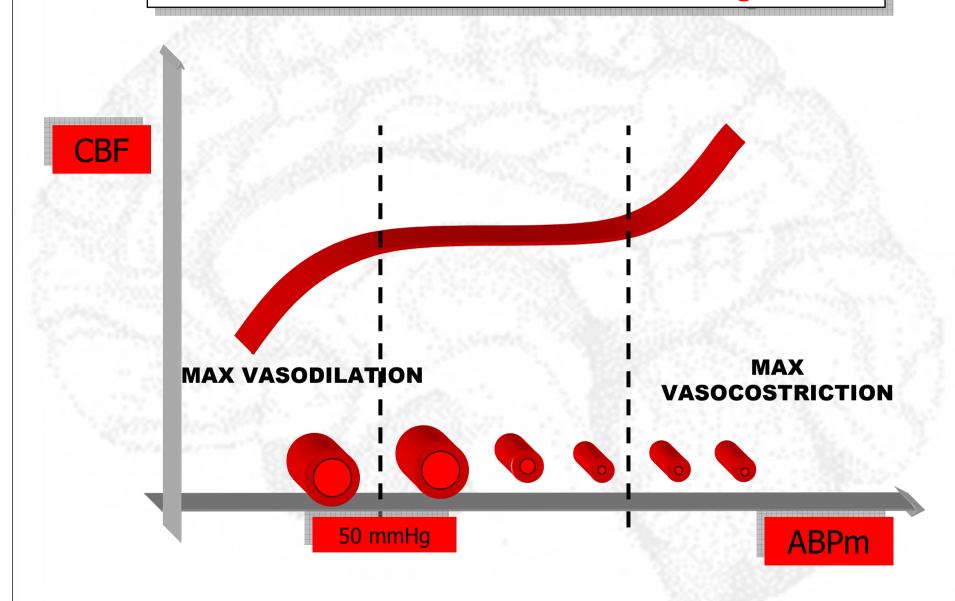




Weyland A. et all

Cerebrovascular tone rather than intracranialpressure determines the effective downstreampressure of the cerebralcirculation in the absence of intracranial hypertension J Neurosurg Anesth 2000;12:210-16







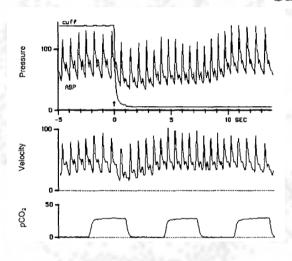
Cerebral Autoregulation Dynamics in Humans



Rune Aaslid, PhD, Karl-Fredrik Lindegaard, MD, Wilhelm Sorteberg, MD, and Helge Nornes, MD

Stroke Vol 20, No 1, January 1989





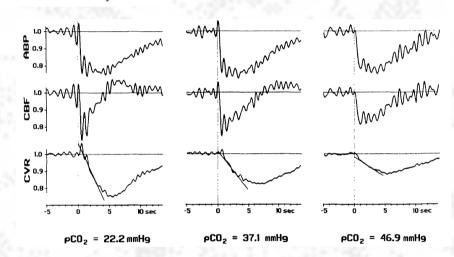


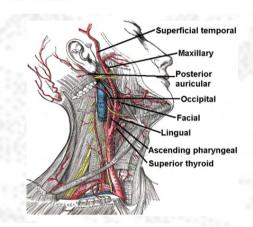
TABLE 1. Parameters Measured in Determining Cerebral Autoregulation Dynamics in Humans

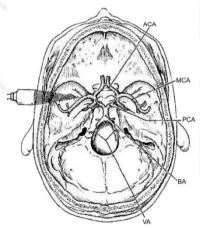
		Paco ₂ state	
Parameter	Hypocapnia (n=10) (22.2 mm Hg)	Normocapnia (n=10) (37.1 mm Hg)	Hypercapnia (n=6) (46.9 mm Hg)
Pco ₂ (mm Hg)	22.2±0.6	37.1±0.8	46.9±0.5
Control V _{MCA} (cm/sec)	46.1±3.1	67.4±5.9	89.3±3.4
Control ABP (mm Hg)	84.5±4.1	81.6±4.2	88.2±7.2
Δ ABP (%)	21.9±1.0	24.1±1.1	21.5±2.2
RoR (sec-1)	0.38±0.04	0.20±0.30	0.11 ± 0.02
Δ Power (%)	-0.4 ± 1.0	-2.55 ± 1.2	

