

# **Course: Advanced Neurosonology – ICP mgmt. with TCD**

**ASN 42<sup>ND</sup> ANNUAL MEETING  
JANUARY 24-26, 2019**



**PUERTO RICO** ★

**Gregory Kapinos, MD, MS, FASN, FCCM**

**Neurologist, Neurointensivist,**

**Neurophysiologist, Neurosonologist, Neuroimager.**



*Save the Date*

# TCD Workshop: Management of vasospasm & tailored ICP treatment

2016

GAYLORD  
NATIONAL HARBOR

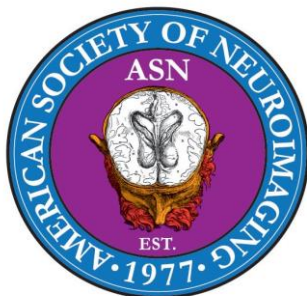
SEPTEMBER 15-18

Gaylord National Resort and Convention Center,  
National Harbor, MD

GREGORY KAPINOS, MD, MS,

Neurologist, neurointensivist,

neurosonologist, neuroimager & neurophysiologist.



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# Neurotrauma 2015

June 28 – July 1 Santa Fe, New Mexico

## PRECISION MEDICINE FOR ICP Treatment: TCD INDIVIDUALIZES TARGETING COMPLIANCE AND/OR PERFUSION AMELIORATION

Acute Surgical Cranial Trauma: “To drill or not to drill”

#10013 on 7/1/2015 at 11AM

Gregory Kapinos, MD, MS,

Ali Sadoughi, MD,

Jamie S. Ullman, MD, FACS, FAANS,

Raj K. Narayan, MD, FACS, FAANS.



Brain & Spine Specialists  
of New York<sup>SM</sup>



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AT HOFSTRA UNIVERSITY

# **TOOLS OF THE TRADE: NEUROMONITORING**

## **In: SYM3 - NEUROCRITICAL CARE & NEUROSURGICAL EMERGENCIES UPDATE**

**Gregory Kapinos, MD, MS, FASN**

**Associate Professor, Neurology,**

**SUNY Downstate School of Medicine.**

**Director, Neurocritical Care Services & Critical Care Neuromonitoring,**

**NYC H+H Kings County.**



# Stylistic influences



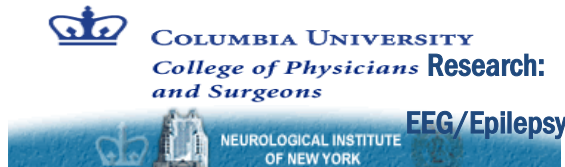
M.Med., M.Sc.

M.D.



Sub-Internships

Internships



Residency:  
Neurology



Visitorship in Neurosurgery & Neurocritical care



Sub-spec. training: Stroke



Research: Stroke MRI

NewYork-Presbyterian  
The University Hospital of Columbia and Cornell  
Clinical Fellowship: Neurocritical care



Clinical Fellowship: Bioethics & Health Law

- I am a neurologist (ABPN-BC), neurointensivist (UCNS-BC), neurosurgical intensivist (CAST/SNS-BC), neurosonologist, neuroimager (UCNS-BC), with additional competencies in advanced neuroimaging, critical care EEG, neurotraumatology, neurocoagulation, as well as in neurohospital medicine, emergency medicine, palliative care & bioethics.
- I am a board member of the editorial board of the JON and of the BOD of the ASN.
- **I have no financial ties to any commercializing company of any neuromonitoring probes or transcranial doppler, any neuroimaging company, or any pharmaceuticals.**
- I am a non-paid advisor for a processed EEG company (Ceribell), as well as a consultant and paid speaker for a cooling company (Zoll).
- I have received honoraria from BARD for devising new venous access catheters.
- I have a commitment to a stipend and royalties in an upcoming Springer book on new frontiers in quantitative EEG.



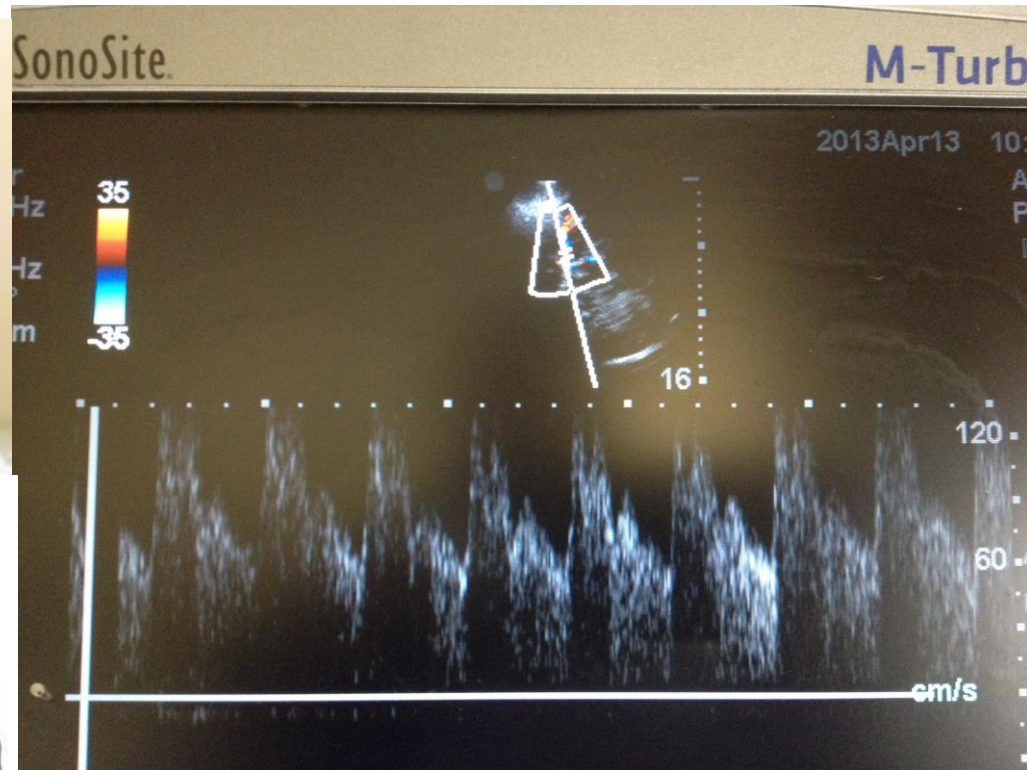


## Topic-focused Disclosures

- No financial ties to any marketed technology for trephination, TCD, neuroimaging or hemodynamic monitoring.
- No financial ties to any company marketing IVF, albumin or vasoactive drugs.
- Received <200\$ honorarium from company marketing vascular access devices.
- Trademarked “Advanced Neurological Life Support (ANLS)” but not in commerce.
- Was involved in CTP research in vasospasm with PI Pina Sanelli, who was receiving AHA, BAF and NIH funding.



# PIERCING THROUGH THE SKULL FOR ICP AND CPP WITHOUT A DRILL



Gregory Kapinos, MD, MS.



# Key messages



- No, TCDs are not ready to replace direct ICP monitors, however...
- Compliance is more important than the ICP value
  - With pressure monitor:  $P2 > P1$ , MOCAIP, etc...
  - With TCDs or regular U/S: PI, ONSD, novel index
- It is judicious to tease out the primordial inadequacy for that pt:
  - high ICP (low compliance),
  - or low CPP (low perfusion),
  - or both
  - or none
- TCD do offer advantages b/o non-invasiveness for
  - Prognosticate in ED death and poor functional outcome after TBI
  - Triaging for decision on ICU and recourse to EVD/ICP monitor
  - Teasing out better if compliance or perfusion is most compromised
  - Earlier detection of imminent crisis, as c/t using ICP/CPP threshold
  - Allocate Rx specific to the primordial inadequacy (tailored individualized goal-directed Rx for cerebral edema/ICP)



## *The* NEW ENGLAND JOURNAL *of* MEDICINE

ESTABLISHED IN 1812

DECEMBER 27, 2012

VOL. 367 NO. 26

### A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury

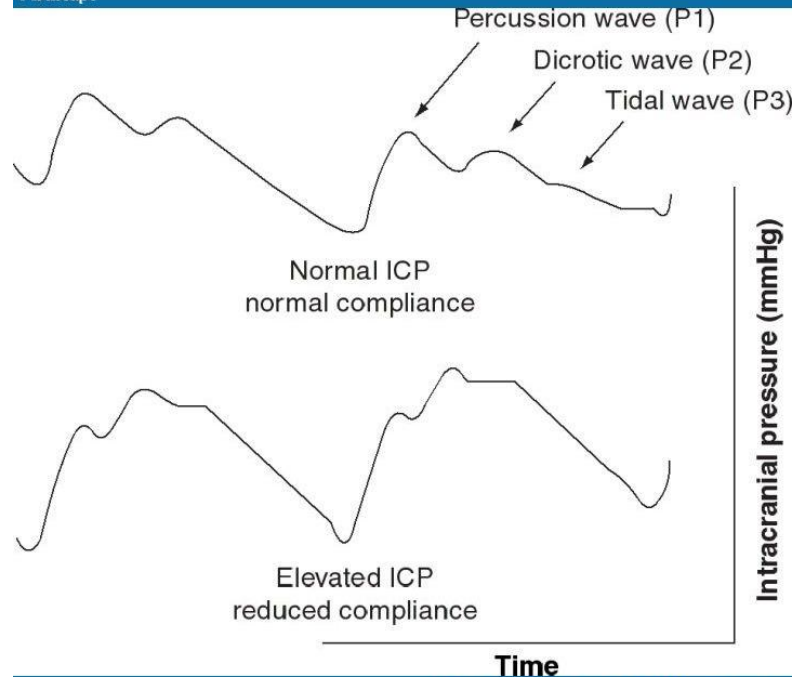
Randall M. Chesnut, M.D., Nancy Temkin, Ph.D., John E. Cantor, M.D., Sureyya Dikmen, Ph.D., Carlos Rondina, M.D.,  
Walter Videtta, M.D., Gustavo Petroni, M.D., Nina Lujan, M.D., Jill Pridgeon, M.H.A., Jason Barber, M.S.,  
Joan Machamer, M.A., Kelley Chaddock, B.A., Juanita M. Celix, M.D., Marianna Cherner, Ph.D., and Terence Hendrix, B.A.

BEST TRIP:

### Clinicoradiologic vs. EVD-guided Rx for ICP

- We got it all wrong with our EVDs in the modern world???
- Or is it just an inadequate threshold to define ICP crisis?
- Should we abandon looking at ICP alone?
- Still has an adjunctive value to vasoreactivity (PRx/Mx), cerebroximetry (PbtO2) and we need to go metabolic (LPR by CMD, MRS) and neurophysiologic (qEEG, SSEPs)?

# Introduction: ICP not the panacea



Source: Expert Rev Neurother © 2013 Expert Reviews Ltd

BARKING AT THE WRONG  
TREE:

Analogical > Digital data...

Should we Rx  
ICP mean  
or low compliance?

J Neurotrauma. 2010 Feb;27(2):317-24.

**What shapes pulse amplitude of intracranial pressure?**

[Carrera E](#), [Kim DJ](#), [Castellani G](#), [Zweifel C](#), [Czosnyka Z](#), [Kasparowicz M](#), [Smielewski P](#), [Pickard JD](#), [Czosnyka M](#).

