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# PROMPT BRAIN DEATH DIAGNOSIS OF THE POTENTIAL DONOR

# IMPLICATIONS OF CARDIOTHORACIC ORGANS


Rafael Badenes MD, PhD

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Hospital Clínico Universitario. Valencia



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**Valencia 2 de Diciembre de 2013**

# PROMPT BRAIN DEATH DIAGNOSIS

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Flash conferences – Lisbon 2012  EACTA – The European Association of Cardiothoracic Anaesthesiologists

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1. Current practice of **brain death** d...nfirmatory tests in an Italian University hospital: a report of 66 cases.
Vicenzini E, Pro S, Pulitano P, Rocco M, Spadetta G, Zarabla A, Di Piero V, Mecarelli O.
Minerva Anestesiol. 2013 May;79(5):485-91. Epub 2013 Feb 18.
PMID: 23419337 [PubMed - in process]
Related citations

2. Endovascular management of acute stroke.
Simonetti G, Stefanini M, Konda D, Marziali S, Da Ros V, Chiaravalloti A, Pampana E, Gandini R.
J Cardiovasc Surg (Torino). 2013 Feb;54(1):101-14. Review.
PMID: 23296420 [PubMed - indexed for MEDLINE]


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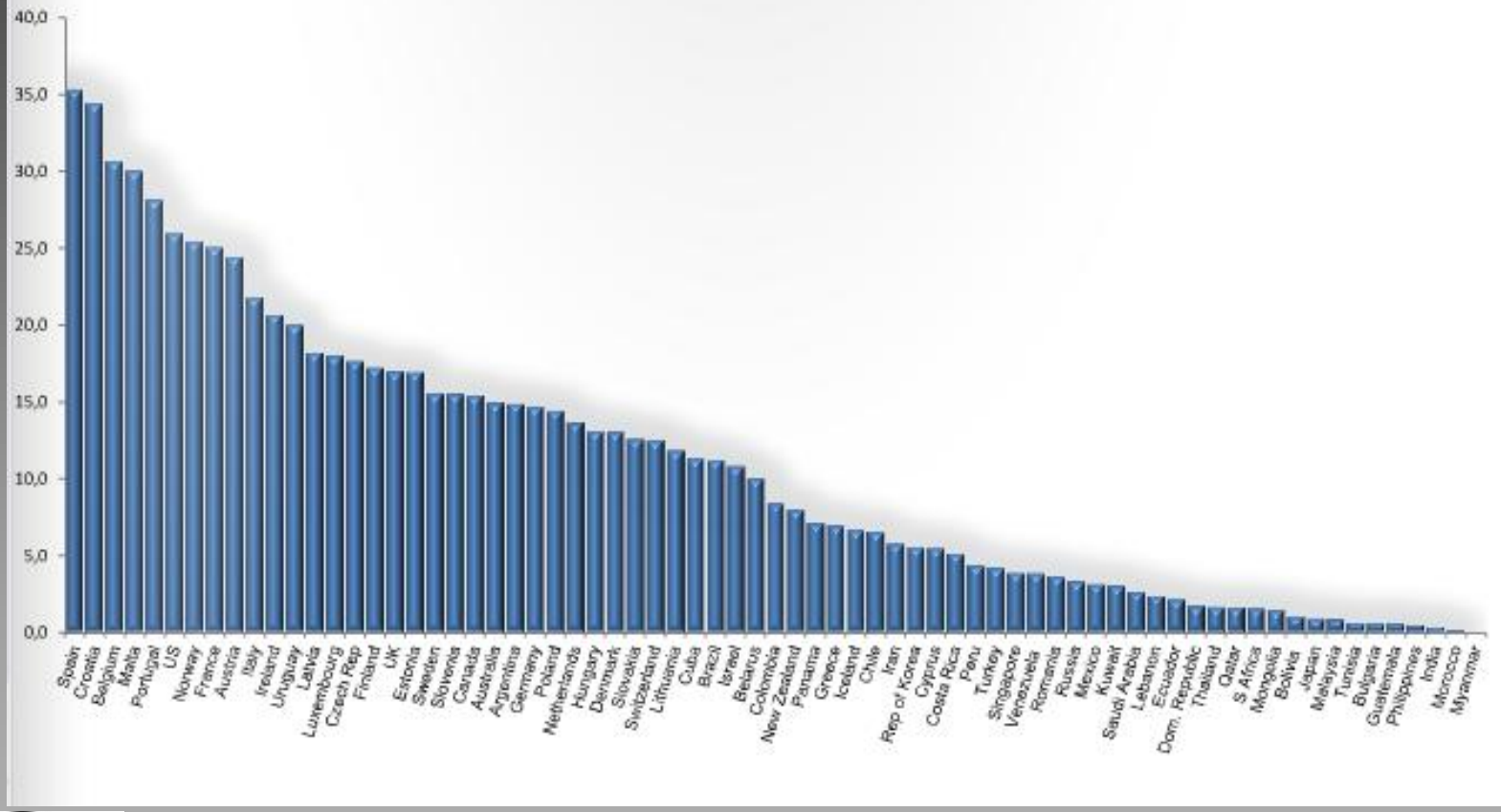
GODT Global Observatory on Donation & Transplantation

In collaboration with World Health Organization

ONT

GODT 2011

# Donation from deceased persons (pmp)

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# Transplants & Waiting Lists in the EU. 2011

## (Newsletter Transplant, 2012)



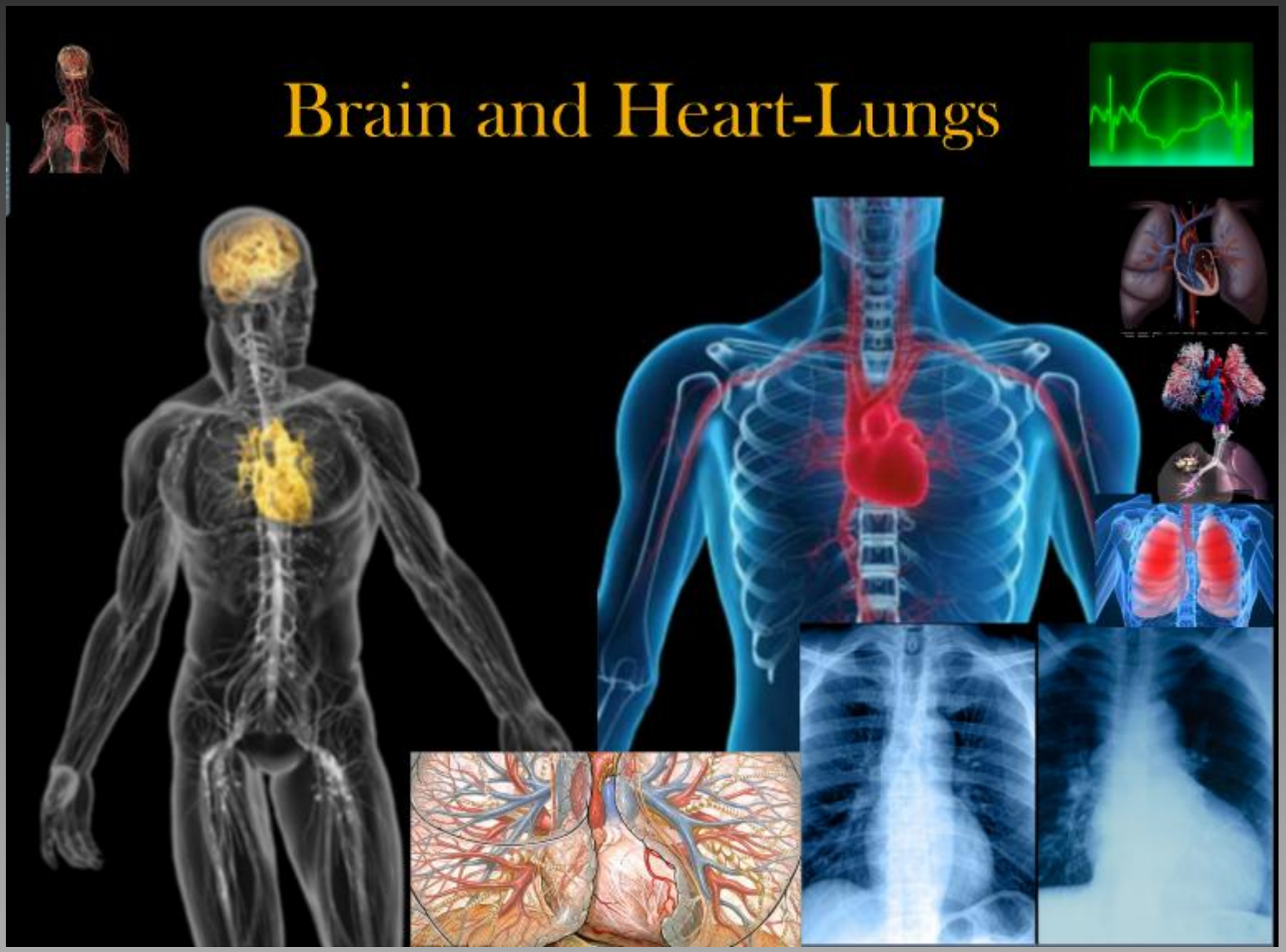
| | TRANSPLANTS | WAITING LIST* |
|---|---|---|
| | 18,712 | 43,396 |
| | 7,006 | 6,810 |
| | 1,980 | 3,568 |
| | 1,677 | 2,342 |
| TOTAL | 29,375 | 56,116 |

*Candidates at the end of the year

For many, the waiting continues …more than 2,700 patients died while wating to be transplanted in 2011

*Newsletter Transplant 2012 http://www.ont.es/publicaciones/Documents/NEWSLETTER2012.pdf*

# Brain and Heart-Lungs



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Neuroscience and Biobehavioral Reviews 33 (2009) 81–88

Contents lists available at ScienceDirect

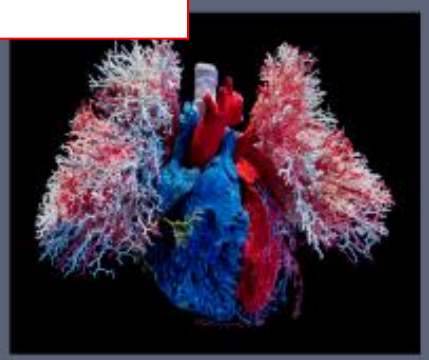
Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review

# Claude Bernard and the heart–brain connection: Further elaboration of a model of neurovisceral integration

Julian F. Thayer [a,b,*], Richard D. Lane [c]



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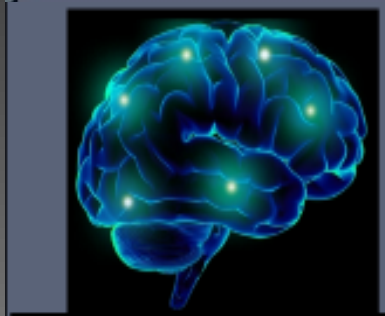
# Organ Crosstalk

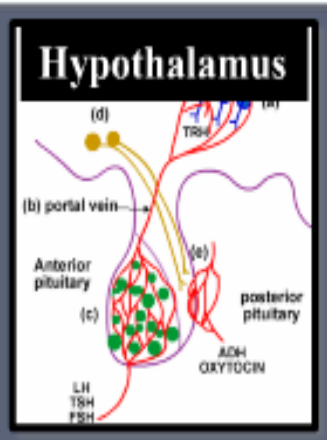


Prefrontal Cortex

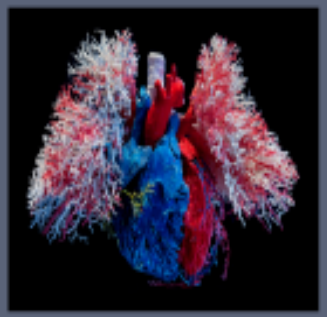
Insula

Cingulatel Cortex

Amygdala



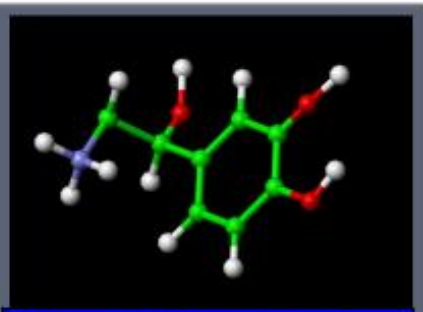

Nucleus Solitary Tract
Ventrolateral Medullary Nuclei



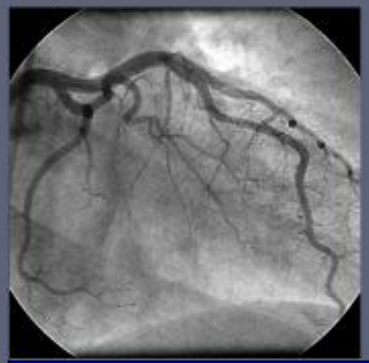
Vagus Nerve
Sympathetic Neurons

# Heart Dysfunction

Pituitary Insufficiency ??
Inflammation ??