A Bedside Test for Cerebral Autoregulation Using Transcranial Doppler Ultrasound

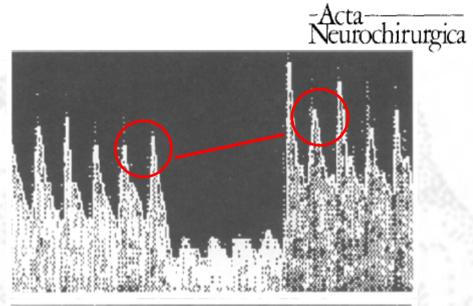
Acta Neurochir (Wien) (1991) 108: 7-14

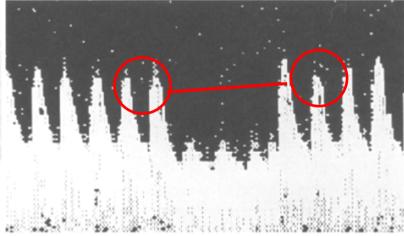
C. A. Giller





Giller CA (1991) Acta Neurochir (Wien) 108:7–14





However, clinical tests do not provide continuous assessment of CVA, which can be performed through...





Continuous Assessment of Autoregulation CORRELATION COEFFICIENTS

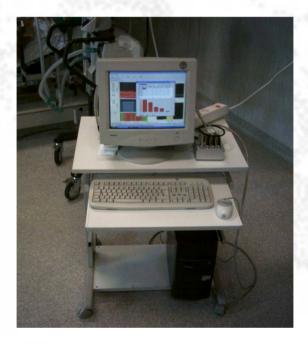
Monitoring of Cerebral Autoregulation in Head-Injured Patients.