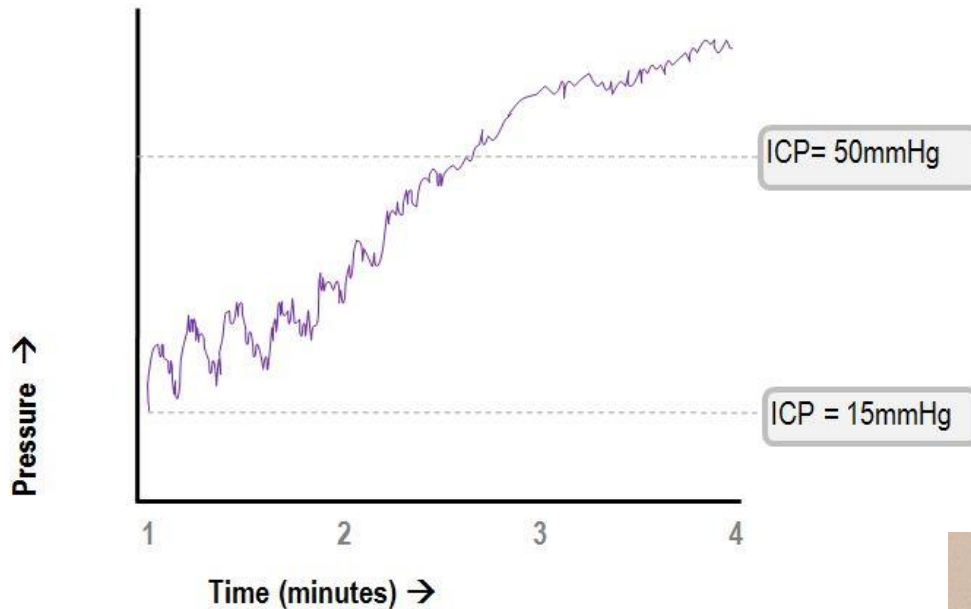
**Source**

Academic Neurosurgical Unit, Department of Clinical Neurosciences, Addenbrooke's Hospital, University of Cambridge, Cambridge

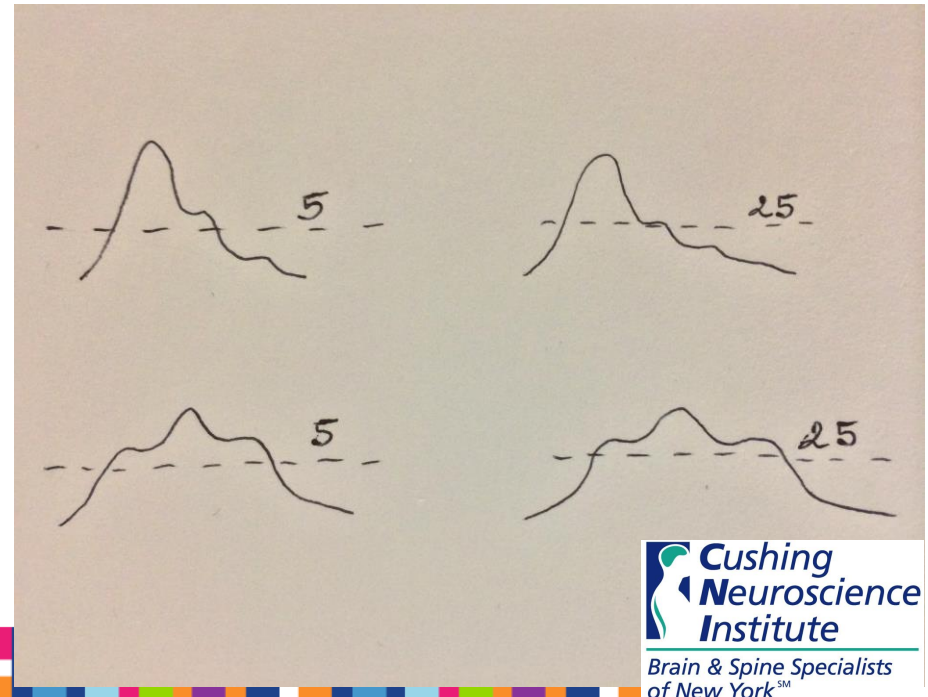


# Compliance before ICP

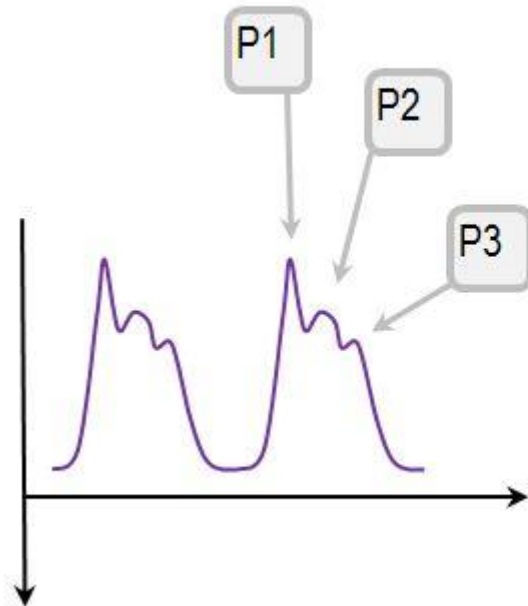
BARKING AT THE  
WRONG TREE:  
Should we Rx  
ICP mean  
or low compliance?



Split in neurosurgeons...  
Which scenario would you treat?  
Favoring ICP mean or  
waveform?



# Compliance on EVD WF

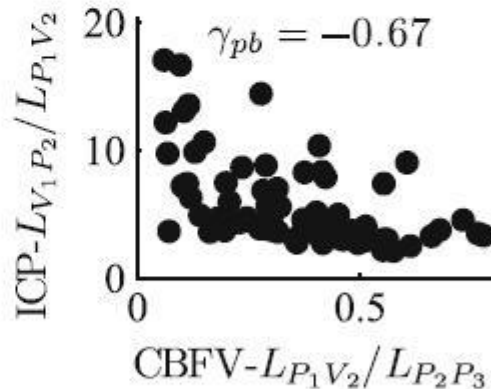
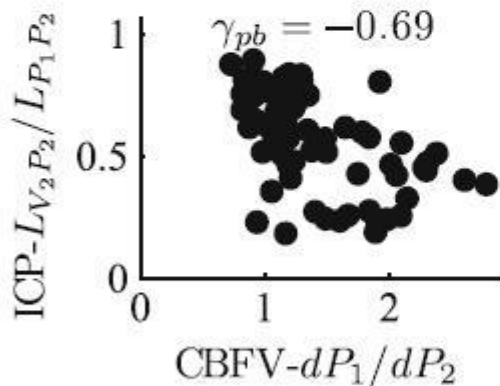
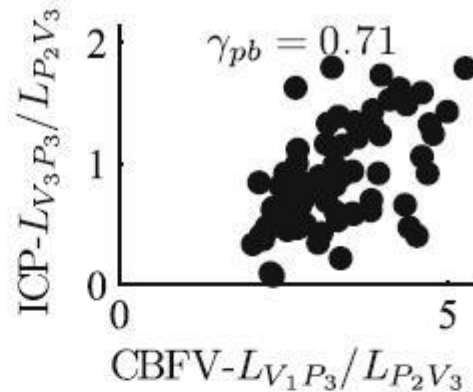
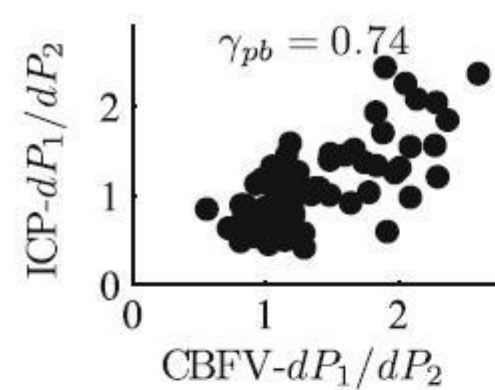


P1: arterial pulse  
P2: vaguely related to cerebral compliance  
P3: aortic valve closure, the dicrotic notch.

- Flourishing science on understanding better interaction of CSF volume onto the edematous or expanded parenchyma and the effect of each heart beat, testing vascular resistivity and parenchymal compliance:
- Pulse Amplitude, Pulsatility Index, Resistivity Index, MOCAIP...



# Perfusion status on EVD WF



MOCAIP from  
UCLA:  
Beyond  
compliance,  
VD/VC state,  
CAR...

**Complex...**  
**Let's start simple:**  
**P2>P1**

The figure visualizes four MOCAIP metrics pairs of ICP and CBFV signals, which are significantly correlated.





In France, neurosonology is embraced by neurosurgery field...



Disponible en ligne sur [www.sciencedirect.com](http://www.sciencedirect.com)



ScienceDirect

Neurochirurgie 54 (2008) 731–738

Article original

## Le doppler transcrânien en neurochirurgie

### Transcranial doppler in neurosurgery

T. Geeraerts <sup>a,□b</sup>, C. Ract <sup>a,b</sup>, J. Duranteau <sup>a,b</sup>, B. Vigué <sup>a,b</sup>

<sup>a</sup> Département d'anesthésie-réanimation, hôpital de Bicêtre, AP-HP, 78, rue du Général-Leclerc, 94276 Le Kremlin-Bicêtre, France

<sup>b</sup> UPRES 3540, laboratoire d'anesthésie, faculté de médecine, université Paris-Sud, 94276 Le Kremlin-Bicêtre, France

Reçu le 2 mars 2008 ; accepté le 25 août 2008

T. Geeraerts et al. / Neurochirurgie 54 (2008) 731–738

Disponible sur Internet le 9 octobre 2008

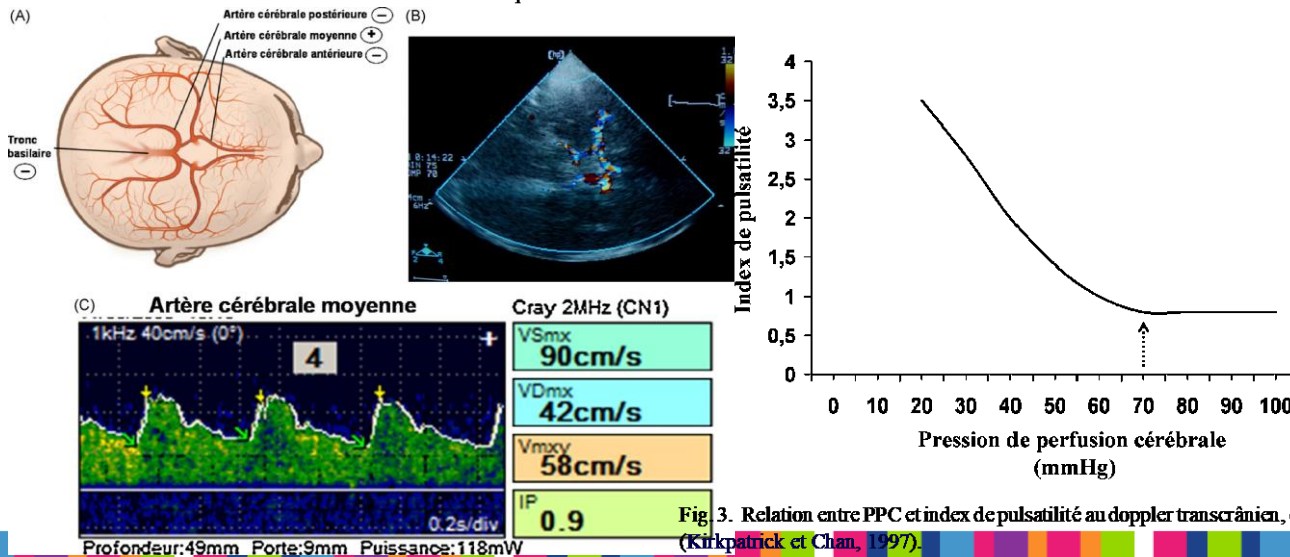


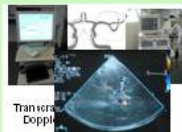
Fig 3. Relation entre PPC et index de pulsativité au doppler transcrânien, d'après (Kirkpatrick et Chan, 1997).