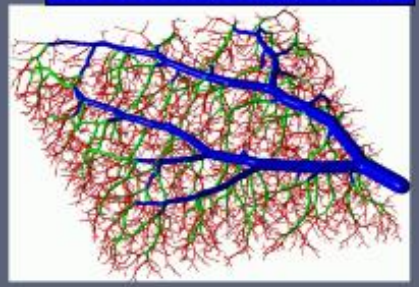



Direct Cathecolamines
Toxic Effects



Epicardial Coronary Spasm

Altered MC and rBF



**Seok, K Circ J 2012**
**Makikallio, Neurology 2004**
**Min, Stroke 2009**
**Yousef, Neurocrit Care 2010**

# Lung Dysfunction


**PRIMARY**
Cathecolamines
Neurokines

**SECONDARY**
Chemokines
Cytokines

Alteration of capillary permeability
Venocostriction
Hemodynamic changes
Bronchocostriction
Reduced surfactant

Microcirculation
Endothelium
Shear stress
Tissue ischemia

**Heuer, Intensive Care Med 2011**
**Kalsotra, J Cer Blood Flow Met 2004**
**Wu, Exp Neurol 2006**
**López Aguilar, CCM 2005**

Heuer, Jan Florian; Selke, Maren; Crozier, Thomas A.; Pelosi, Paolo; Herrmann, Peter; Perske, Christina; Quintel, Michael:

# Effects of Acute Intracranial Hypertension on Extracerebral Organs: A Randomized Experimental Study in Pigs

J Neurol Surg A Cent Eur Neurosurg. 2012 Sep;73(5):289-95.

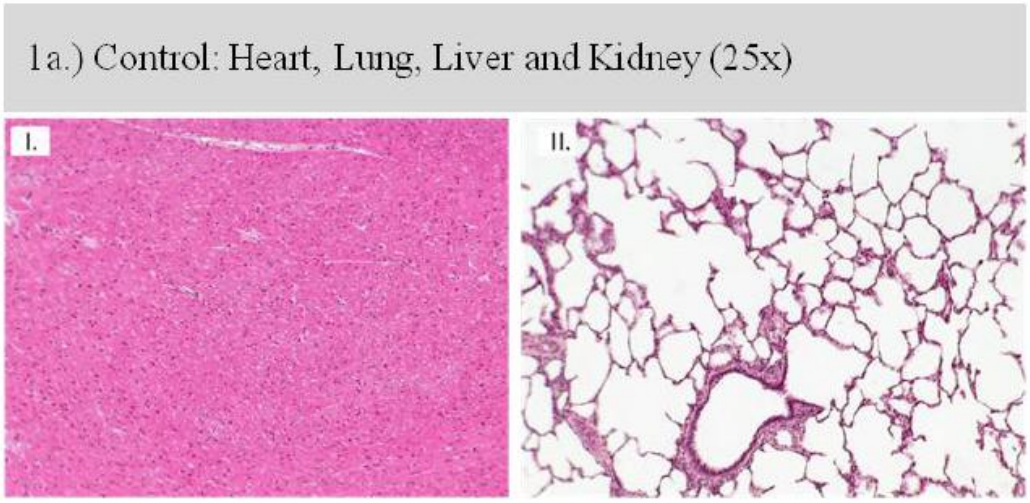

A total of 14 mechanically ventilated pigs were randomized to two groups of seven each: (1) control and (2) AICH.

Heuer, Jan Florian; Selke, Maren; Crozier, Thomas A.; Pelosi, Paolo; Herrmann, Peter; Perske, Christina; Quintel, Michael:

# Effects of Acute Intracranial Hypertension on Extracerebral Organs: A Randomized Experimental Study in Pigs

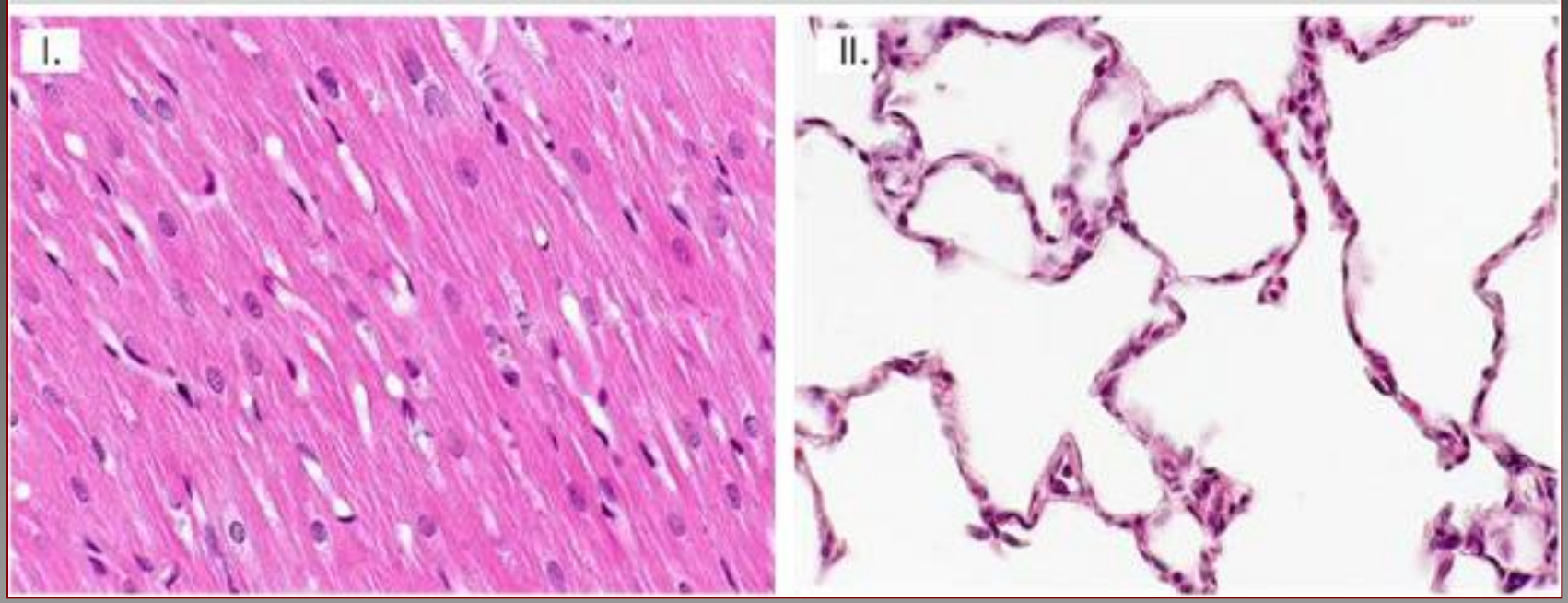
J Neurol Surg A Cent Eur Neurosurg. 2012 Sep;73(5):289-95.

Figure 1. Histology in the Control Group at 25x and 100x magnification



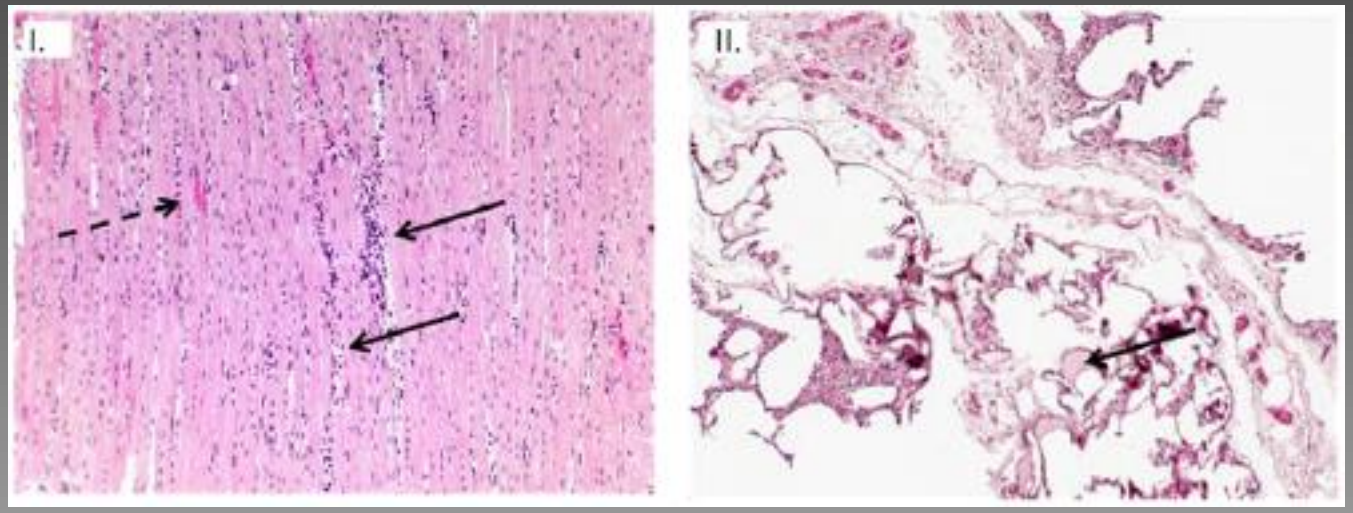

1a.) Magnification 25x. I.) left ventricle: normal cardiomyocytes; II.) Lung: normal lung tissu