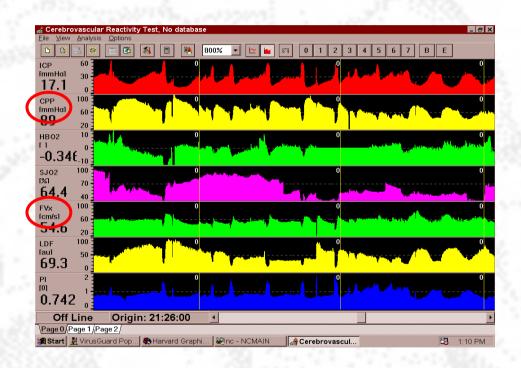
M Czosnyka, et al., Stroke, 2000

- ✓ (PRx) <u>ABP/ICP</u>
- √ (Sx) FVs/CPP
- ✓ (Mx) <u>FVm/CPP</u>











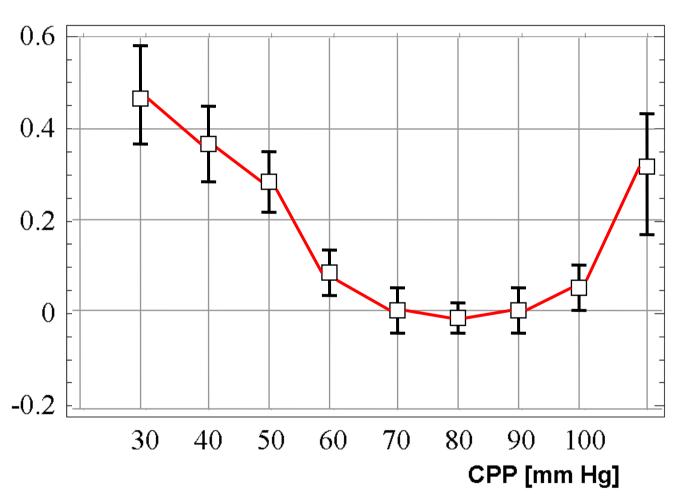
FVm/CPP



Autoregulation and Cerebral Perfusion Pressure

Czosnyka M.,et al. J Neurosurg 2001

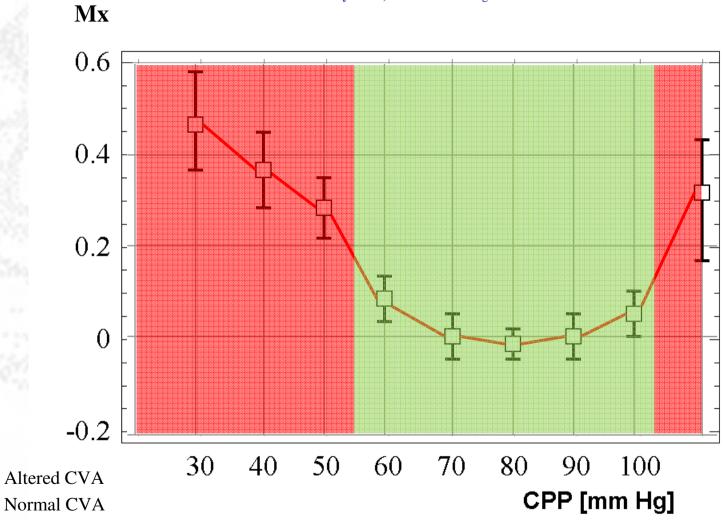






Autoregulation and Cerebral Perfusion Pressure

Czosnyka M.,et al. J Neurosurg 2001





Continuous Assessment of Cerebral Autoregulation in Subarachnoid Hemorrhage

Soehle M et al., *Anesth Analg 2004;98:1133-9*

Group	n	MABP (mm Hg)	Mean MCA FV (cm/s)	Mx
Baseline	15	93 ± 14	76 ± 20	0.21 ± 0.24
Vasospasm	15	100 ± 13	148 ± 19	0.46 ± 0.32
P value (Student's t-test)		0.007	< 0.001	0.021

Table 3. Summary of Mx and Sx Values Obtained in Different Pathologies

Author	Year	Reference	Pathology	n	Mx	Sx	n	Mx	Sx
				Bas	seline				
Piechnik	1999	19	Volunteers	14	0.21 ± 0.16	-0.07 ± 0.15			
Schmidt	2003	36	Volunteers	44	0.18 ± 0.19				
Temporal compar	rison								
				Int	racranial norm	otension	Intr	acranial hyp	ertension
Czosnyka	1999	37	Head injury	8	0.07 ± 0.31		8	0.50 ± 0.29	
				Preserved autoregulation			Imp	paired autore	gulation
Lang	2003	33	Head injury		0.15 ± 0.28		21	0.43 ± 0.23	3
				Bas	seline		Vas	ospasm	
Our study	2004		SAH	15	0.21 ± 0.24	0.05 ± 0.21	15	0.46 ± 0.32	0.22 ± 0.26
Spatial compariso	n								
				Co	ntralateral side	of stenosis	Side	e of stenosis	
Reinhard	2003	35	CAOD	56	0.30 ± 0.17		56	0.51 ± 0.18	3
				Contralateral side of vasospasm		Sid	e of vasospa	sm	
Our study	2004		SAH	15	0.34 ± 0.29	0.16 ± 0.25	15	0.44 ± 0.27	7 0.24 ± 0.23

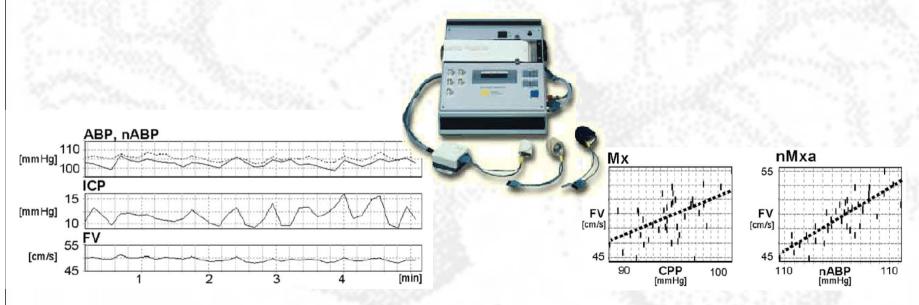
"Mx showed impaired autoregulation in all the patients with Vasospasm and correlated with the side affected"



Noninvasive Evaluation of Dynamic Cerebrovascular Autoregulation Using Finapres Plethysmograph and Transcranial Doppler

Lavinio et. al. Stroke Feb. 2007;38:402-4

Mx can be estimated noninvasively with the use of a finger plethysmograph arterial blood pressure measurement instead of an invasive cerebral perfusion pressure measurement.



Proposed as a practical tool to assess CVA in patients who do not require invasive monitoring.