Relationship between cerebral perfusion pressure and transcranial doppler pulsatility index, from (Kirkpatrick et Chan, 1997).

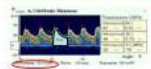
2008,  
Paris &  
Toulouse:  
Triaging  
severe TBI  
in ED:  
 $V_d < 20$   
or  $PI > 1.4$   
= Neurosurgical  
care



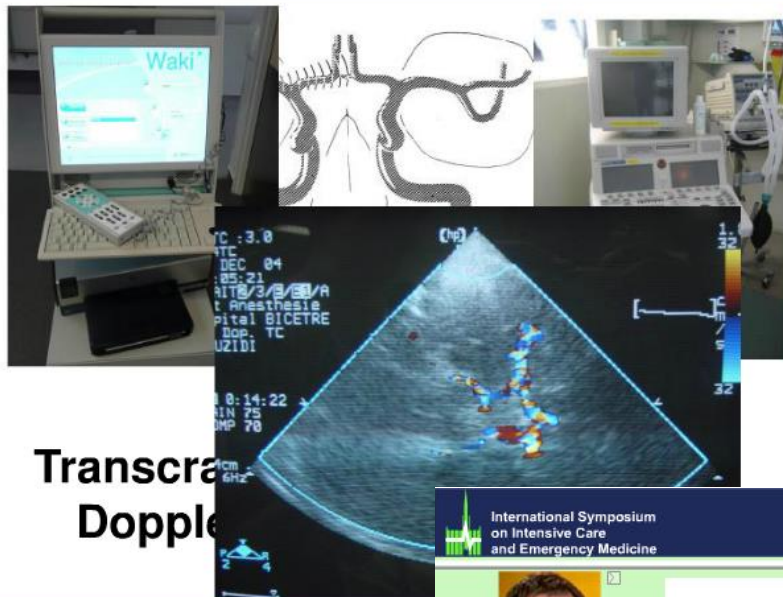
Bernard Vigué  
(Kremlin Bicêtre, France)



Slide 6



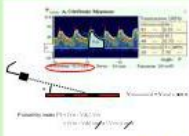
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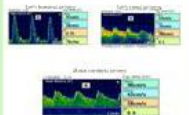
Transcranial  
Doppler



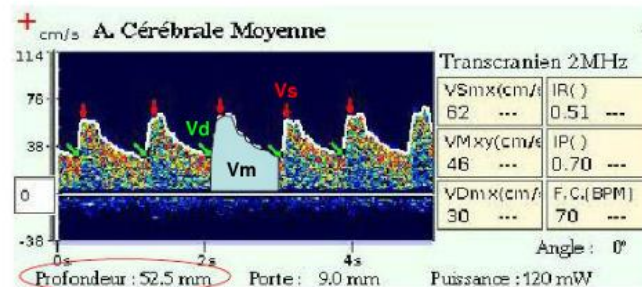
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Slide 9



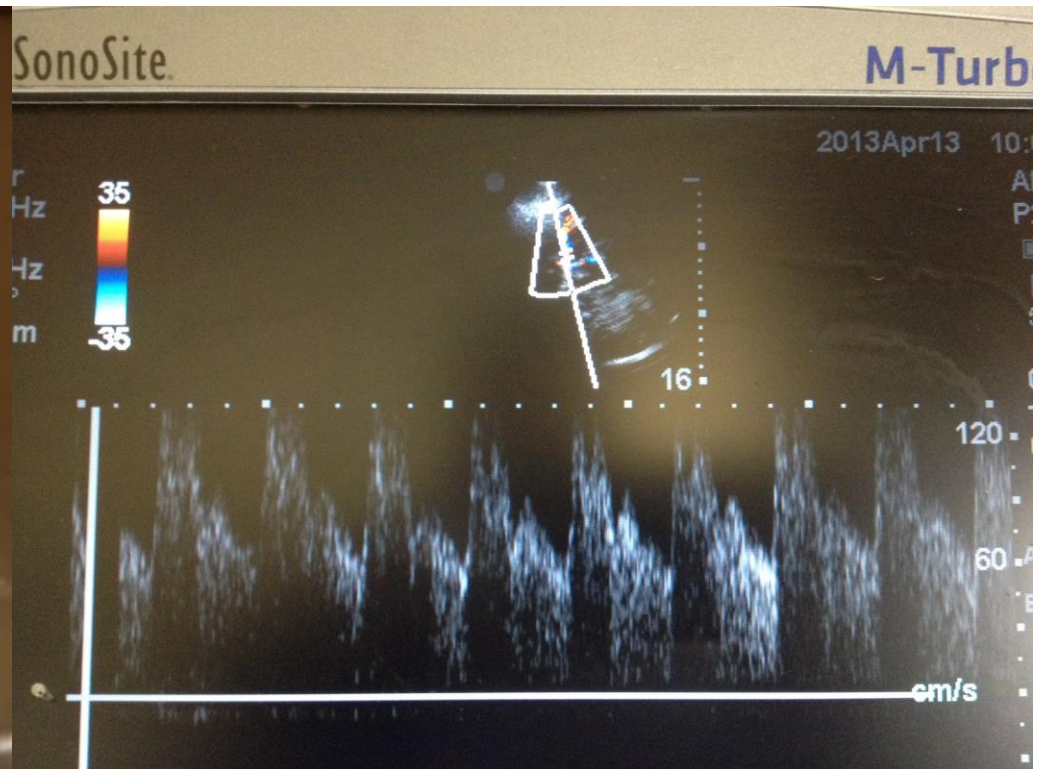
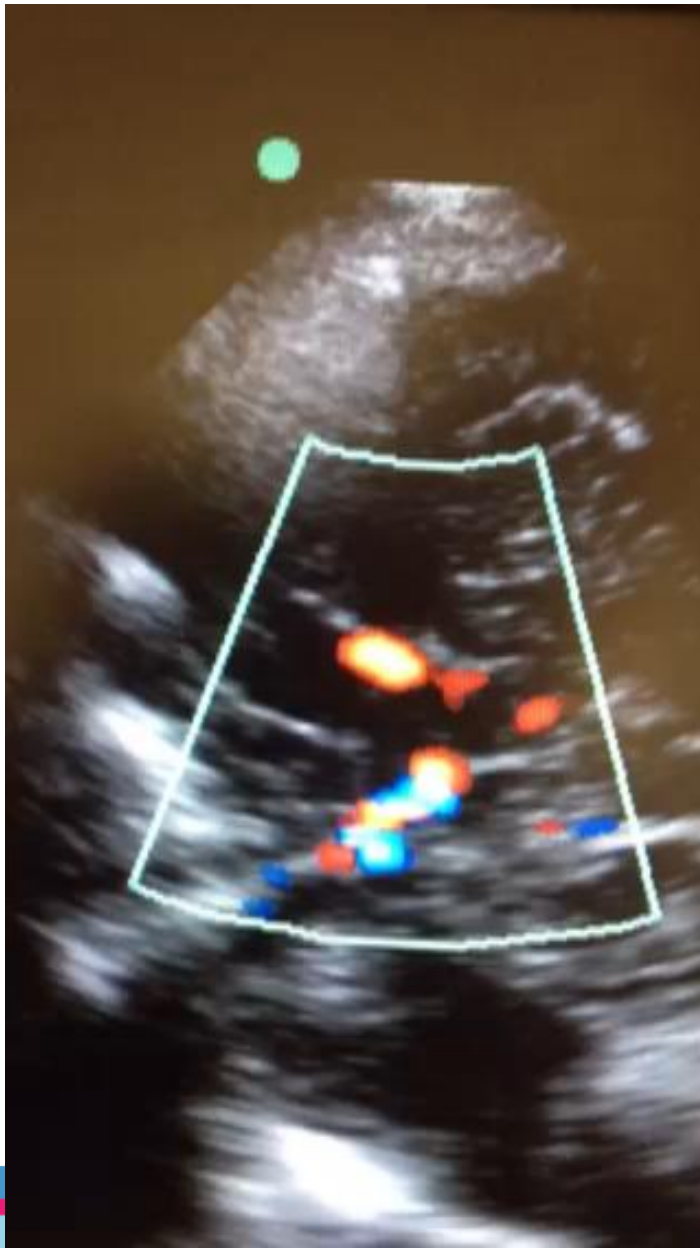
Slide 10



$V_{measured} = V_{real} \times \cos \alpha$

Pulsatility index PI =  $(V_s - V_d) / V_m$   
 $= (V_{sr} - V_{dr}) \cos \alpha / V_{mr} \times \cos \alpha$

# Neurosonology for TBI at NSLIJ



Good thing about PI:  
angle doesn't matter...  
Good thing about cosinus:  
we only underestimate...



# PI not the panacea...



PI actually not very well correlating  
with gold-standard ICP by EVD...

Neurosurgery. 2011 Jan;68(1):E289-92.

We still do not have a reliable and validated noninvasive technique that can provide an accurate quantitative measurement of intracranial pressure (ICP) that could replace invasive quantitative measurements of ICP.

[Razumovsky A, Armonda RA.](#)

Neurosurgery. 2010 Jun;66(6):1050-7.

Transcranial Doppler pulsatility index: not an accurate method to assess intracranial pressure.

[Behrens A, Lenfeldt N, Ambarki K, Malm J, Eklund A, Koskinen LO.](#)

Because the correct “gold-standard”  
is compliance, not ICP!



Vigue/Geeraerts: either PI or Vd or both  
tell you that brain physiology is bad...

vs Kapinos: they each tell you something different!

Needed distinction by **extricating PI from Vd (or EDV)**:

PI addresses a primary ICP (or actually compliance) issue, which is really what we care about... is there something taking too much space in the brain or is the brain too edematous...