# 1b.) Control: Heart, Lung, Liver and Kidney (100x)



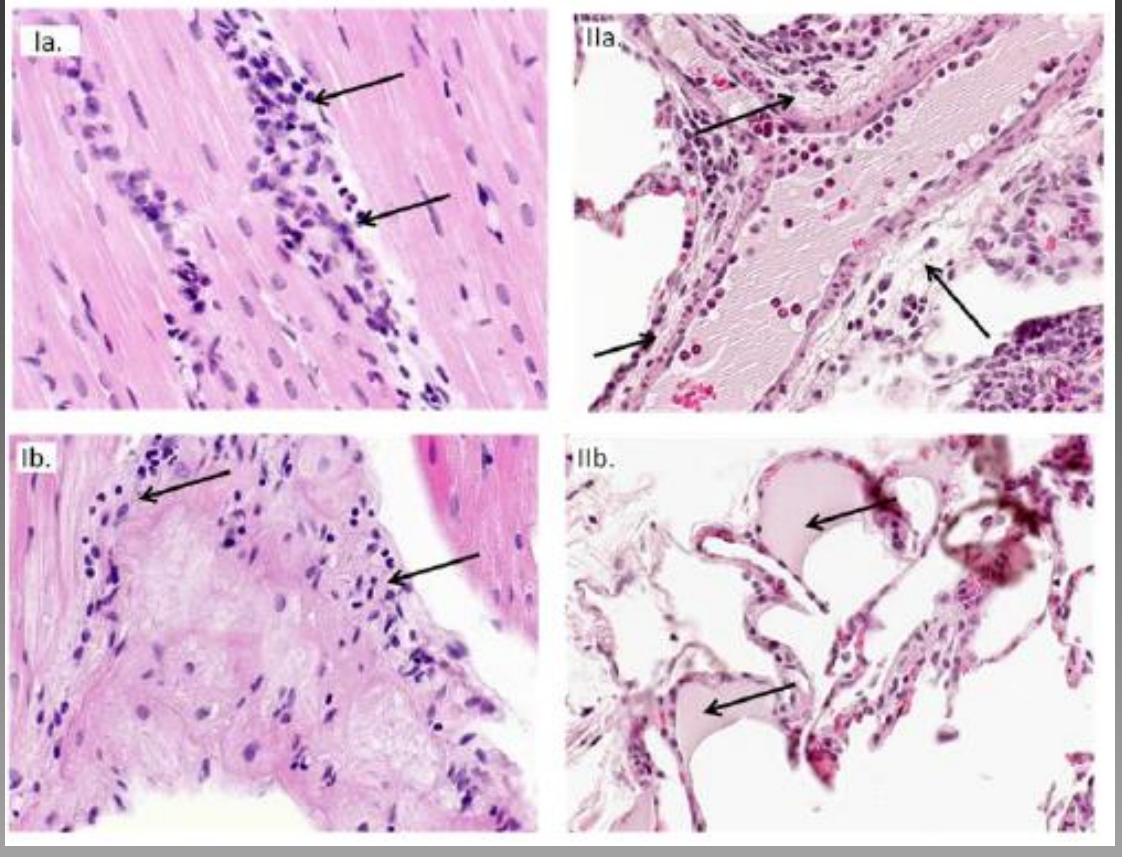

1b.) Magnification 100x. I.) left ventricle: normal cardiomyocytes; II.) Lung: normal lung tissue;

Figure 2. Histology in the AICH Group at 25x and 100x magnification

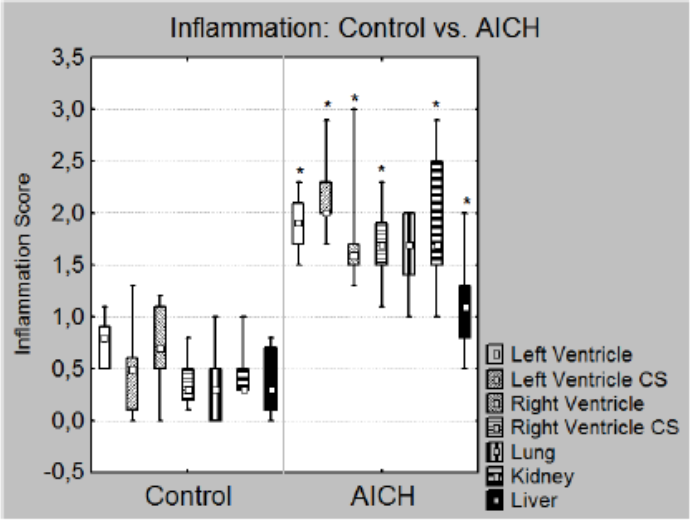


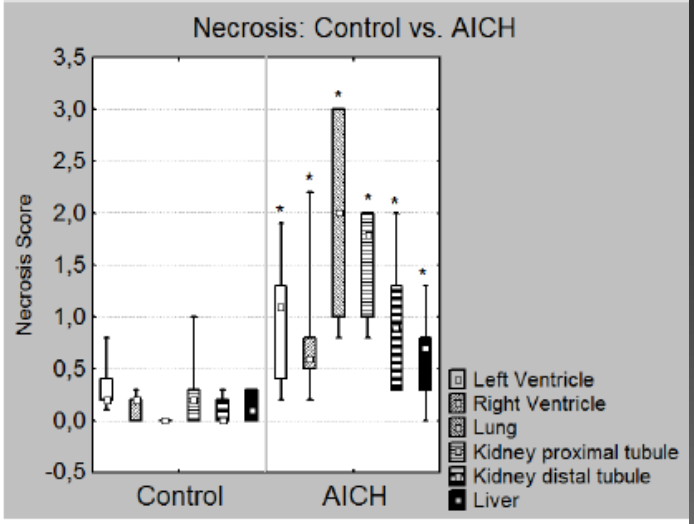

Magnification 25x. I.) Left ventricle: massive leukocyte infiltration (←) and damaged cardiomyocytes (⇢); II.) Lung: intra- + extra alveolar exudation;

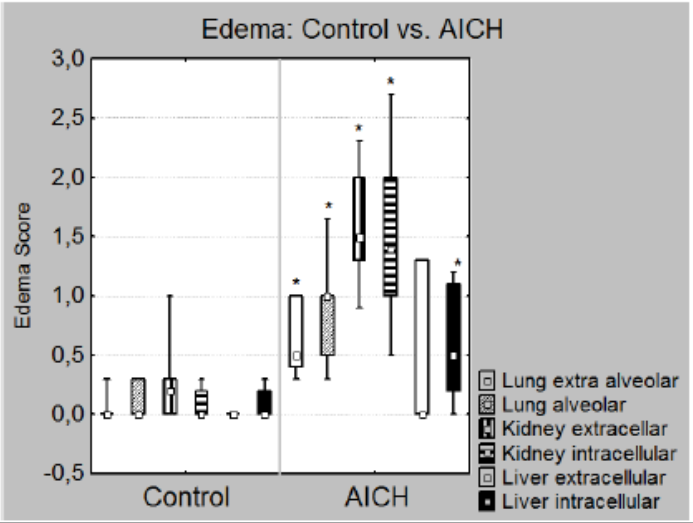
## AICH: Heart, Lung, Liver and Kidney (100x)




Magnification 100x. Ia.) Left ventricle: massive increased number of inflammatory cells, Ib.) Left ventricle: inflammatory cell along the impulse conduction system; IIa.) Lung: interstitial exudation(←), neutrophilic granulocytes, IIb.) Lung: intraalveolar edema (←); IIIa.) Liver: infiltration



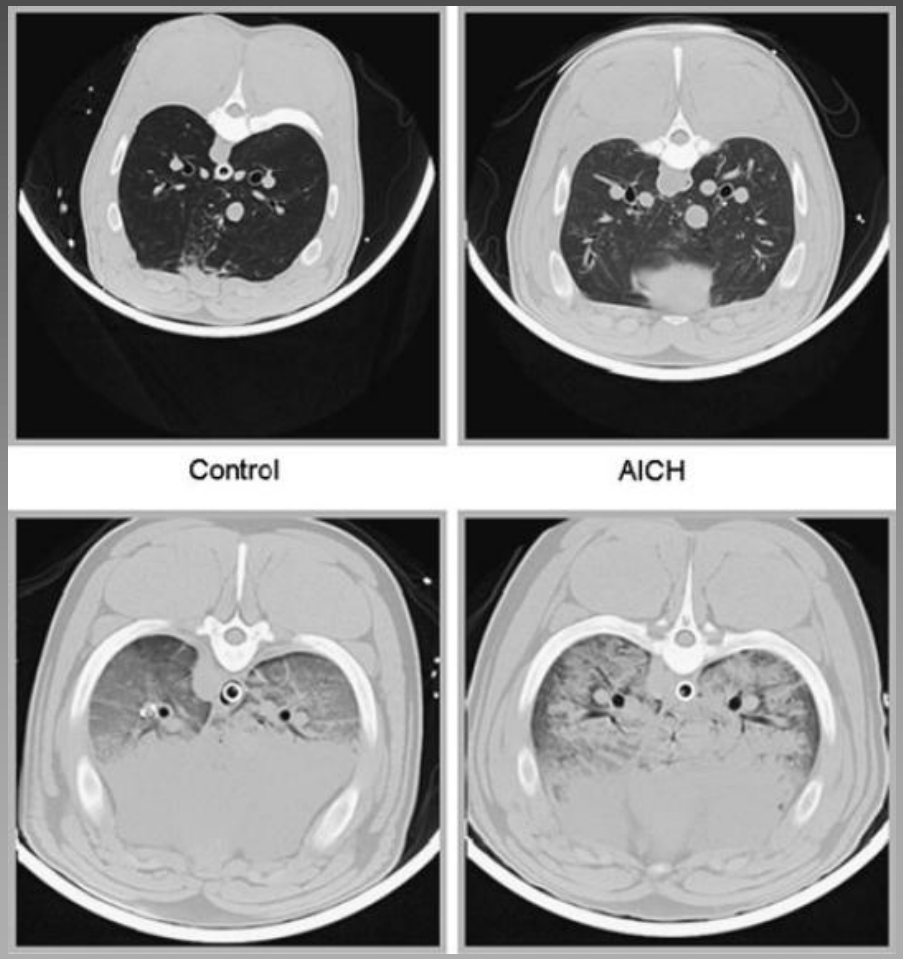




**CONCLUSIONS:** Isolated AICH induces injury to multiple extracerebral organs, even in the absence of hypoperfusion or hypoxemia.

# Acute effects of intracranial hypertension and ARDS on pulmonary and neuronal damage: a randomized experimental study in pigs

Intensive Care Med (2011) 37:1182–1191
DOI 10.1007/s00134-011-2232-2

Jan Florian Heuer
Paolo Pelosi
Peter Hermann
Christina Perske
Thomas A. Crozier
Wolfgang Brück
Michael Quintel

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doi:10.1093/brain/awr282 Brain 2012: 135; 1321–1331 | 1321

BRAIN
A JOURNAL OF NEUROLOGY

OCCASIONAL PAPER

# The transatlantic divide over brain death determination and the debate

Eelco F. M. Wijdicks

Division of Critical Care Neurology, Department of Neurology, Mayo Clinic, Rochester, MN, USA


Figure 1 Opening shot of the BBC programme, with David Dimbleby presenting.

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doi:10.1093/brain/awr282 Brain 2012: 135; 1321–1331 | 1321

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A JOURNAL OF NEUROLOGY

OCCASIONAL PAPER

# The transatlantic divide over brain death determination and the debate

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Division of Critical Care Neurology, Department of Neurology, Mayo Clinic, Rochester, MN, USA

## Lessons learned and afterword

The development of brain death criteria—both in the UK and USA—has been well chronicled (Pallis, 1983; Wijdicks, 2003, 2011).