Cerebral Circulatory Arrest in Brain Death





TCD and Cerebral Circulatory Arrest

Assessment: Transcranial Doppler ultrasonography

Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology

M. A. Sloan, et al: NEUROLOGY 2004;62:1468-1481

INDICATION	SENSITIVITY (%)	SPECIFICITY (%)	REFERENCE STANDARD
Cerebral Circulatory Arrest and Brain Death	91-100	97-100	Conventional angiography, EEG, clinical outcome

Recommendation: Type A, Class II evidence



TCD and Cerebral Circulatory Arrest

Transcranial Doppler Ultrasonography to confirm Brain Death: a metanalysis

Monteiro LM et al. Intensiva Care Med 2006;32:1937-44

Table 2 Meta-analysis of high quality studies and sensitivity analysis of all included studies. *CI* confidence interval

Primary analysis: only high-quality studies						
95% CI						
Sensitivity (%)	95	92	97			
Specificity (%)	99	97	100			
Sensitivity analysis: all studies. Velthoven et al. [24]: cerebral angiography and clinical criteria as reference test 95% CI						
Sensitivity (%)	89	86	91			
Specificity (%)	99	99	100			
Sensitivity analysis: all studies. Velthoven et al. [24]: EEG and clinical criteria as reference test 95% CI						
Sensitivity (%)	89	86	91			
Specificity (%)	99	98	100			



TCD in the ICU

- > VASOSPASMO **CEREBRALE**
- > IPERTENSIONE **INTRACRANICA**
- > ARRESTO DI CIRCOLO **CEREBRALE**
- > AUTOREGOLAZIONE **CEREBROVASCOLARE**
- > STENOSIS

- > MENINGITIS
- > ENICHPRACIATIS **SUBARACNOIDEA**
- > ECLAMPSIA
- > TRAUMA CRANICO
- > LIVER FAILURE
- > STROKE
- > SICKLE CELL DESEASE
- > MORTE CEREBRALE > DIABETES
- > SEPSIS



TCD and MENINGITIS

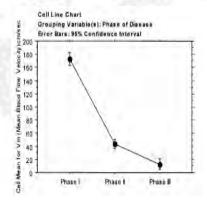
Three phases of cerebral arteriopathy in meningitis: vasospasm and vasodilation followed by organic stenosis

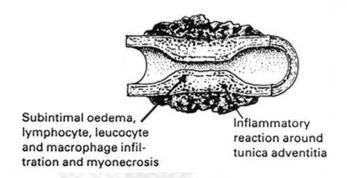
Yamashima T et al. Neurosugery 1985 Apr; 16:546-53

Vasodilation – (vasoparalysis) due to decreased contractile energy in association with myonecrosis.

Vasospasm – (stimulus phenomenon) produced by the surrounding purulent material "narrowing of the the vessels caused by the inflamatory process" (arteritis)

Stenosis – (repair process) due to the organization of subendothelial edema with resultant intimal thickening.







TCD and MENINGITIS

Utility of transcranial Doppler ultrasonography in the diagnosis and follow-up of tuberculous meningitis-related vasculopathy Türker Kiliç Child's Nerv Syst (2002) 18:142–146

TCD allowed to distinguish three phases of tuberculous meningitis related vasculopathy.

Phase I vasculopathy - increased Vm and normal to moderately decreased PI. In this phase reversible ischemic deficits are seen clinically and radiologically.

Phase II - decreased Vm. At this stage patients reveal radiological and clinical signs related to proximally evolving vasculopathy in the basal main arteries.

Phase III - almost absent CBF in one or more basal arteries and, accordingly, by associated brain tissue infarction and permanent severe neurological deficit or fatal outcome.



TCD and MENINGITIS

Relationship between short-term outcome and occurence of cerebral artery stenosis in survivors of bacterial meningitis

Martin M. et al. J Neurol 1998, 245:87-92

Patients with stenosis were associated with high Fvm and a poor short-term outcome

Multimodal cerebral monitoring and decompressive surgery for the treatment of severe bacterial meningitis with increased intracranial pressure

B. Baussart et al. Acta Anaesthesiol Scand 2006; 50: 762-765

ICP and TCD monitoring helped to decide when to perform early and late surgery (hemicraniectomy and shunting)



TCD and ECLAMPSIA

Do maternal cerebral vascular changes assessed by transcranial Doppler antedate pre-eclampsia?

Williams K et al. Ultrasound Obstet Gynecol. 2004 Mar;23:254-6

Maternal transcranial Doppler in pre-eclampsia and eclampsia.