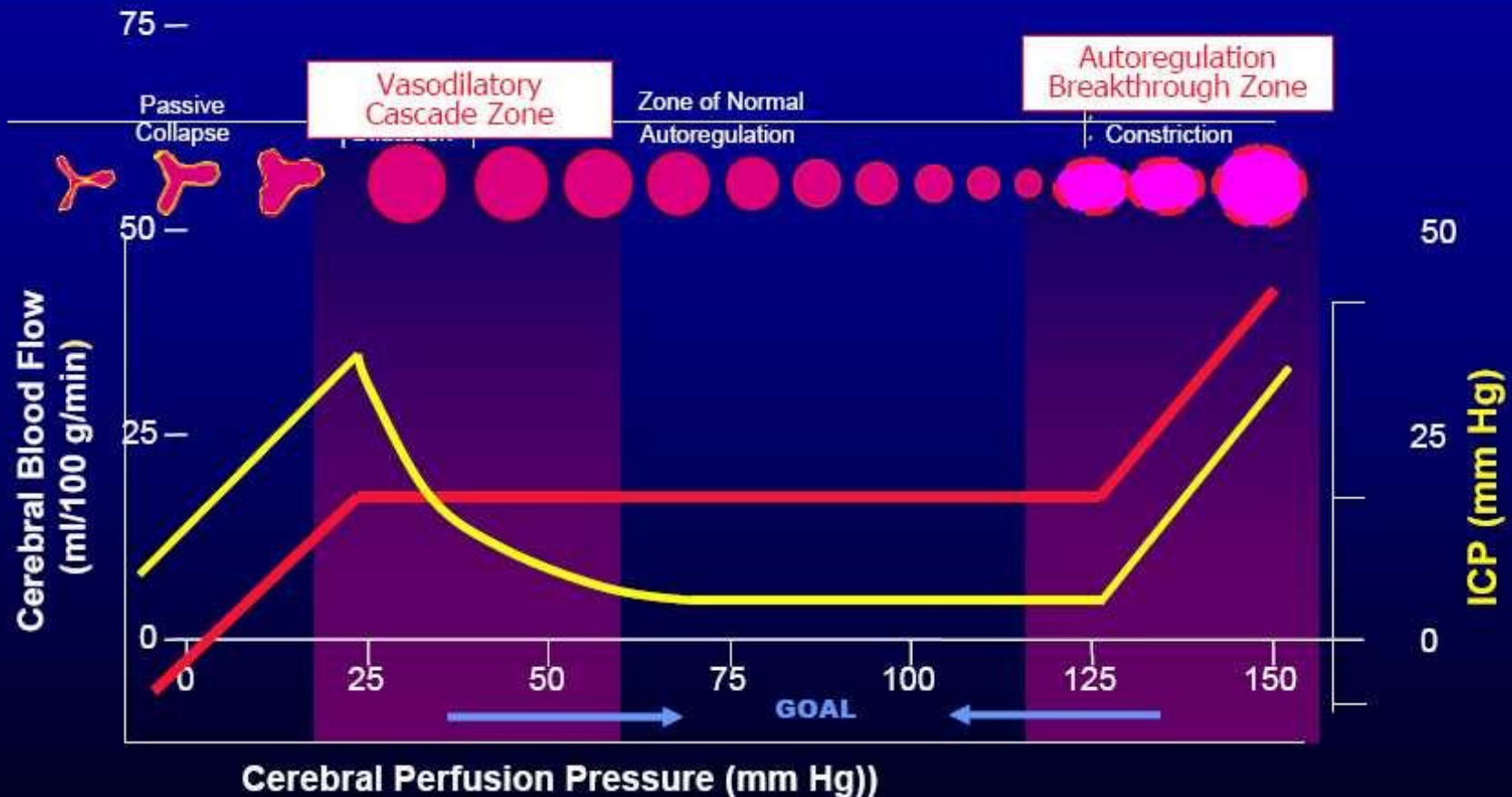
Rx? CSF diversion, tumor/blood evacuation, craniectomy or osmotherapy.

**vs.** Vd depicts a primary CPP issue with inadequately low CPP, the beginning of compromised CBF, and if it persists, VD cascade will ensue with subsequent ischemia and rise in ICP too...

Rx? the issue is the low MAP and Rx should consist of course of raising MAP, not only to reach an optimal individualized CPP goal but also because this change by itself can intrinsically lower the ICP...



# Hypertension Can Drive Elevated Intracranial Pressure ... But so can hypotension!





Crit Care Med. 1999 Oct;27(10):2086-95.

**Prevention of secondary ischemic insults after severe head injury.**

[Robertson CS](#), [Valadka AB](#), [Hannay HJ](#), [Contant CF](#), [Gopinath SP](#), [Cormio M](#), [Uzura M](#), [Grossman RG](#).

## Source

Department of Neurosurgery, Baylor College of Medicine, Houston, TX 77030, USA. [claudiar@bcm.tmc.edu](mailto:claudiar@bcm.tmc.edu)

- Now it is easy to understand why 2 schools of thoughts fight for no reason (Lund vs. Houston approaches)...
- But they are both right for a specific subset of patients...
- I propose to create an individualized Rx tailored to ICP vs. CPP being the preponderant issue...

# I'm not a PI-advocate...



## assessment of the lower cerebral autoregulatory threshold

<sup>1</sup>S. B. Lewis MBBS, <sup>2</sup>M. L. H. Wong B MED SC (HONS),

<sup>2</sup>P. E. Bannan FRACS, <sup>2</sup>I. R. Piper PHD, <sup>2</sup>P. L. Reilly MD

<sup>1</sup>Department of Neurosurgery, Sir Charles Gairdner Hospital, Verdun St, Nedlands 6009 Western Australia

<sup>2</sup>Department of Neurosurgery, Royal Adelaide Hospital, North Terrace, Adelaide, SA 5000, Australia

**Summary** Continuous transcranial Doppler ultrasonography the middle cerebral artery (TCD-MCA) has been proposed a

Yes, but its sensitivity for detecting an elevated ICP or a poor compliance is also its weakness...

PI is the best!  
It goes up in the VD cascade too...

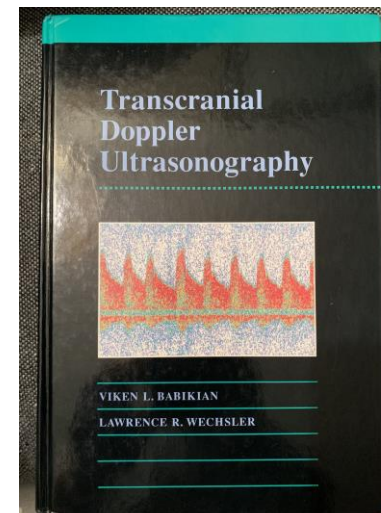
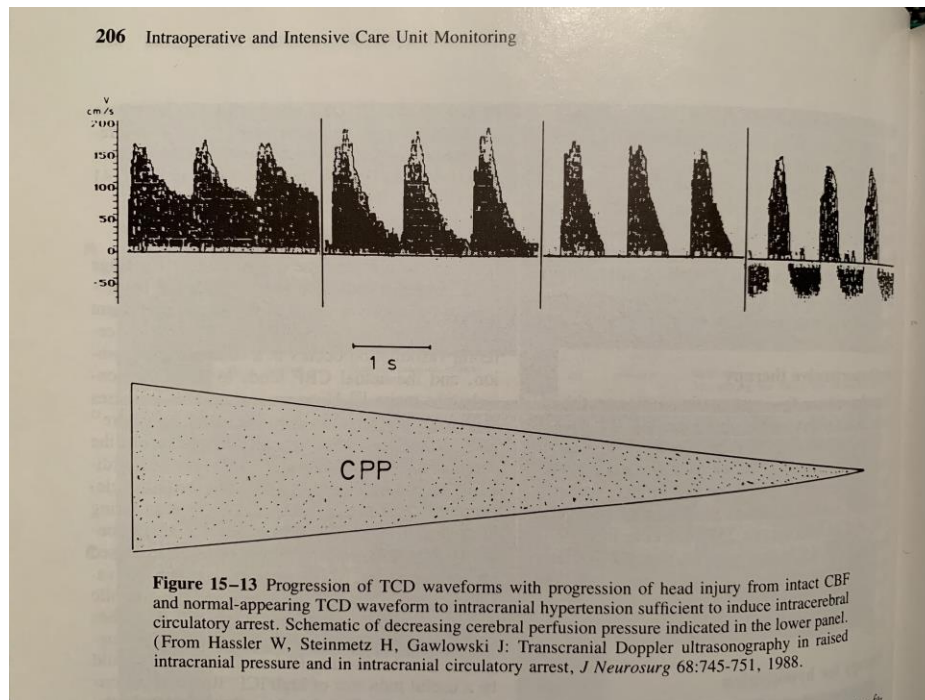
This study conformed to the guidelines established by the National Health and Medical research Council and was approved by the Animal Ethics Committees of the Royal Adelaide Hospital, University of Adelaide and Institute of Medical and Veterinary Science.

## RESULTS

There was neither hypoxia nor sustained hyper- or hypocarbia over the experimental period for any animal. As CPP decreased, CBF was initially maintained until a sharp breakpoint occurred at a mean value of  $50 \pm 1.5$  mmHg CPP. The grouped CBF data and the mean breakpoint are presented in Figure 2. The lower limit of autoregulation indicated by the CBF breakpoint ranged from 41 to 59 mmHg CPP. Similarly, as CPP fell, systolic Doppler flow velocity was preserved until a mean breakpoint of  $50 \pm 1.8$  mmHg CPP (range 38–60 mmHg CPP), shown in Figure 3. In contrast, the mean dias-

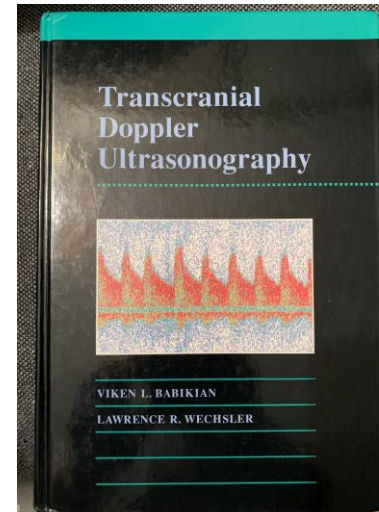
# CPPe in landmark discoveries

- CPPe correlates to  $MFV/(PSV-EDV)$
- $PI = (PSV-EDV)/MFV$
- Thus CPPe correlates to  $1/PI$



# Estimates of ICP and CPP by Aaslid et al. and Klingelhofer et al.

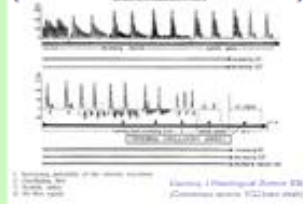
- ICP/MAP correlates to RI/MFV
- Thus  $ICPe = (MAP \times RI) / MFV$
- And  $CPPe = MFV / FV^1 \times MAP^1$