Once accepted as a neurological definition of death, medical interventions are now futile and unnecessary. The diagnosis of

SPECIAL ARTICLE

AMERICAN ACADEMY OF NEUROLOGY

# Evidence-based guideline update: Determining brain death in adults

Report of the Quality Standards Subcommittee of the American Academy of Neurology

Eelco F.M. Wijdicks, MD, PhD
Panayiotis N. Varelas, MD, PhD
Gary S. Gronseth, MD
David M. Greer, MD, MA

Neurology® 2010;74:1911-1918

The determination of brain death can be considered to consist of 4 steps.

I. The clinical evaluation (prerequisites).

II. The clinical evaluation (neurologic assessment).

III. Ancillary tests.

IV. Documentation.

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Neurology® 2010;74:1911-1918


# I. The clinical evaluation (prerequisites).

## A. Establish irreversible and proximate cause of coma.

The cause of coma can usually be established by history, examination, neuroimaging, and laboratory tests.

Exclude the presence of a CNS-depressant drug effect by history, drug screen, calculation of clearance using 5 times the drug's half-life (assuming normal hepatic and renal function), or, if available, drug plasma levels below the therapeutic range. Prior use of hypothermia (e.g., after cardiopulmonary resuscitation for cardiac arrest) may delay drug metabolism. The legal alcohol limit for driving (blood alcohol content 0.08%) is a practical threshold below which an examination to determine brain death could reasonably proceed.

**Neurology® 2010;74:1911-1918**


## I. The clinical evaluation (prerequisites).

### A. Establish irreversible and proximate cause of coma.

The cause of coma can usually be established by history, examination, neuroimaging, and laboratory tests.

There should be no recent administration or continued presence of neuromuscular blocking agents (this can be defined by the presence of a train of 4 twitches with maximal ulnar nerve stimulation).

There should be no severe electrolyte, acid-base, or endocrine disturbance (defined by severe acidosis or laboratory values markedly deviated from the norm).

Neurology® 2010;74:1911-1918


I. The clinical evaluation (prerequisites).

A. Establish irreversible and proximate cause of coma.

B. Achieve normal core temperature.
In most patients, a warming blanket is needed to raise the body temperature and maintain a normal or near-normal temperature (>36°C). After the initial equilibration

**Neurology® 2010;74:1911-1918**


I. The clinical evaluation (prerequisites).

A. Establish irreversible and proximate cause of coma.

C. Achieve normal systolic blood pressure. Hypotension from loss of peripheral vascular tone or hypovolemia (diabetes insipidus) is common; vasopressors or vasopressin are often required. Neurologic examination is usually reliable with a systolic blood pressure ≥100 mm Hg.

**Neurology® 2010;74:1911-1918**


I. The clinical evaluation (prerequisites).

A. Establish irreversible and proximate cause of coma.

D. Perform 1 neurologic examination (sufficient to pronounce brain death in most US states).

**Neurology® 2010;74:1911-1918**


I. The clinical evaluation (prerequisites).

A. Establish irreversible and proximate cause of coma.

BOLETÍN OFICIAL DEL ESTADO

Núm. 313 Sábado 29 de diciembre de 2012 Sec. I. Pág. 89315

## I. DISPOSICIONES GENERALES

### MINISTERIO DE SANIDAD, SERVICIOS SOCIALES E IGUALDAD

**15715** *Real Decreto 1723/2012, de 28 de diciembre, por el que se regulan las actividades de obtención, utilización clínica y coordinación territorial de los órganos humanos destinados al trasplante y se establecen requisitos de calidad y seguridad.*

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II. The clinical evaluation (neurologic assessment).

A. Coma.

B. Absence of brainstem reflexes.

C. Apnea.


III. Ancillary tests.

In clinical practice, EEG, cerebral angiography, nuclear scan, TCD, CTA, and MRI/MRA are currently used ancillary tests in adults (see appendix 1). Most hospitals will have the logistics


II. The clinical evaluation (neurologic assessment).

A. Coma.

B. Absence of brainstem reflexes.

C. Apnea.


IV. Documentation.

The time of brain death is documented in the medical records. Time of death is the time the arterial $P_{CO_2}$ reached the target value. In patients with an aborted apnea test, the time of death is when the ancillary test has been officially interpreted. A checklist is filled out,

# Hemodynamic and Oxygen Transport Patterns After Head Trauma and Brain Death: Implications for Management of the Organ Donor


*Howard Belzberg, MD, William C. Shoemaker, MD, Charles C. J. Wo, BS, Timothy P. Nicholls, MD, Alexis B. C. Dang, BS, Vladimir Zelman, MD, J. Peter Gruen, MD, Thomas V. Berne, MD, and Demetrios Demetriades, MD, PhD*

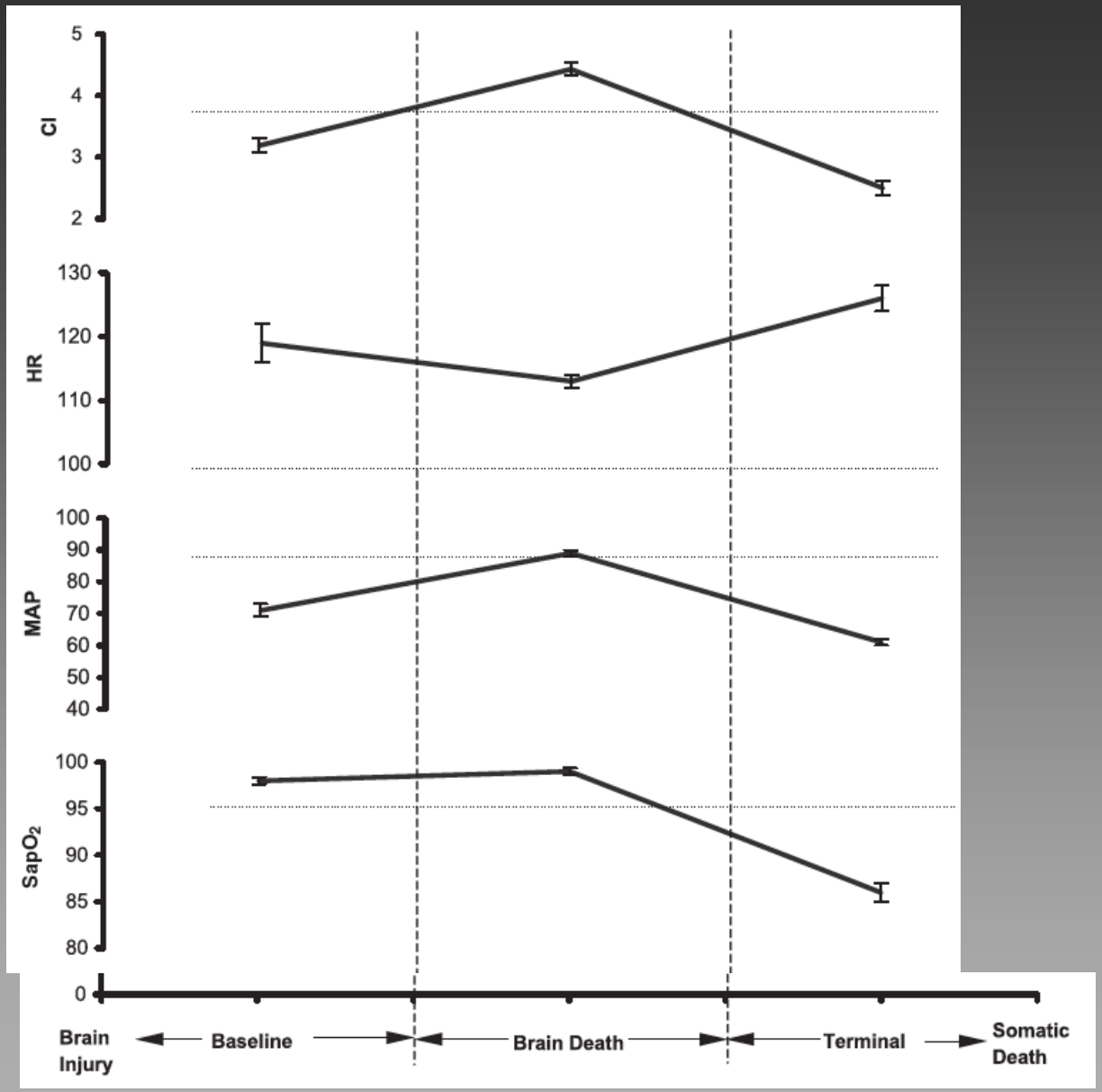
388 patients
79 BD

*J Trauma.* 2007;63:1032–1042.

**Table 3** Mean Values ± SD for Monitored Variables: Baseline at the Time of Diagnosis of Brain Death, During Brain Death, and at the Terminal Hemodynamic Stage

| Variable | Baseline Brain Death (N = 21)* 1.3 ± 1.7 h† | During Brain Death (N = 87) 4.9 ± 2.4 h | Terminal Stage (N = 9) 1.3 ± 1.6 h |
|---|---|---|---|
| CI ($L \cdot min^{-1} \cdot m^{-2}$) | 3.19 ± 1.26 | 4.43 ± 1.33 | 1.76 ± 1.10 |
| HR (beats/min) | 119 ± 29 | 113 ± 23 | 126 ± 28 |
| MAP (mm Hg) | 71 ± 23 | 89 ± 22 | 61 ± 16 |
| $Sapo_2$ (%) | 98 ± 3 | 99 ± 3 | 86 ± 21 |
| $Ptcco_2$, (mm Hg) | 54 ± 24 | 40 ± 15 | 59 ± 33 |
| $Ptco_2/Fio_2$ | 103 ± 86 | 238 ± 186 | 70 ± 53 |
| $DO_2$, ($mL \cdot min^{-1} \cdot m^{-2}$) | 399 ± 152 | 738 ± 185 | 283 ± 108 |

* Number of patients in each group.
† Mean ± SD hours of continuous monitoring.

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# Hemodynamic and Oxygen Transport Patterns After Head Trauma and Brain Death: Implications for Management of the Organ Donor

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Therapy for brain-dead patients awaiting organ donations may differ from that of other late stage trauma patients who may have low flow, hypotension, and poor tissue perfusion that require circulatory resuscitation with transfusions, fluids, and vasopressors. The hypotension of brain death is more often caused by the absence of central vasoconstriction than hypovolemia, particularly when CI and tissue perfusion are higher than normal. Of course, transfusions and fluids are indicated for low flow and poor tissue perfusion. Mild hypotension, which is characteristic of brain-dead patients (Table 3), is tolerated if accompanied by high flow and tissue perfusion. Severe hypotension (MAP <60 mm Hg) associated with hypovolemia and low flow should be treated with transfusions and fluids.