Williams K et al. Ultrasound Obstet Gynecol. 2003 May;21:507-13.

Pre-eclampsia CPP T CVR CBF



Eclampsia

CPP T CVR CBF





TCD and LIVER FAILURE

Transcranial Doppler Sonography in Fulminant Hepatic Failure

A. Abdo, O. López, A. Fernández, J. Santos, J. Castillo, R. Castellanos, L. González, F. Gómez, and D. Limonta

*Transplantation Proceedings, 35, 1859–1860 (2003)

"The pattern mostly found is that of cerebral hypoperfusion."

Therapy of Intracranial Hypertension in Patients With Fulminant Hepatic Failure

Murugan Raghavan, Paul E. Marik

Neurocritical Care, April 2006, Volume 4, Issue 2, pps. 179-189

"Many patients die prior to the availability of donor organs, often because of cerebral herniation"

Cerebral Hemodynamic and Metabolic Changes in Patients With Fulminant Hepatic Failure During Liver Transplantation

G. Ardizzone, A. Arrigo, F. Panaro, S. Ornis, R. Colombi, S. Distefano, T.M. Jarzembowski, and E. Cerruti *Transplantation Proceedings*, 36, 3060–3064 (2004)

"A hallmark of FHF seems to be failure of autoregulation, which is linked to uncoupling between CBF and CMRO2"



TCD and SICLE CELL DISEASE

Transcranial Doppler ultrasonography in adults with sickle cell disease

N. Valadi, MD; G.S. Silva, MD; L.S. Bowman, BSc; D. Ramsingh, BSc; P. Vicari, MD; A.C. Filho, MD; A.R. Massaro, MD; A. Kutlar, MD; F.T. Nichols, MD; and R.J. Adams, MD



Prevention and Management of Stroke in Sickle Cell Anemia

Orah S. Platt

"Children with elevated velocities (≥ 200 cm/sec) have high rate of stroke. When asymptomatic children with abnormally high flow velocities demonstrated by using screening TCD are preemptively treated with maintenance transfusion, 90% of strokes are prevented."



TCD, SEPSIS & Encephalopathy

Dysfunction of vasomotor reactivity in severe sepsis and septic shock

Christoph Terborg

Intensive Care Med (2001) 27: 1231-1234

Metabolic changes

Muscular proteolysis Hepatic disfunction Reduced AA uptake

SEPSIS ENCEPHALOPATHY (70%)

Inflammatory hypothesis

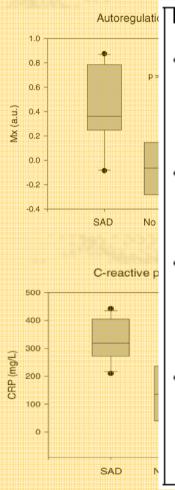
Cytochine Arachidonic ac. metabolytes





Cerebral Perfusion in Sepsis-Assosiated Delirium

David Pfister et al. Critical Care 2008, 12:R63



Key messages

- In this small group of patients, cerebral perfusion assessed with transcranial Doppler and near-infrared spectroscopy did not differ between patients with and without sepsis-associated delirium.
- We found a <u>significant association between disturbed</u> cerebrovascular autoregulation and sepsis-associated delirium.
- A significant correlation between higher values of Creactive protein and increasingly disturbed cerebrovascular autoregulation suggests a harmful effect of inflammation on cerebrovascular endothelial function.
- The significant associations between sepsis-associated delirium and elevated S-100β and cortisol suggest that further investigations defining the role of these markers as aids in the diagnosis of sepsis-associated delirium are warranted.



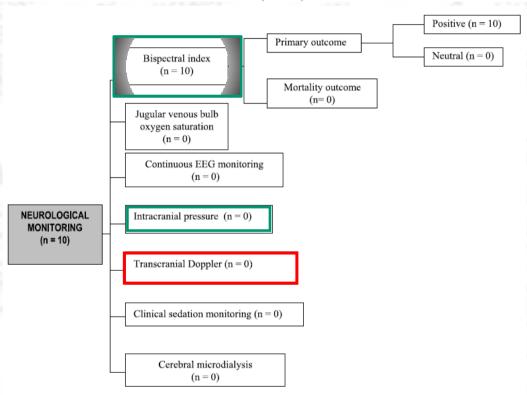


Neuromonitoring

Gustavo A. Ospina-Tascón Ricardo L. Cordioli Jean-Louis Vincent

What type of monitoring has been shown to improve outcomes in acutely ill patients?