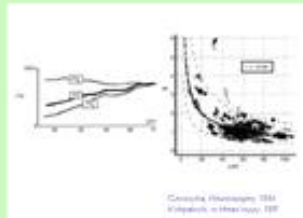




**Bernard Vigué**  
(Kremlin-Bicêtre, France)



Slide 11



Slide 12

Major role of secondary ischemic injuries  
in the very first hours after trauma

Low CPP is associated 30% of cases 30d

Recovery of consciousness after 30d

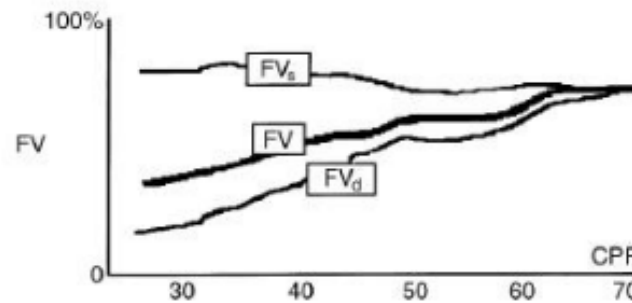
Recovery of consciousness after 30d

Recovery of consciousness after 30d

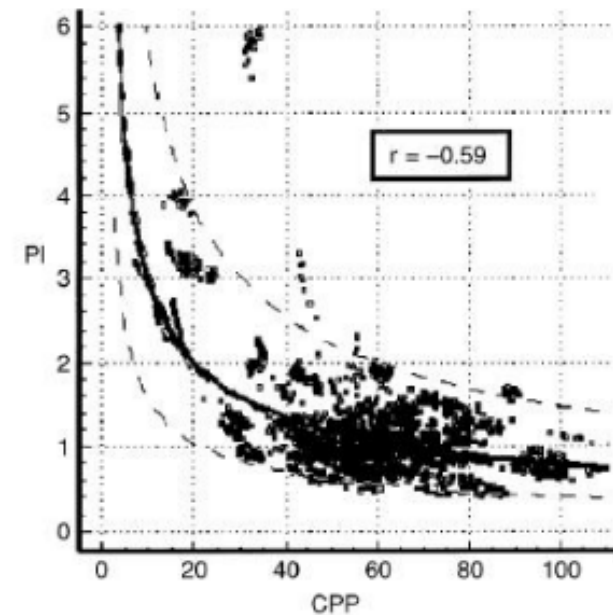
Recovery of consciousness after 30d

Recovery of consciousness after 30d

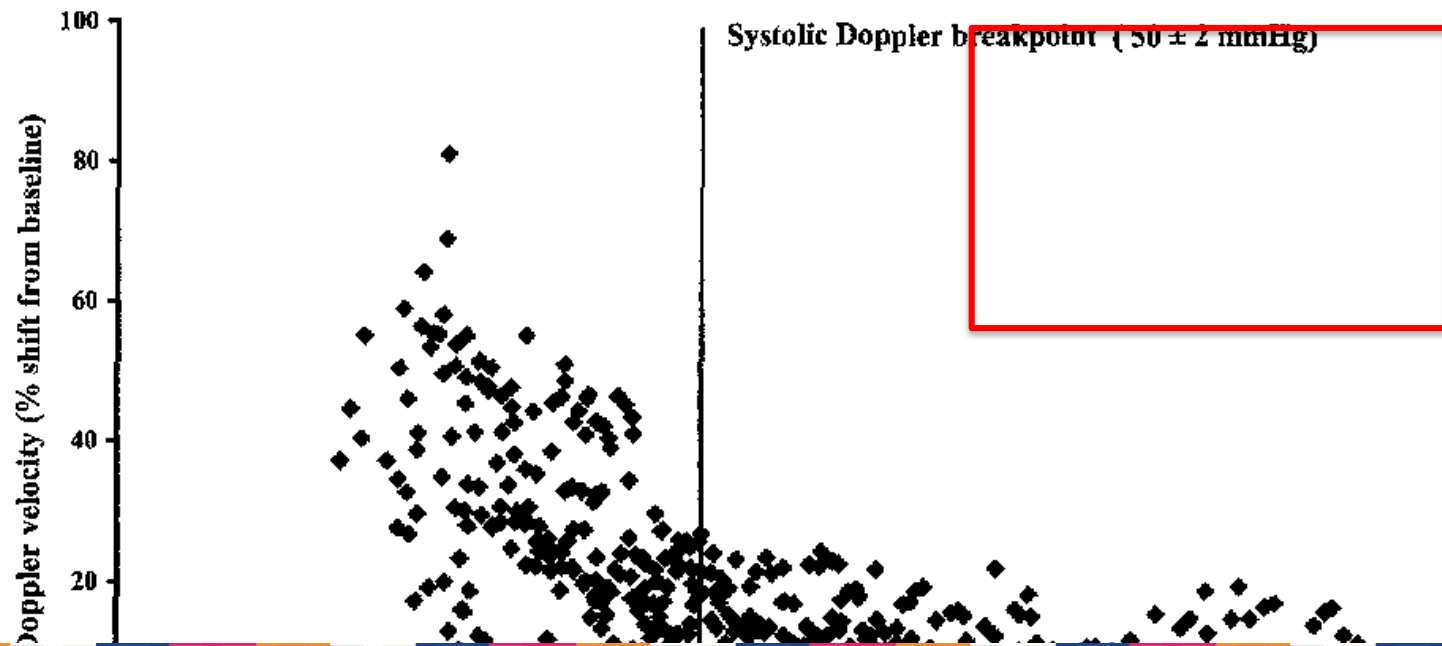
Recovery of consciousness after 30d



Peaked my interest  
in FVd (EDV) !



Czosnyka, Neurosurgery 1994  
Kirkpatrick, in Head injury 1997



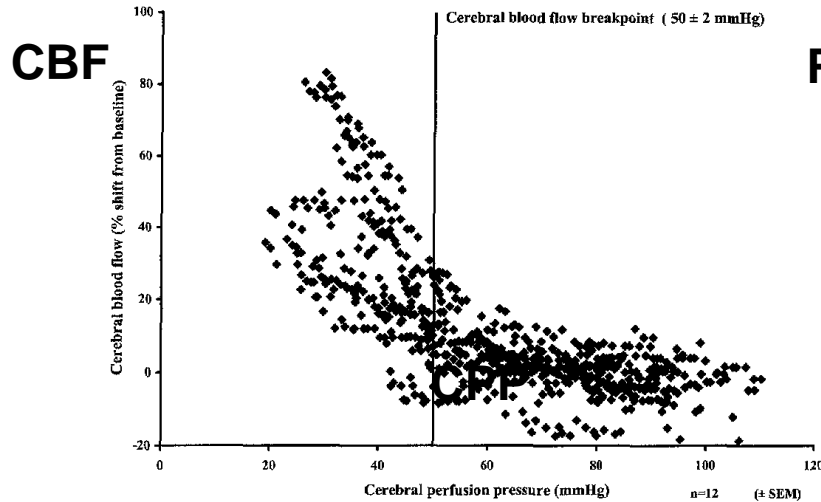


Fig. 2 Scatter plot for grouped cerebral blood flow data.

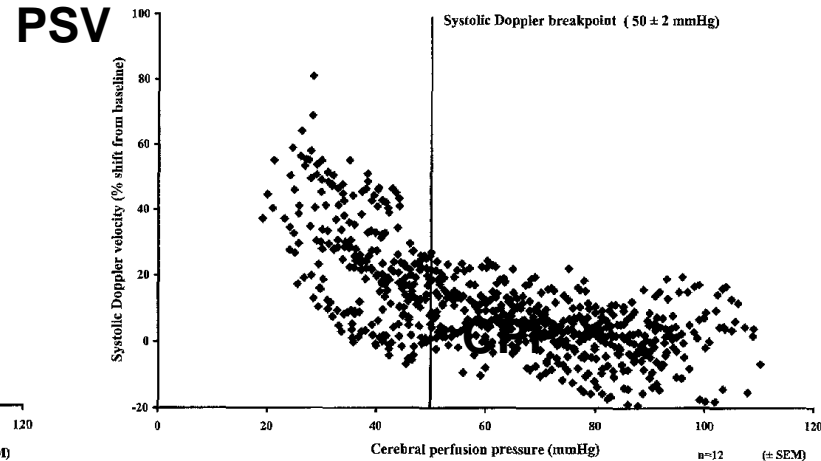
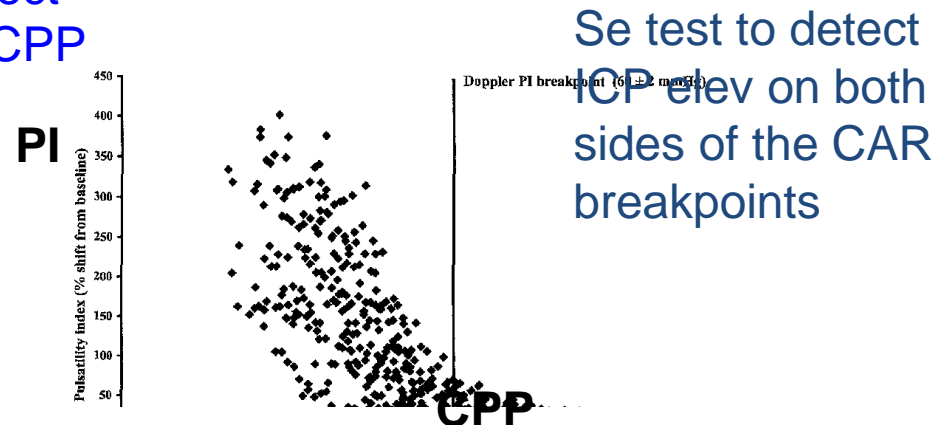
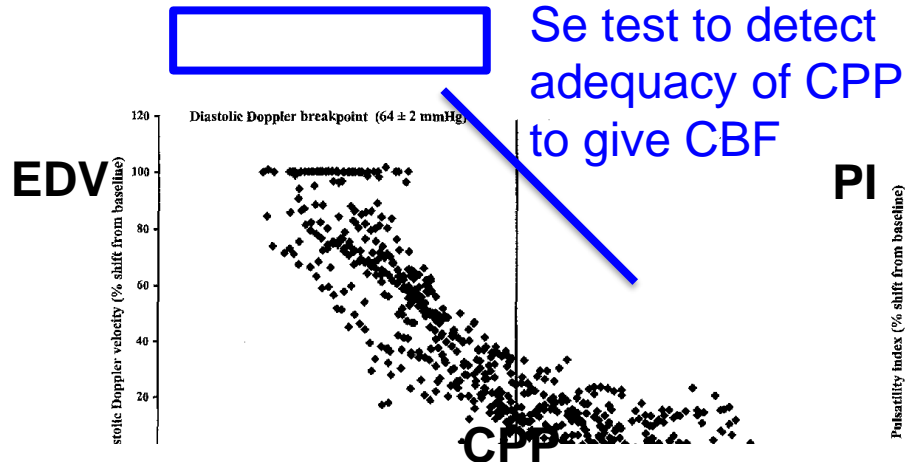


Fig. 3 Scatter plot for grouped systolic Doppler flow data.



Only once in the midst of the VD cascade...

# Rx'ic implications: Opposite goals for MAP

Lund vs. Houston:  
ICP>>>CPP vs. CPP>>>ICP  
Or a little bit of both...

The issue of  
Ischemia vs. Edema  
deserves  
to tease out  
PI from EDV...