# PULMONARY TRANSPLANTATION: THE ROLE OF BRAIN DEATH IN DONOR LUNG INJURY

VASSILIOS S. AVLONITIS,[1,4] ANDREW J. FISHER,[2] JOHN A. KIRBY,[1] AND JOHN H. DARK[1,3]

TRANSPLANTATION

Vol. 75, 1928–1933, No. 12, June 27, 2003

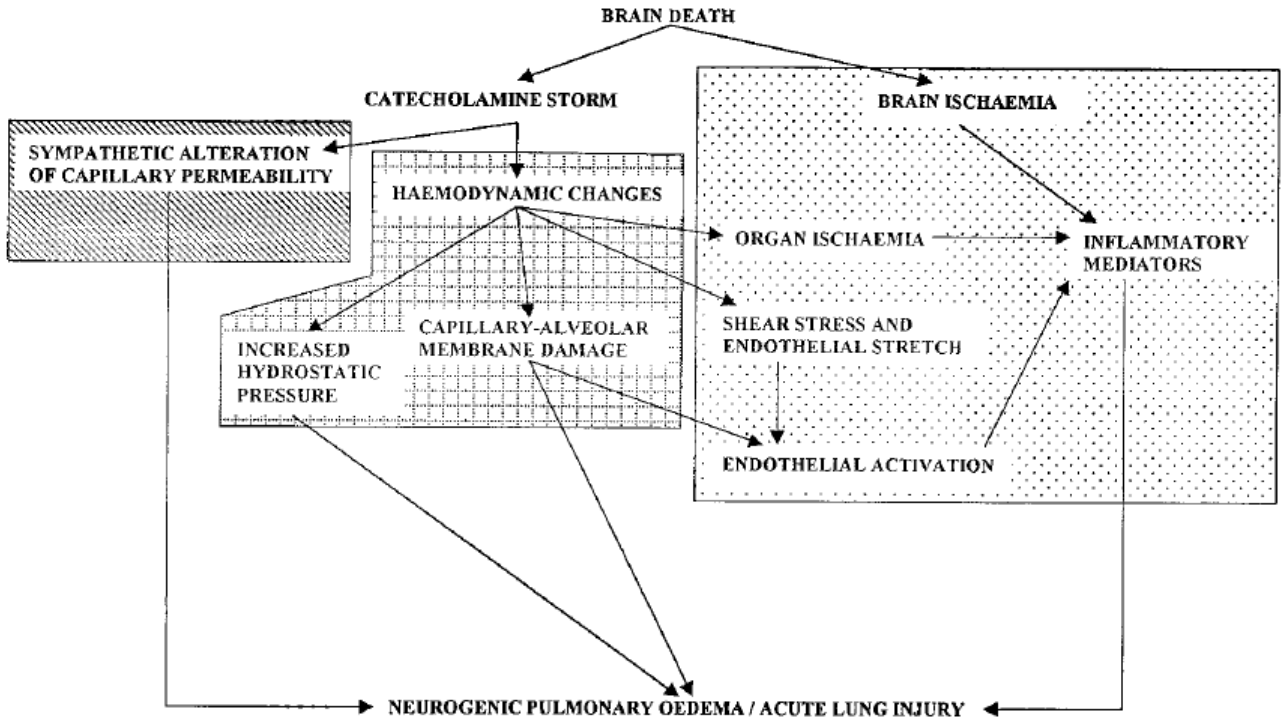


FIGURE 1. The three possible mechanisms (sympathetic, hemodynamic, and inflammatory) of lung injury resulting from brain death and their interrelations. The $\alpha$-adrenergic stimulation resulting from the catecholamine storm can cause neurogenic pulmonary edema (NPO) either by direct sympathetic alteration of the pulmonary capillary permeability (*left shaded box*) or by its hemodynamic effects (*middle shaded box*). At the same time, brain death induces an inflammatory response (*right shaded box*), which could be the result of brain ischemia or may have its origin in the hemodynamic effects of the catecholamine storm.

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# Evaluating lung injury at increasing time intervals in a murine brain death model

JOURNAL OF SURGICAL RESEARCH XXX (2013) E1–E8


BD, defined as an injury to the brain stem with complete irreversible loss of its function, is associated with severe hemodynamic changes, extensive hormonal alterations, and intense inflammatory activity influencing organ quality prior to transplantation [4–6]. After the earliest experimental

# Evaluating lung injury at increasing time intervals in a murine brain death model

JOURNAL OF SURGICAL RESEARCH XXX (2013) E1–E8


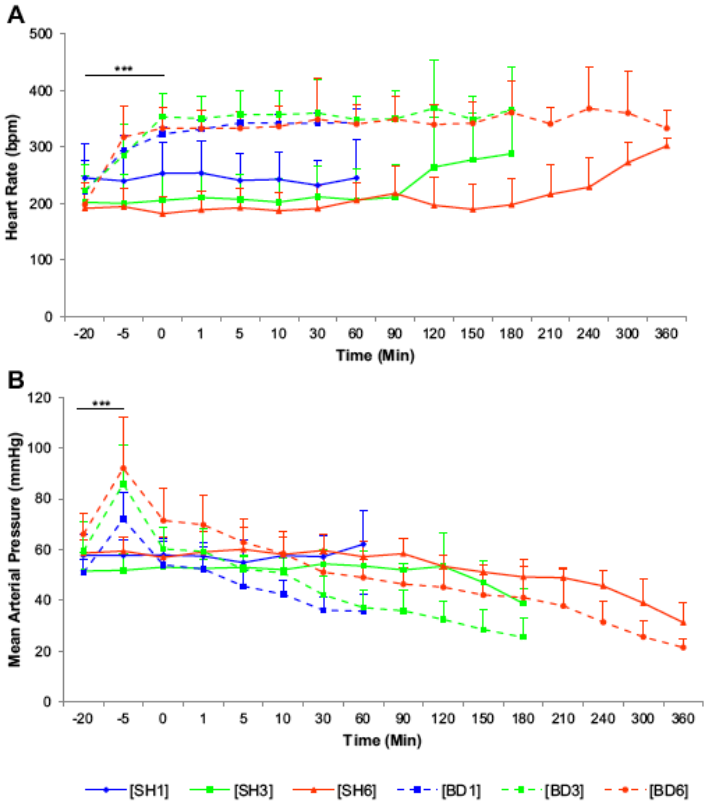


Fig. 2 – (A) Time course of heart rate in [BD1], [BD3], and [BD6] compared with [SH1], [SH3], and [SH6]. ***$P < 0.001$ for [BD1], [BD3], [BD6] at −20 min *versus* 0 min. (B) Time course of mean arterial pressure in [BD1], [BD3], and [BD6] compared with [SH1], [SH3], and [SH6]. ***$P < 0.001$ for [BD1], [BD3], [BD6] at −20 min *versus* −5 min. [SH1], [SH3], [SH6]: sham-operated animals after 1 h, 3 h, and 6 h follow-up; [BD1], [BD3], [BD6]: brain-dead groups with 1 h, 3 h, and 6 h follow-up after BD. The values represent the mean ± SD of observations made at each time point. Time point of balloon inflation in BD groups at −20 min. Time point of 0 min reflects diagnosis of brain death.

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# Evaluating lung injury at increasing time intervals in a murine brain death model