Intensive Care Med (2008) 34:800-820



There are no RCTs assessing the effects of jugular venous bulb saturation, **transcranial Doppler**, Intracranial pressure, or cerebral microdialysis.









Monitor Interpretation

• The first step to demonstrate efficacy of monitoring is to ensure adequate training in interpretation.



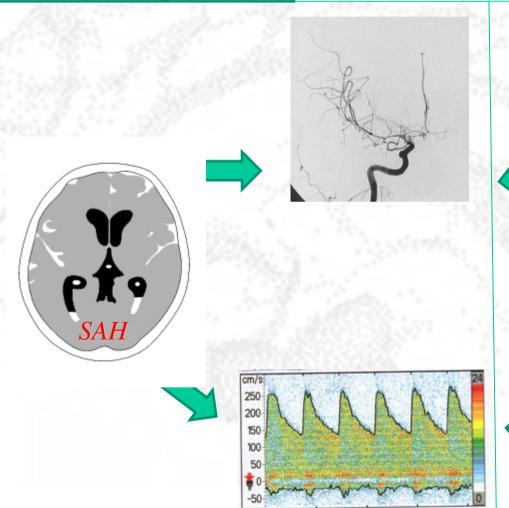
Therapeutic Intervention

- A monitor cannot affect patient outcome unless it identifies a preclinical abnormality and prompts timely and appropriate therapeutic intervention
- This abnormality must be an integral determinant of patient outcome.
- For example, intracranial hypertension cannot be reliably detected by clinical exam and has been associated with unfavorable neurological outcome.



Monitor Interpretation

Therapeutic Intervention

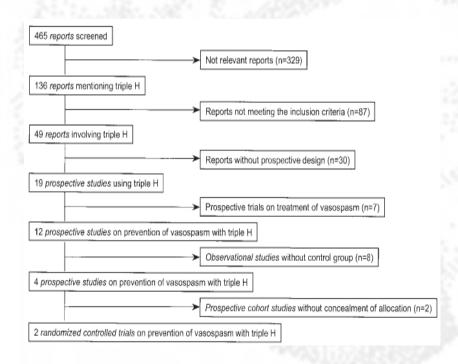




- **H**yperVOLEMIA
- **H**yperTENSION
- HemoDILUTION

Systematic review of the prevention of delayed ischemic neurological deficits with hypertension, hypervolemia, and hemodilution therapy following subarachnoid hemorrhage

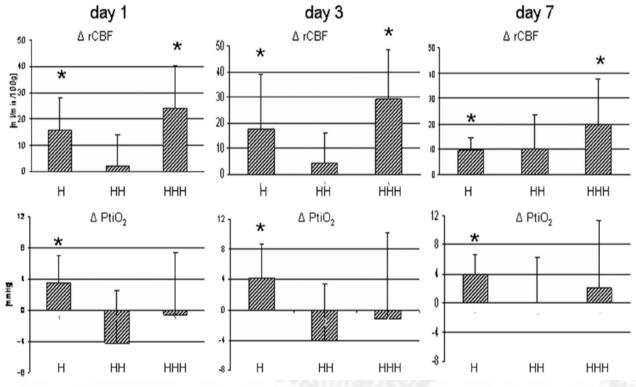
MIRIAM M. TREGGIARI J Neurosurg 98:978-984, 2003



there is insufficient "EBM" data to recommend the use of 3-H strategy as a prophylactic treatment after SAH.



Effects of hypervolemia and hypertension on regional cerebral blood flow, intracranial pressure, and brain tissue oxygenation after subarachnoid hemorrhage Elke Muench et al., Crit Care Med 2007 Vol. 35, No. 8



Vasopressor-induced elevation of MAP causes a significant increase of CPP and cerebral oxygenation in SAH patients.

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



Central Hypervolemia with Hemodilution Impairs Dynamic Cerebral Autoregulation

Yojiro Ogawa et al. Neurosurgical Anesthesiology 2007;105:1389-96

Frequent changes in the perioperative central blood volume could affect cerebral autoregulation through alterations in sympathetic activity, cardiac output, blood viscosity, and cerebral vasomotor tone.

Although steady-state CBF velocity changes under both central hypervolemia and hypovolemia, only hypervolemic hemodilution impairs dynamic cerebral autoregulation.