**Bernard Vigué**  
(Kremlin Bicêtre, France)



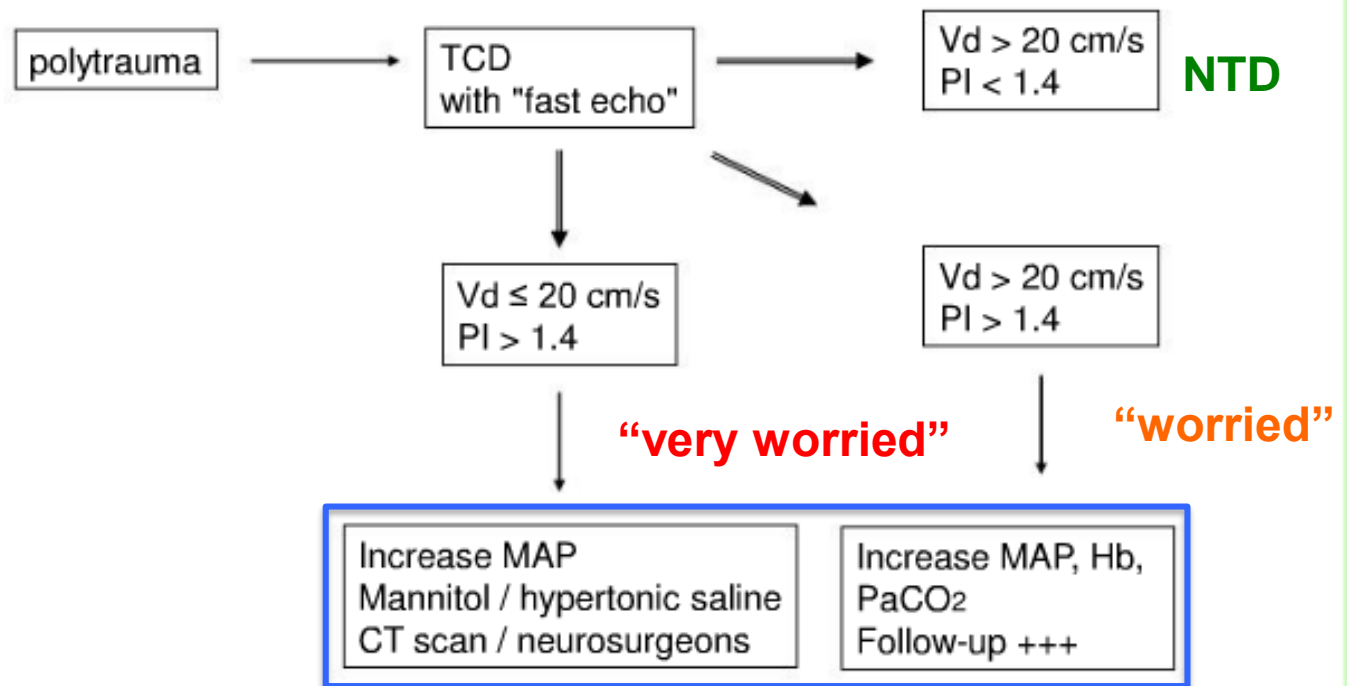
Slide 25



Slide 26

Conclusion

We propose :



# Vigue & Ract

Catherine Ract  
Sophie Le Moigno  
Nicolas Bruder  
Bernard Vigué

## Transcranial Doppler ultrasound goal-directed therapy for the early management of severe traumatic brain injury

	Abnormal admission TCD ( <i>n</i> = 11)		Normal admission TCD ( <i>n</i> = 13)	
	T0	T1	T0	T1
Delay from admission (min)	16 ± 8	219 ± 96	20 ± 12	262 ± 123
Abnormal TCD ( <i>n</i> )	11	2	0	0
Mean velocity (cm/s)	30 ± 6	43 ± 10*	49 ± 13**	51 ± 11
Diastolic velocity (cm/s)	13 ± 5	25 ± 8*	34 ± 11**	36 ± 11**
Pulsatility index	2.1 ± 0.5	1.4 ± 0.3*	1.2 ± 0.6**	0.9 ± 0.3**
MAP (mmHg)	89 ± 15	105 ± 17*	89 ± 11	93 ± 19
ICP (mmHg)		32 ± 13		22 ± 10**
CPP (mmHg)		73 ± 15		71 ± 14
SjvO <sub>2</sub> (%)		67 ± 2		72 ± 9
pH	7.39 ± 0.04	7.39 ± 0.02	7.32 ± 0.06**	7.36 ± 0.07*
PaCO <sub>2</sub> (mmHg)	40 ± 5	42 ± 5	45 ± 6**	41 ± 6*
Haemoglobin (g/dl)	12 ± 1	11 ± 1	12 ± 2	11 ± 2
Norepinephrine ( <i>n</i> )	1	9	2	4
Mannitol ( <i>n</i> )	0	5	1	0
Neurosurgery ( <i>n</i> )	-	3	-	0

\*  $p < 0.05$  between T0 and T1

\*\*  $p < 0.05$  between groups

# MY PROPOSAL: nuanced Rx

Rx tailored

not to the primary issue  
(ICP vs CPP) alone,  
but to both issues...

Segregate pts into  
2x2 simple table  
to allocate the best Rx



# TABLE: Tailored treatment plan by categories of patients with acute brain injury at risk for ICP elevation.

Best Diastolic Flow Velocity /  
Pulsatility Index (PI)

PI < 1.2

PI > 1.2

Vd > 40

**Compliant brain** (unlikely to have significantly raised ICP), **well perfused** (likely adequate CPP):  
**Treat** by **continuing** current meds, current ventilator settings and same MAP goal in an **Intermediate** level of monitoring.

**Poorly compliant brain** (likely edematous or with raised ICP), but **well perfused** (with likely adequate CPP):  
**Treat** with **osmotherapy** (prefer **mannitol** over HTS), consider **mild hyperventilation** and try **mild decrease in MAP** (to reduce potential edema) in **NSICU**.

Vd < 40

**Compliant brain**, but **hypoperfused** (with likely inadequately low CPP):  
**Treat** with **no osmotherapy** (but if mandated by herniation, prefer **HTS** over mannitol), **mild hypoventilation** (a.k.a. permissive hypercapnia), and definitely **raise MAP** goal (with volume and pressors) in **NSICU**.

**Poorly compliant brain** (likely edematous or with raised ICP), **not well perfused** (with likely inadequately low CPP):  
**Treat** with **osmotherapy** (with **HTS**, not mannitol), normal ventilation and definitely **raise slowly MAP** goal (with volume and pressors) in **NSICU**.

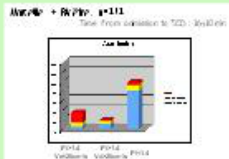




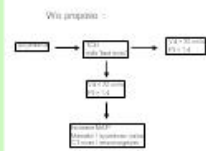
# TRIAGING, TREATING AND PROGNOSTICATING outcomes



Bernard Vigué  
(Kremlin Bicêtre, France)



Slide 24



Slide 25

Marseille + Bicêtre, n=151

Time from admission to TCD :  $16 \pm 10$

min

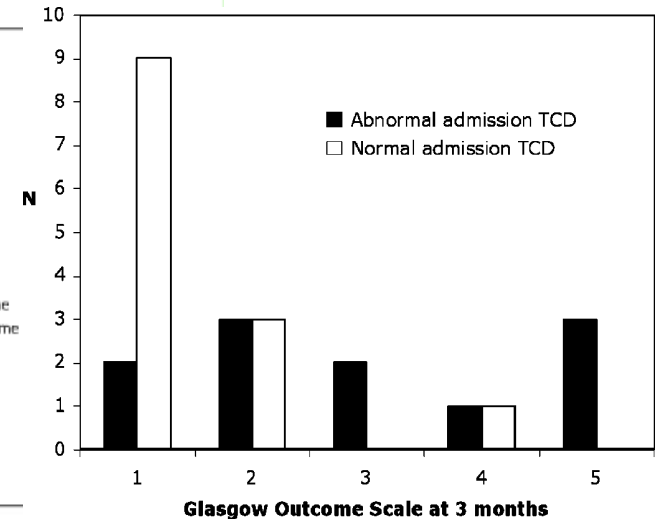
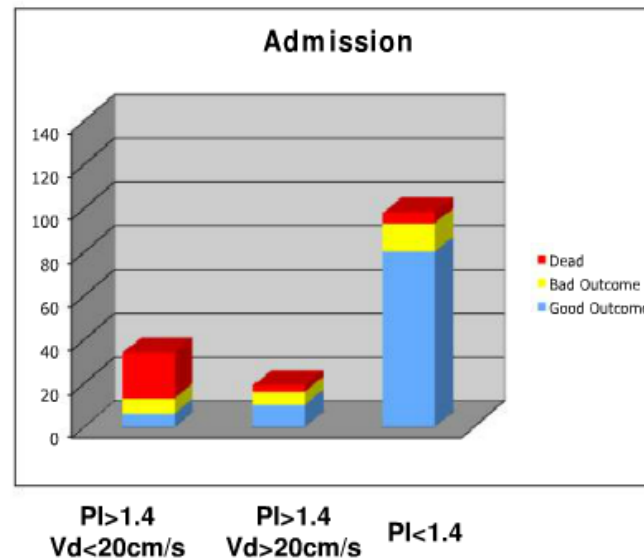


Fig.2 Three-month Glasgow Outcome Score in patients with abnormal admission TCD (filled columns) and in patients with normal admission TCD (open columns). 1 Good recovery, 2 moderate disability, 3 severe disability, 4 vegetative state, 5 dead



Ract et al. Intensive Care  
Med (2007) 33:645–651.

## Our Material & methods @NSLIJ 2013:

TCD for non-invasive ICP monitoring in 5 patients in our ICU with acute cerebral edema and risk for ICP-related secondary deterioration from global ischemia.

Deemed non-salvageable, non-surgical or at high bleeding risk for EVD.

- 1 hepatic failure with GCE
- 1 meningitis with GCE and hemispheric IPH with MLS
- 1 TBI (moderate, no surgical lesion, compensated DIC)
- 2 large hypertensive IPH with mass effect.

TCD was used to derive PI as a surrogate marker for brain compliance and diastolic velocity ( $V_d$ ) reflecting diastolic CBF inferring adequacy of CPP.

Applied Rx'ic choices falling under the 4 described categories to specifically address the cerebral needs of each group. (Table)

Measured adequacy of Rx by appreciating the response of each patient in terms of clinical stability, normalization of derived ICP parameters, decrease in GCE and absence of secondary ischemia.