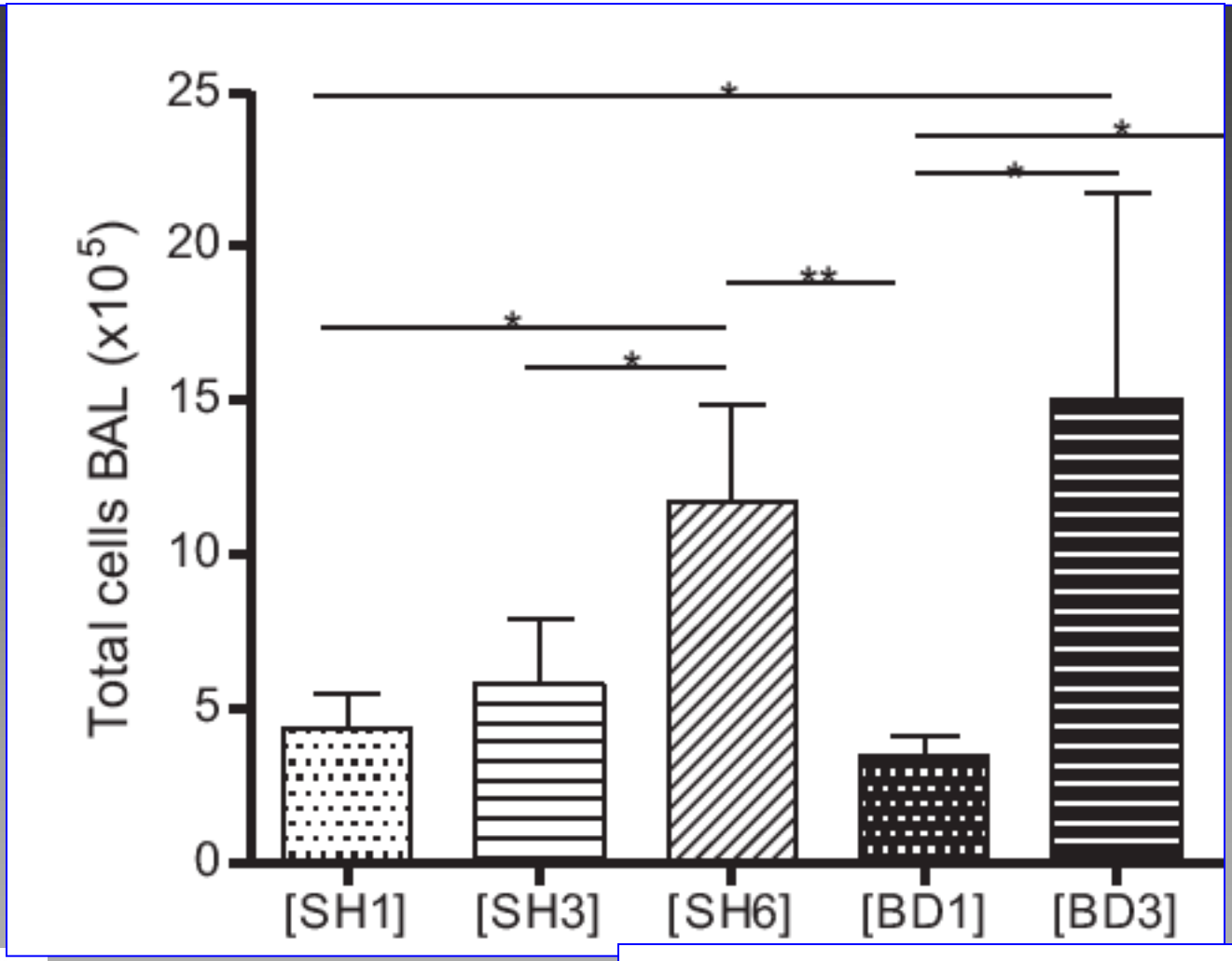



**Fig. 3 – Total cell count in BAL.**

# Evaluating lung injury at increasing time intervals in a murine brain death model

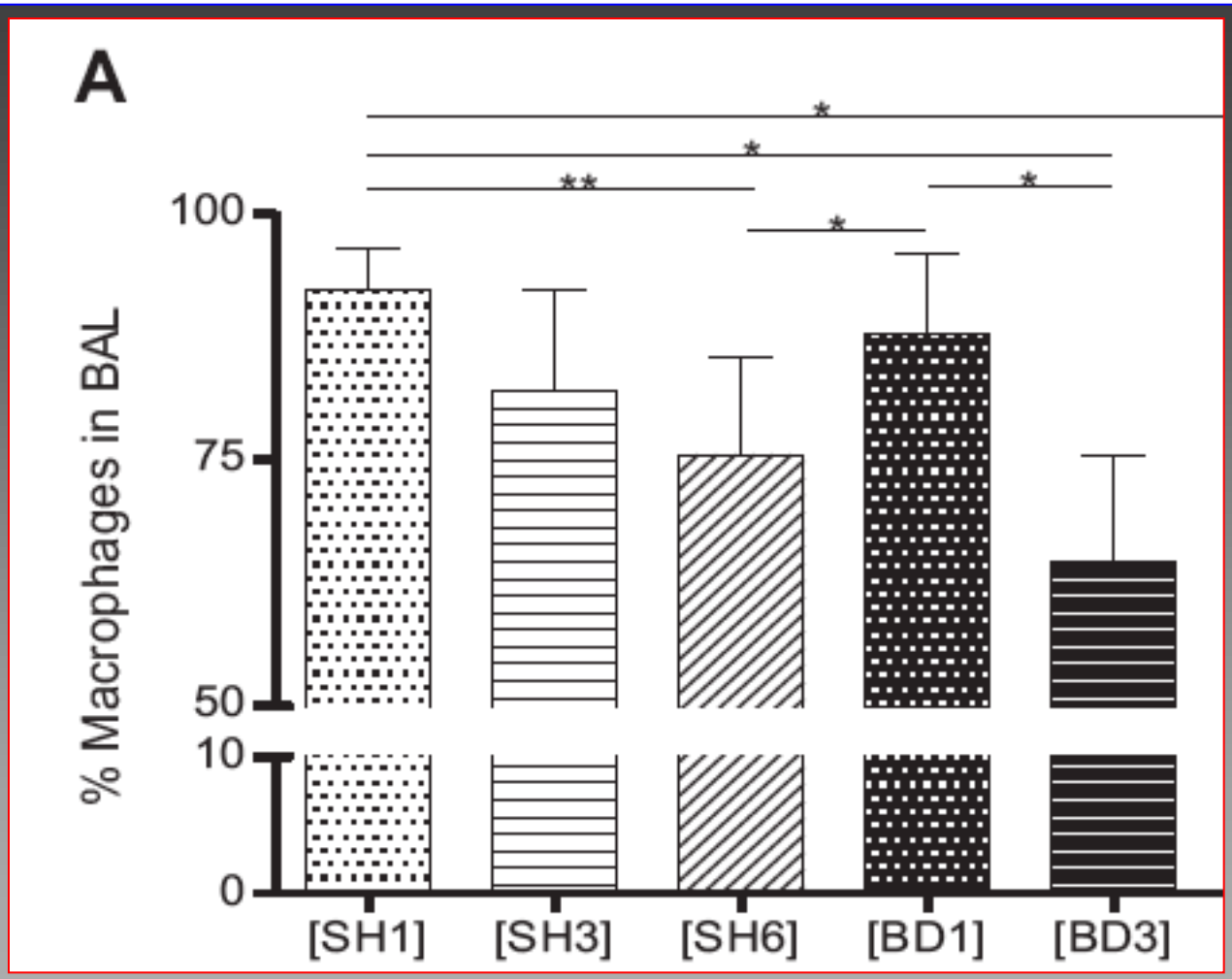



**Fig. 3 – Total cell count in BAL.**

# Evaluating lung injury at increasing time intervals in a murine brain death model

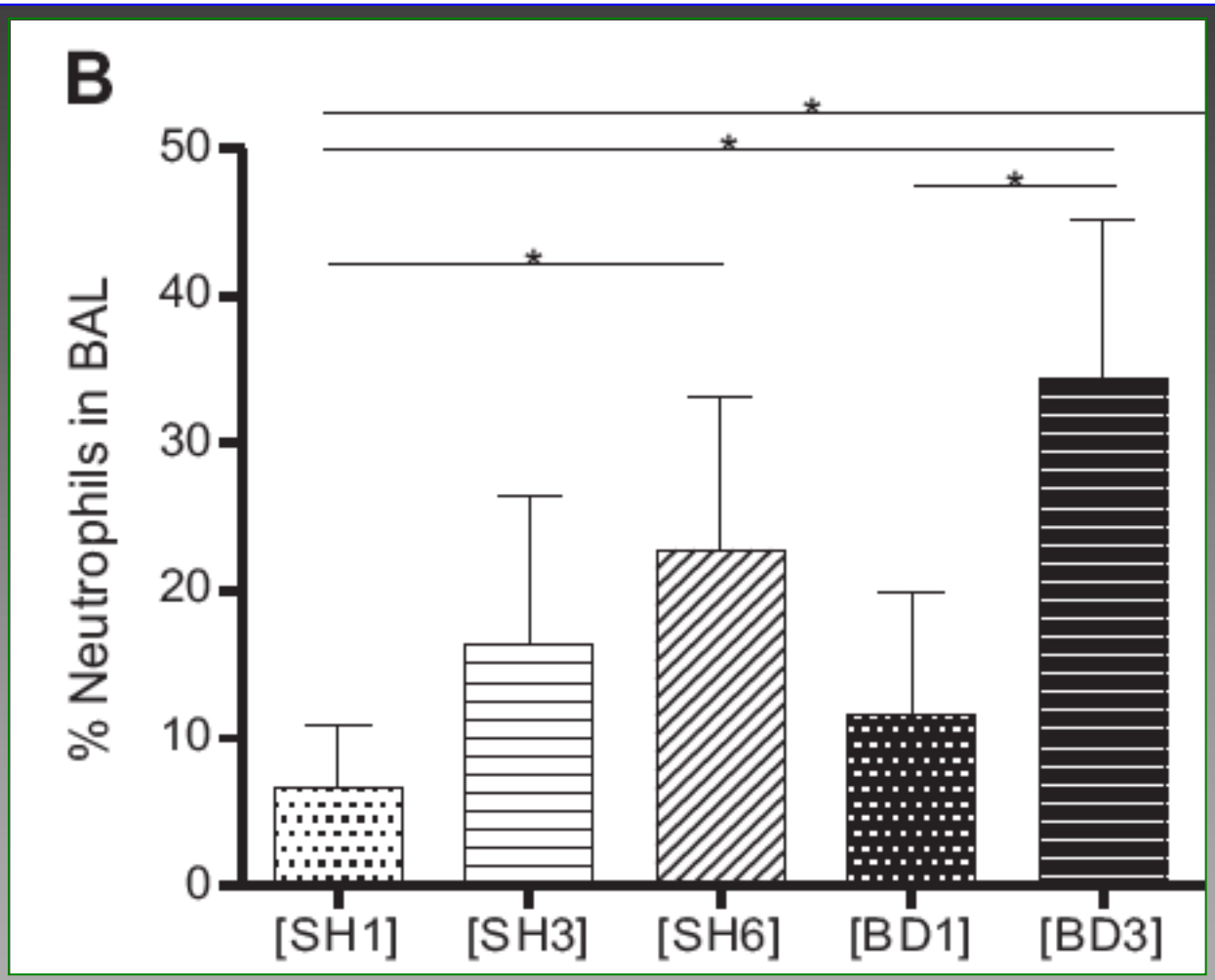
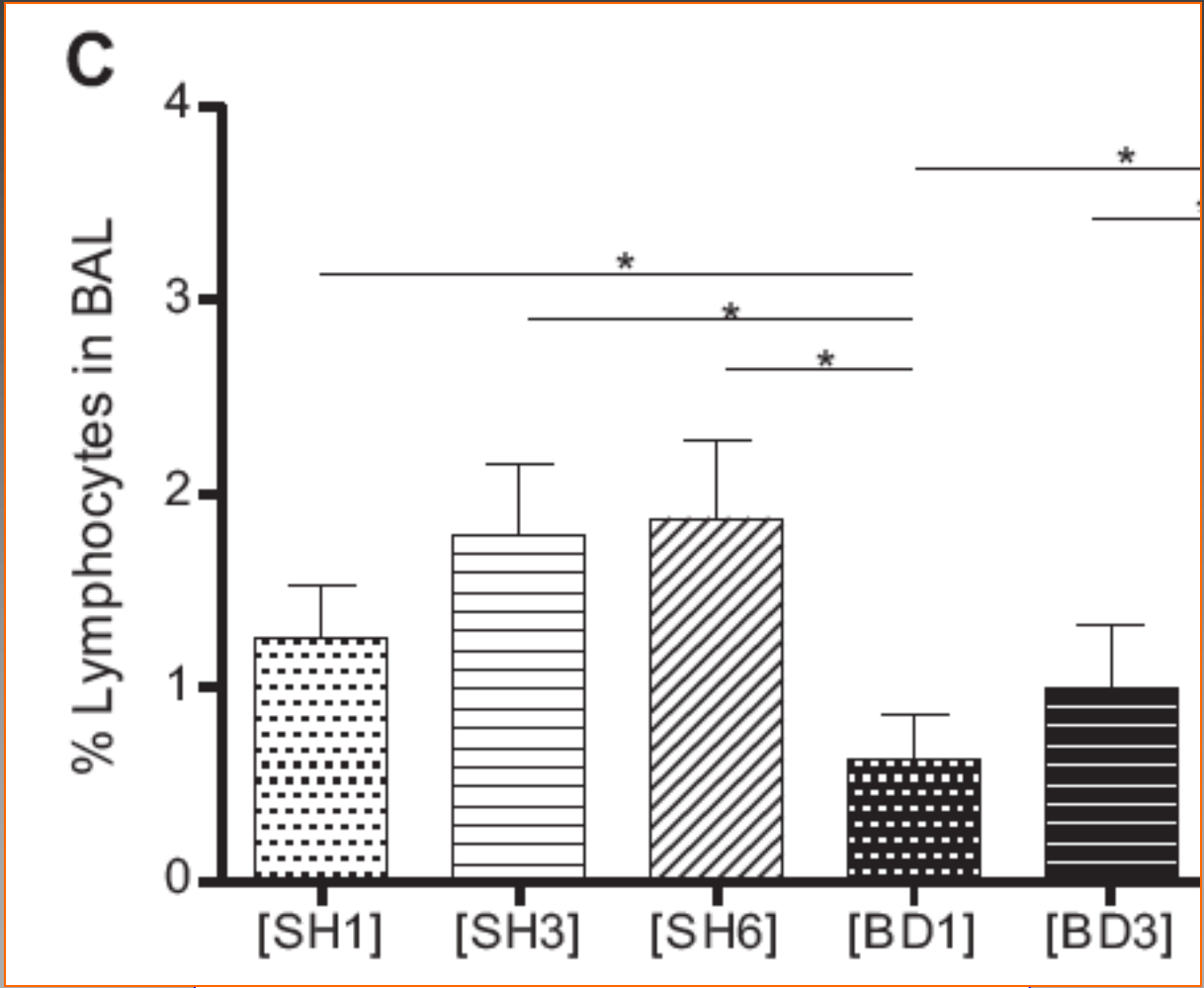



**Fig. 3 – Total cell count in BAL.**

# Evaluating lung injury at increasing time intervals in a murine brain death model

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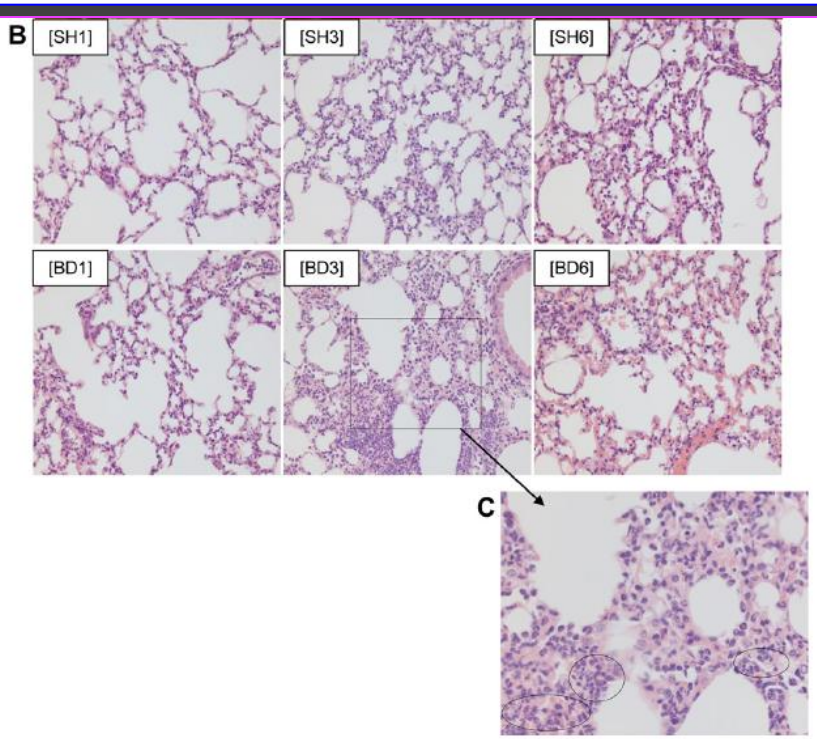


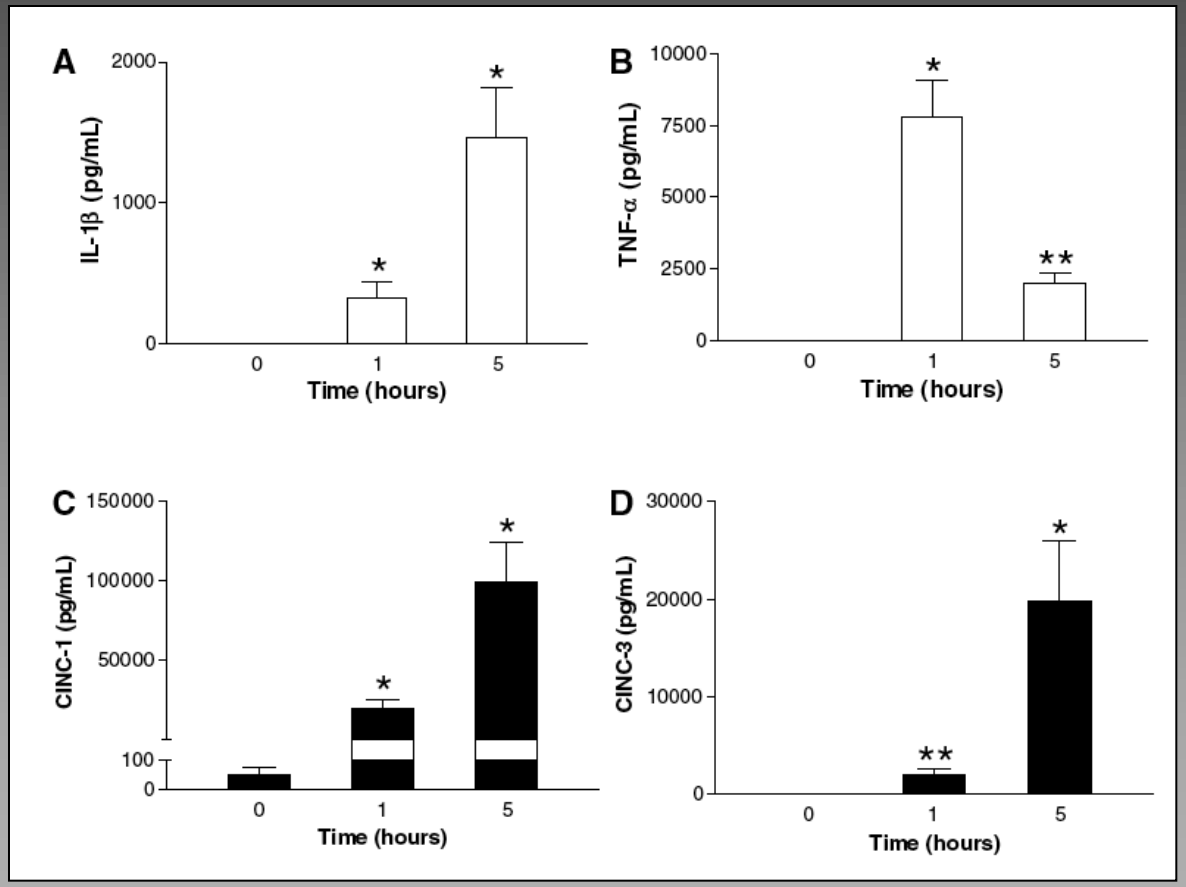

Fig. 6 – (A) Macroscopic images of sham-operated ([SH1], [SH3], [SH6]) and brain-dead animals ([BD1], [BD3], [BD6]) after 1 h, 3 h, and 6 h follow-up. (B) Hematoxylin-eosin-stained biopsies in [SH1], [SH3], [SH6], [BD1], [BD3], and [BD6]. Original magnification ×200. (C) Neutrophilic infiltrations (circles), original magnification ×400.

In conclusion, a 3-h follow-up period after brain death resulted in significant signs of inflammation and lung injury compared with sham-operated animals. The inflammation

# Early Hemodynamic Injury During Donor Brain Death Determines the Severity of Primary Graft Dysfunction after Lung Transplantation

American Journal of Transplantation 2007; 7: 83–90

V. S. Avlonitis[a,*]

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Valencia 2 de Diciembre de 2013

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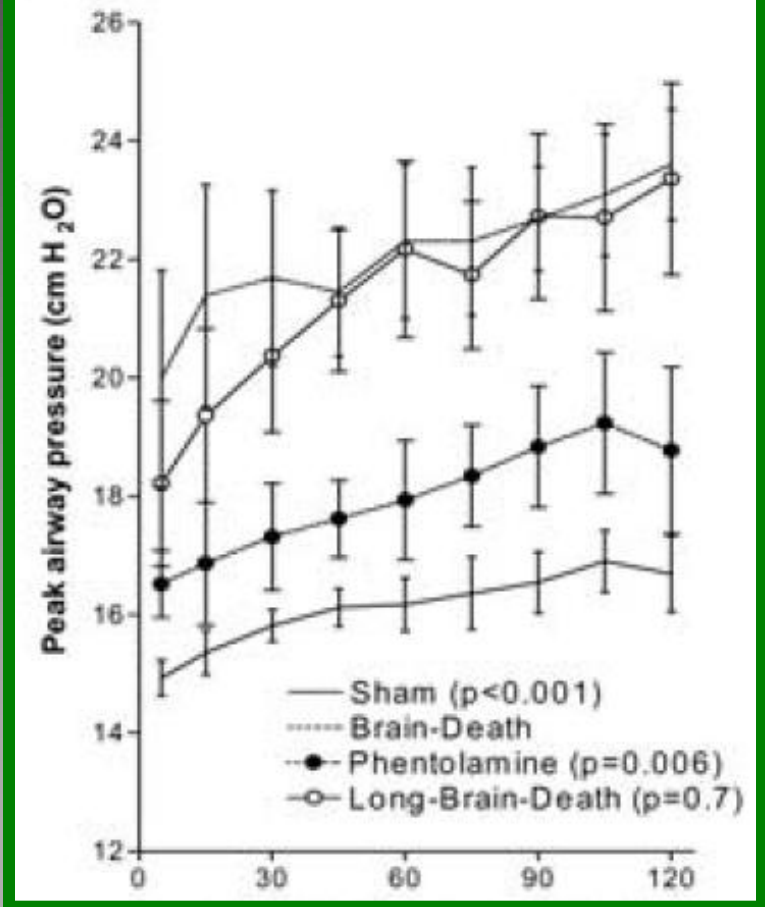


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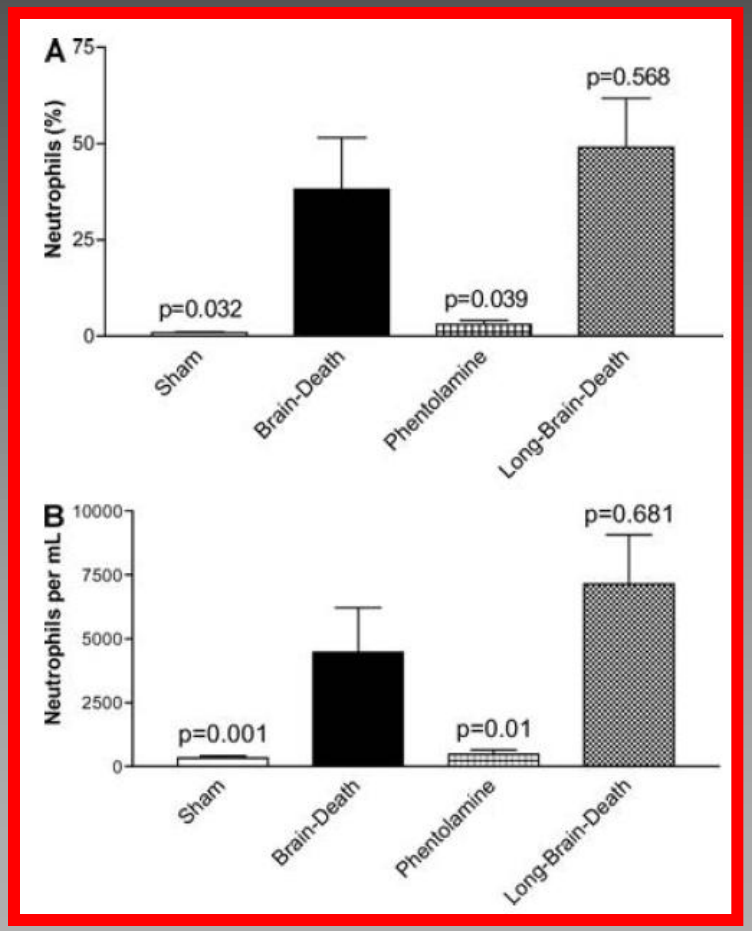


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# Prolonged time between donor brain death and organ retrieval results in an increased risk of mortality in cardiac transplant recipients☆

Sheila Ramjug*, Nehal Hussain, Nizar Yonan

Interactive CardioVascular and Thoracic Surgery 12 (2011) 938-942

INTERACTIVE CARDIOVASCULAR AND THORACIC SURGERY

One factor which is frequently overlooked, is the effect of brain death upon the donor heart. This is of particular importance in current times given the increasing use of marginal organs. This current study aims to examine the impact of the time of brain death to retrieval of the heart against long-term cardiac transplant recipient survival. By their nature marginal organs are at increased risk of organ dysfunction [2]. Combine this tendency with the detrimen-

Table 1. Donor and recipient characteristics

| Variables | |
|---|---|
| Donors $n=157$ | |
| Age (years) | |
| Mean ± S.D. | 35.7 ± 12.2 |
| Range | 13–61 |
| Male sex | 93 |
| Smoking history | 81 |
| CMV+ | 59 |
| CMV positive mismatch | 55 |
| CMV reverse mismatch (donor positive:recipient negative) | 32 |
| Recipients $n=157$ | |
| Age (years) | |
| Mean ± S.D. | 47.5 ± 12.3 |
| Range | 18–67 |
| Male sex | 123 |
| Female donor to male recipient | 39 |
| CMV+ | 81 |
| Rejection | 91 |
| Transplant related | |
| Brain death interval (h) | |
| Mean ± S.D. | 13.2 ± 3.96 |
| Range | 5.5–31.0 |
| Ischaemic time (h) | |
| Mean ± S.D. | 3.75 ± 0.80 |
| Range | 1.5–5.5 |

CMV, cytomegalovirus; S.D., standard deviation.