## Our Results: PROOF OF CONCEPT / FEASIBILITY

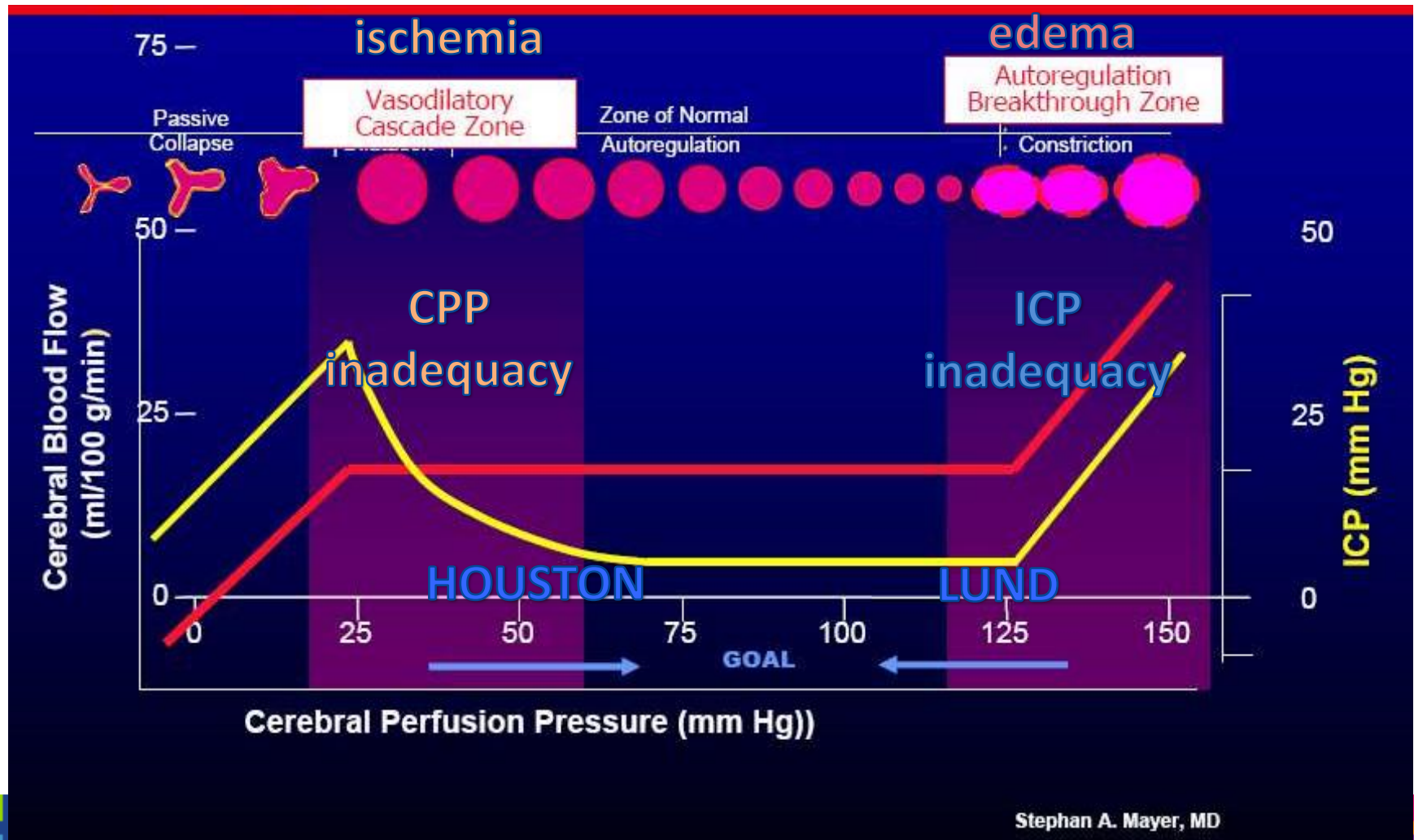
- 1 Pt had no change in mgmt b/o normal PI and Vd.
- 2 pts received HTS as favored osmotherapy along with induced HTN to alleviate the risk of ischemia due to raised ICP.
- 1 pt received mannitol and had vasopressors tapered off to address break-through pressure edema driving her ICP (Lund).
- 1 pt received hemodynamic augmentation for significant CPP amelioration, without any significant rise in PI (Houston).
- All patients had normalization of PI and Vd within our target range within an hour of the tailored therapy.
- No patient had neurological deterioration, worsening of GCE, MLS, new hemorrhage or developed infarcts within 48h of our repeated interventions.
- All succumbed to their brain injury before discharge from the ICU, except for the TBI patient.

# Conclusions

- PI and waveform analysis more informative than mean ICP.
- Compliance is a better gold standard to decide on osmotherapy.
- Extracting PI from EDV may make sense to tease out ICP vs CPP issues.
- TCD easily predict/detect early ICP/CPP issues in the ED.
- My 4-category-tailored goal-directed Rx makes sense, seems beneficial and not detrimental.
- Larger feasibility study with pts with EVDs, then RCT to compare conventional monolithic ICP therapy (mainly osmotherapy and MAP augmentation for all) to this 4-category tailored ICP therapy.
- It can then be even more refined, adding ONSD, RI, novel index, or adding CAR (vasoreactivity testing), MOCAIP (VD/VC state), optimal CPP (PRx or pbtO<sub>2</sub>), LPR (CMD), OEF and spectroscopy (neuroimaging).



Enhanced accuracy:  
NIRS, SVJO2, CBF  
ONSD, CBF H2O CONTENT  
ameliorate PI & RI, MOCAIP...



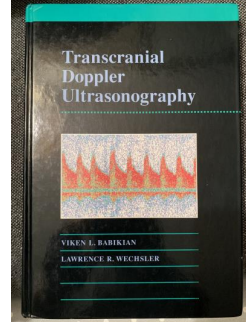
# Arterial and arteriolar resistance

- Pulsatility Index (PI):
  - $V_s - V_d / \text{mean}$
  - $\text{PSV} - \text{EDV} / (2\text{EDV} + 1\text{PSV}/3)$
  - Marek Czosnyka (Cambridge)
- Resistivity Index (RI):  $V_s - V_d / V_s$ 
  - Leandre Pourcelot (Tours)



# RI better than PI?

- RIx100 correlates to shunt malfunction:
  - Normal ~50% vs. sx'ic ~70%
  - Chadduck et al.



Klingelhofer et al.: **RI is important in discerning whether pts are at risk for poor CPP from VSP or raised ICP.**

RI may change as a function either ICP or MFV:

At a fixed MFV, RI rises when ICP rises and falls when ICP falls.  
At a fixed ICP, however, RI is  $1/\text{MFV}$ .

When  $\text{RI} < 0.5$  and  $\text{MFV} > 120$ , ICP is always  $< 20$ .  
But when  $\text{ICP} > 20$  and  $\text{MFV} < 150$ , RI is always  $> 0.6$ .

“Thus, when  $\text{RI} < 0.5$ , changes in MFV reflect severity of VSP and ICP is expected to be low.

But if RI rose to  $> 0.6$  and MFV declines simultaneously in a pt with VSP, there is a problem with ICP rise compromising CPP.”



# Refining compliance and perfusion adequacy biomarkers

- RI better than PI?
- RI/MFV?
- Trends rather than thresholds?
- Moving correlation coefficients?
- Active dynamic better than passive Mx?
- Challenges (BHI, acetazolamide, carbogen)?



- Term “cerebral autoregulation”:
  - Pressure autoregulation vs. CO<sub>2</sub> reactivity
    - One does not predict the other...
  - CO<sub>2</sub> reactivity in direction of VC by HV (drop in CO<sub>2</sub>) vs. CO<sub>2</sub> reactivity in direction of VD by apnea (BHI accumulating CO<sub>2</sub>)...
    - One does not predict the other...
- “Loss vs. conserved” CAR is too Manichean:
  - F/u trends gradual amenutising CO<sub>2</sub> reactivity might predict VSP/DCI or impaired compliance even better than overt impairment of vasoreactivity
  - Peak slope of decrease or EDV trends and correlation coefficient RI/MAP might correspond better to adequacy of CPP irrespective of ICP





# We should do more TCDs in the Neuro-ICU:



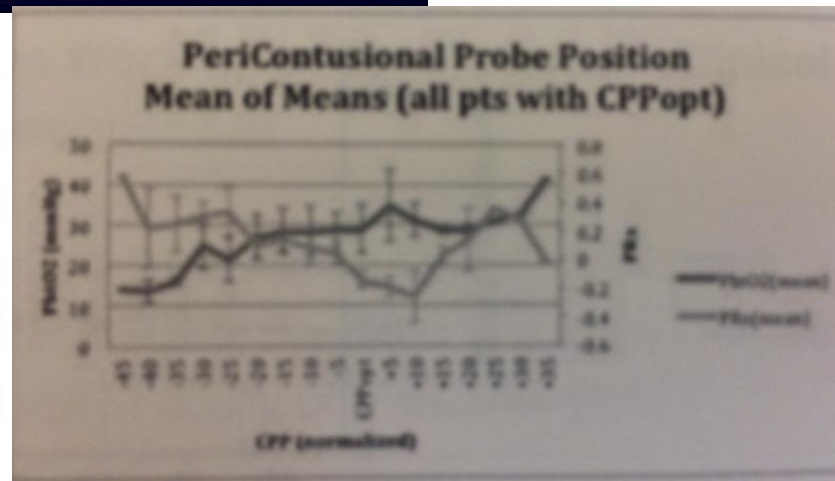
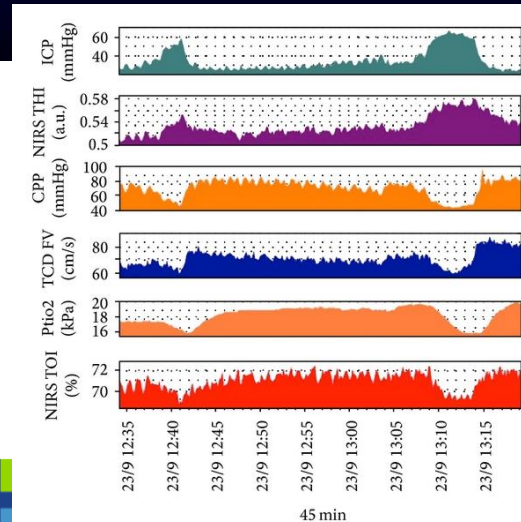
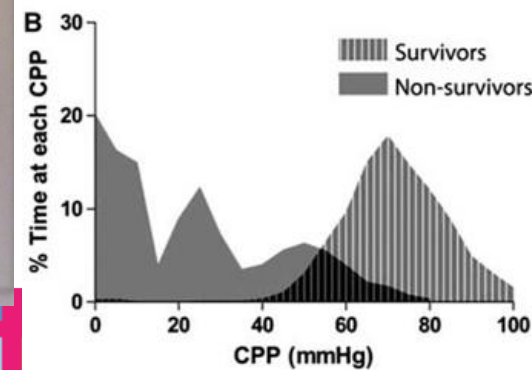
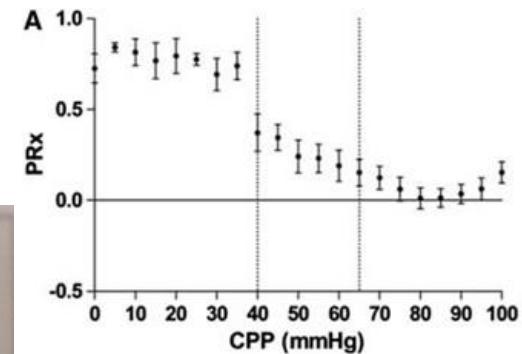
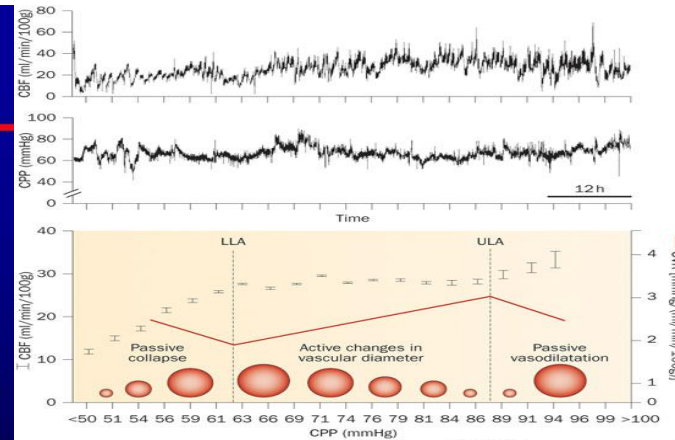
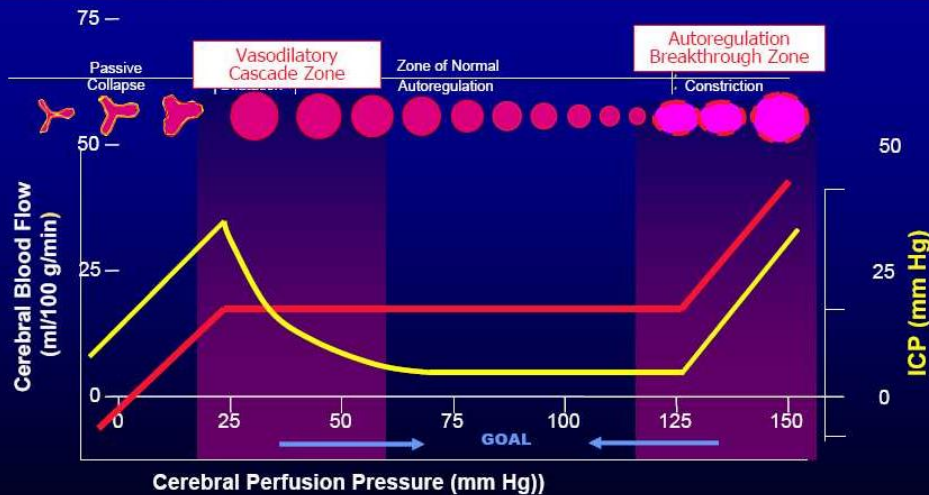
- TCD to tailor BP in AIS to evaluate:
  - Recanalized post IV tPA but also w/o tPA (penumbral salvage by HDA)
  - RI inferring VD in capillary bed
  - CBF conservation/restoration
  - **Collaterals status**
  - Reserve in all ischemic pts (not only Moya-Moya) by acetazolamide
  - **Hyperemia, risk of severe edema and HT**

In order to tailor BP and AC Rx



# Revisiting Laasen's and Czosnyka's curves and K: CI replaces MAP/CPP, better PRx/Mx and ORx/pbtO2-K and then what if API replaces CBF or neuronal fxn/clinical exam...

## Hypertension Can Drive Elevated Intracranial Pressure



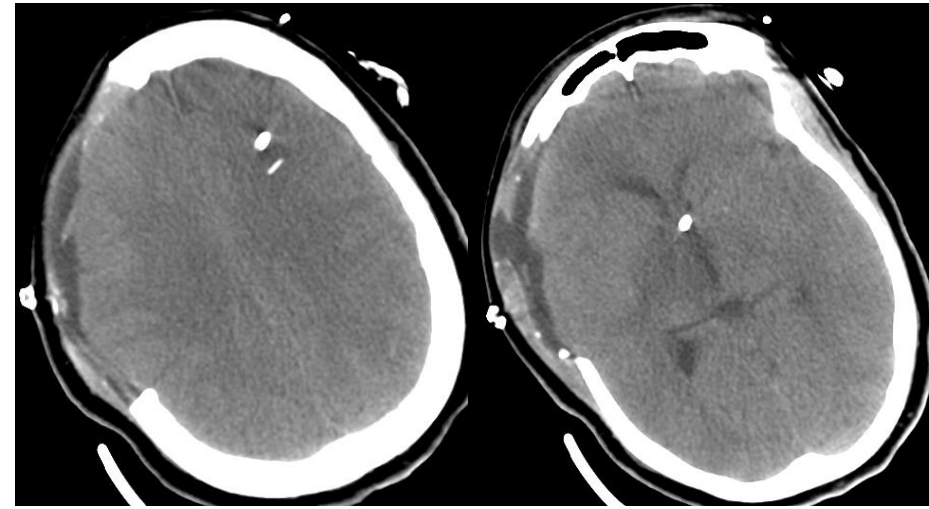
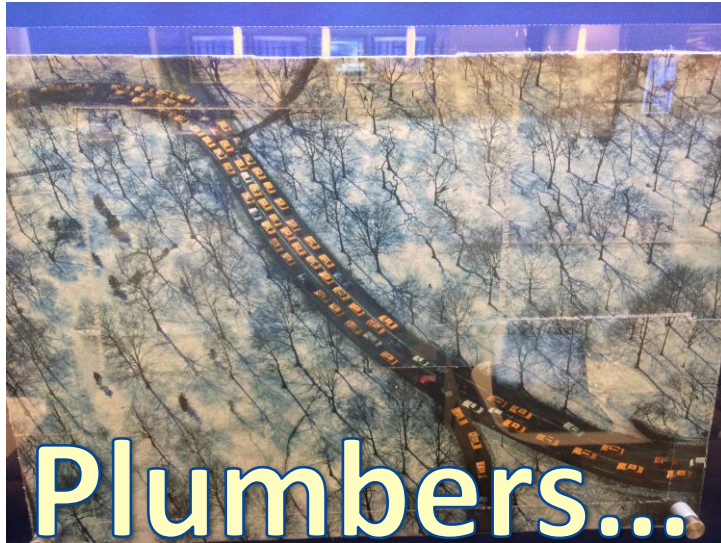
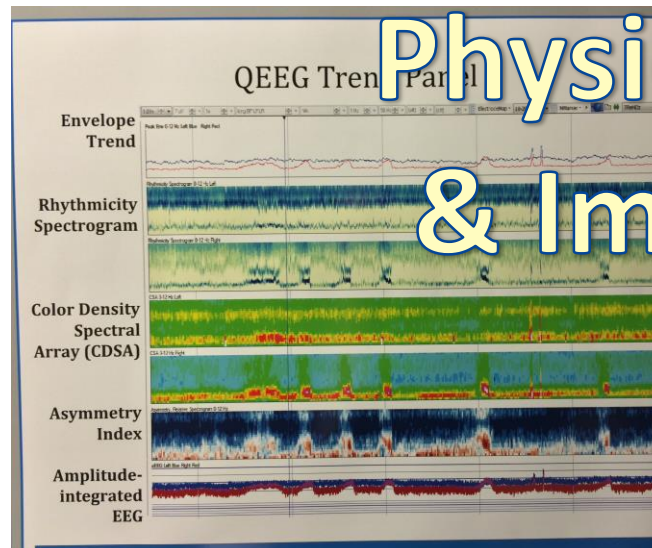


Figure 12: Post-bleed day 15 (at the peak of vasospasm), head CT reveals no lucency to suggest any new infarcted territories, but persistent global cerebral edema.

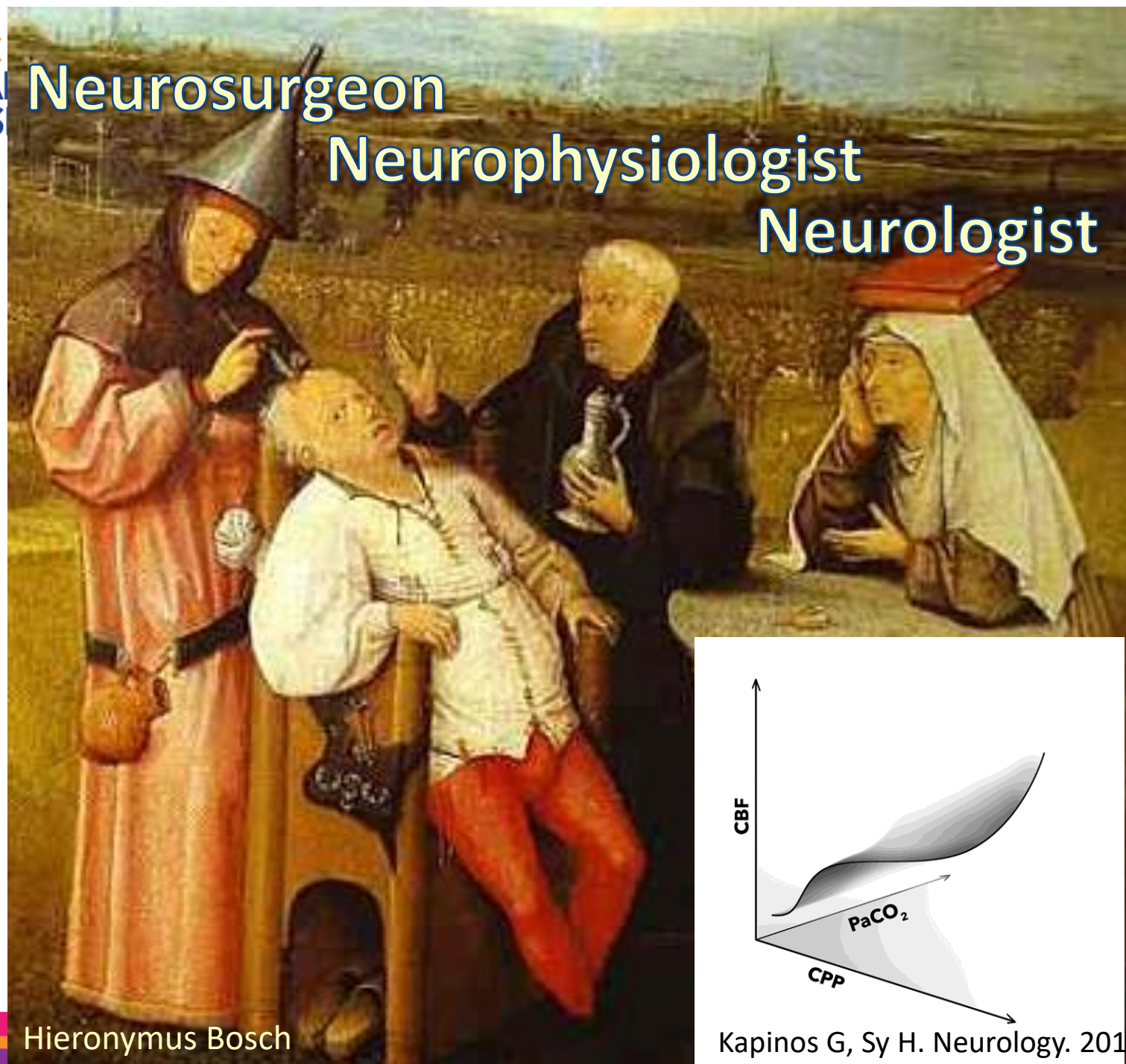


Physiologists  
& Imagers...

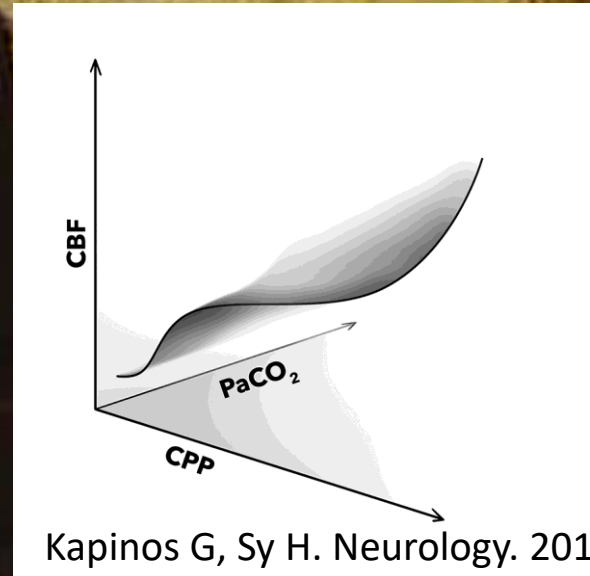




# Neurosurgeon Neurophysiologist Neurologist



Hieronimus Bosch



Kapinos G, Sy H. Neurology. 2016



Thank you!

[kapigreg@gmail.com](mailto:kapigreg@gmail.com)

Fellowships

NCC+ICUEEG+NI+Nu/s

**ASN 42<sup>ND</sup> ANNUAL MEETING  
JANUARY 24-26, 2019**



**PUERTO RICO** ★