Table 2. Analysis of donor and recipients characteristics in relation to mortality

| Variables | Univariate analysis (*P*-value) | HR | Multivariate analysis (*P*-value) | HR |
|---|---|---|---|---|
| Donor | | | | |
| Age | 0.93 CI 0.98–1.03 | 1.00 | 0.99 CI 0.97–1.03 | 1.00 |
| Sex | 0.84 CI 0.56–2.06 | 1.07 | 0.17 CI 0.85–1.56 | 0.37 |
| Smoking history | 0.19 CI 0.80–2.99 | 1.55 | **0.08 CI 0.92–3.89** | **1.89** |
| CMV+ | 0.43 CI 0.68–2.48 | 1.30 | 0.54 CI 0.49–3.97 | 1.39 |
| CMV reverse mismatch | 0.31 CI 0.70–3.00 | 1.45 | 0.66 CI 0.32–5.94 | 1.38 |
| Recipients | | | | |
| Age | 0.64 CI 0.98–1.04 | 1.01 | 0.95 CI 0.97–1.03 | 1.00 |
| Sex | **0.06 CI 0.97–3.98** | **1.96** | **0.04 CI 1.08–12.35** | **3.64** |
| Female donor male recipient | 0.57 CI 0.38–1.71 | 0.80 | 0.33 CI 0.44–11.70 | 2.27 |
| CMV+ | 0.85 CI 0.56–2.03 | 1.06 | 0.83 CI 0.33–2.48 | 0.90 |
| Rejection | **0.08 CI 0.29–1.07** | **0.56** | 0.15 CI 0.29–1.21 | 0.59 |
| Transplant related | | | | |
| Brain death interval | **<0.001 CI 1.08–1.24** | **1.16** | **<0.001 CI 1.06–1.24** | **1.15** |
| Ischaemic time | 0.47 CI 0.77–1.76 | 1.16 | 0.48 CI 0.75–1.86 | 1.18 |

Bold values relate to variables that are statistically significant at the 90% level or above. CMV, cytomegalovirus; HR, hazard ratio.

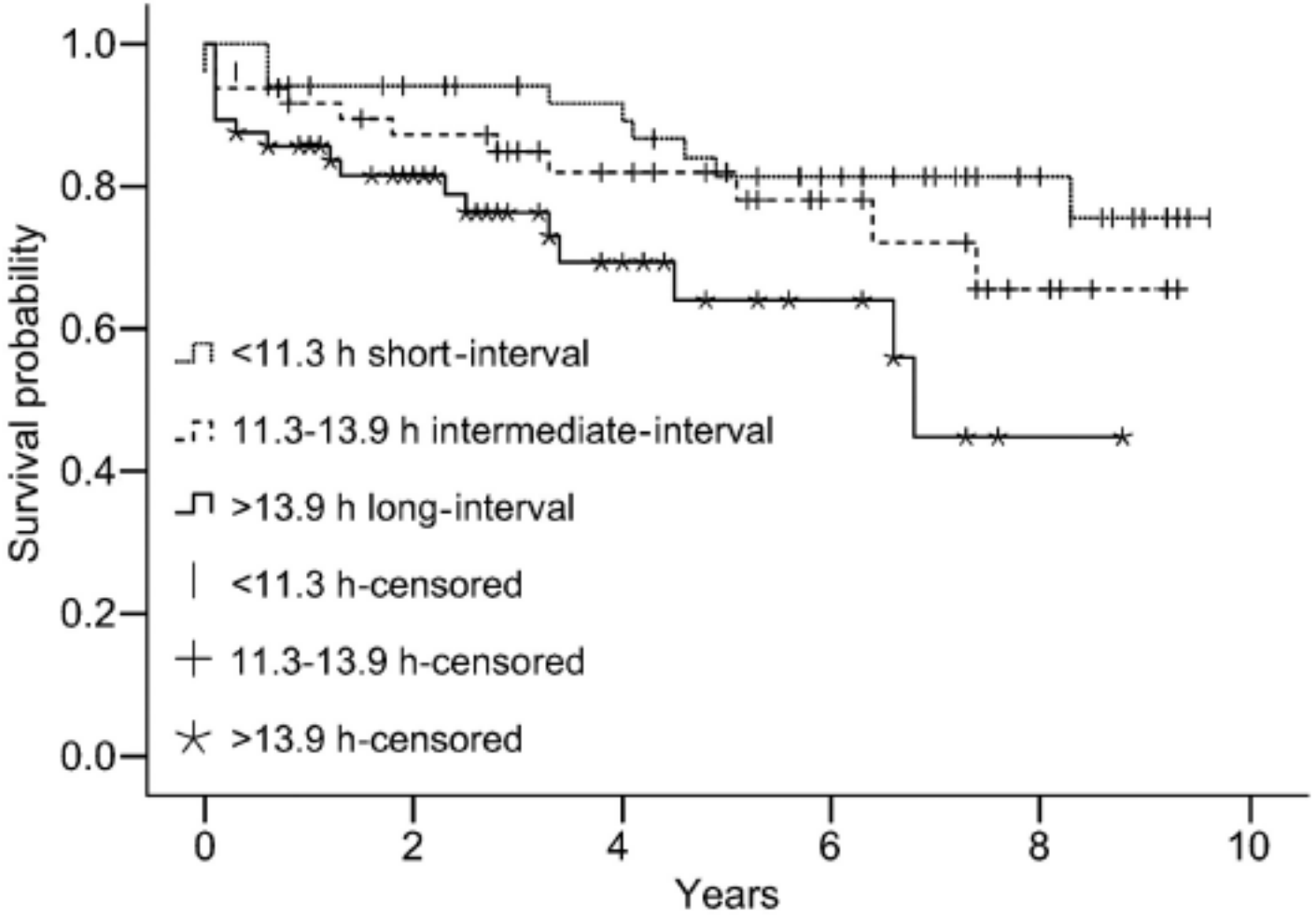


Fig. 2. Kaplan–Meier survival curve with stratified brain death interval time.

This period of sympathetic drive is associated with a significant increase of plasma catecholamines. Therefore, organs are exposed not only to direct sympathetic stimulation but also to circulating catecholamines. This inflammatory response is associated with the up-regulation of pro-inflammatory cytokines leading to deleterious effects on the heart as shown by Plenz et al. [12]. The period of normotension or hypotension is caused by a reduction in sympathetic flow. It is during this time where the inotropic and chrontropic state of the heart is impaired. Consequently, cardiac output falls affecting blood and oxygen delivery to tissues and organs.

Our findings suggest that donors should be managed promptly from the declaration of brain death, in order to reduce the mortality of cardiac transplant recipients.

# Advanced Hemodynamic Monitoring: Principles and Practice in Neurocritical Care

Christos Lazaridis

neurocritical care society
Neurocrit Care
2012;16:163-9

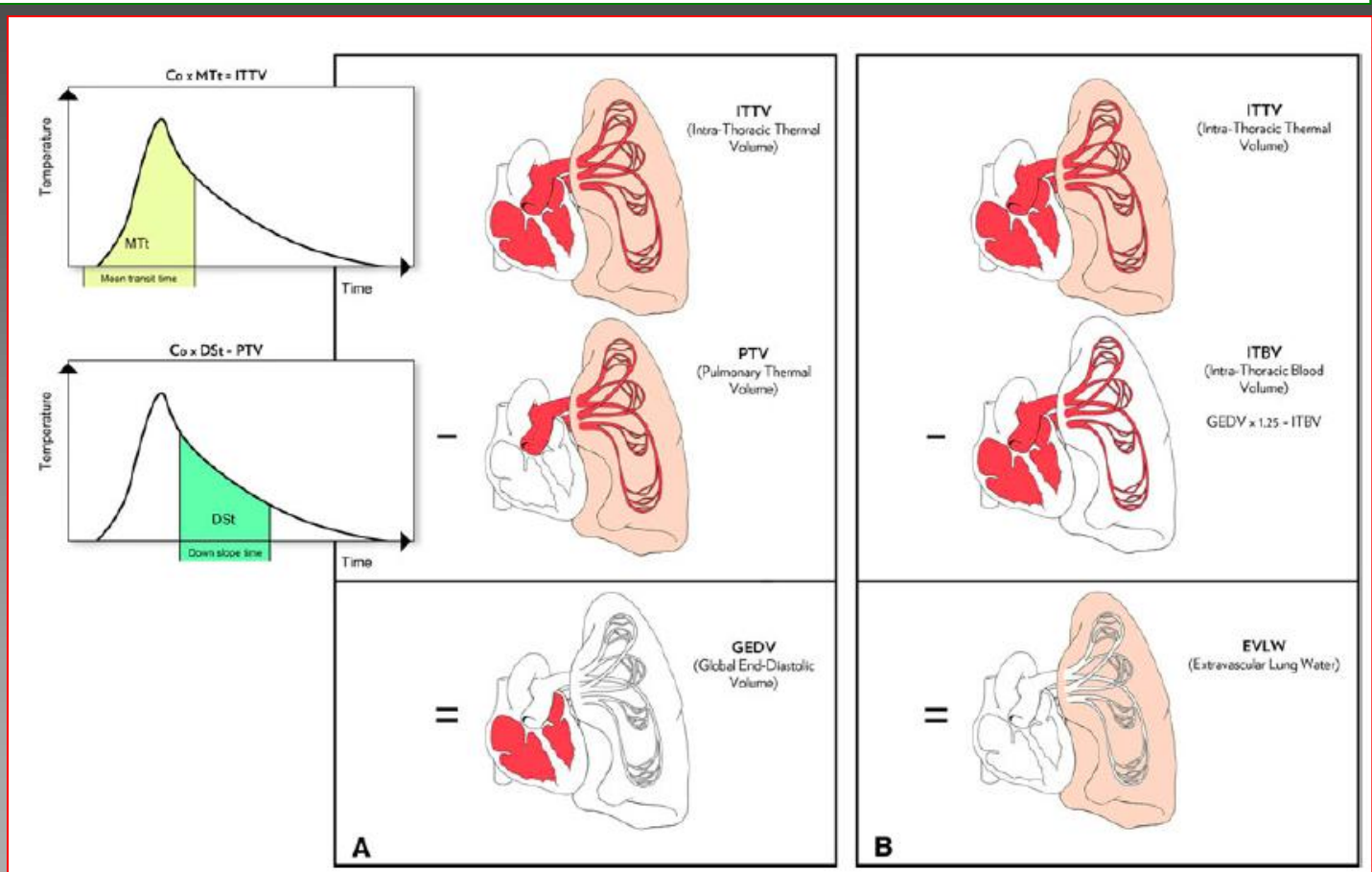


**Fig. 1** Transpulmonary thermodilution—calculation of filling volumes and extravascular lung water

# Advanced Hemodynamic Monitoring: Principles and Practice in Neurocritical Care

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neurocritical care society Neurocrit Care

2012;16:163-9

| General goals–normal ranges |
|---|
| LV performance |
| CI = 3.0–5.0 l/min/$m^2$ |
| CFI = 4.5–6.5 $min^{-1}$ (<3.2 predictive of EF <35%) |
| TTE = Visual inspection of LV/RV size and contractility |
| Preload |
| GEDI = 680–800 ml/$m^2$ |
| TTE = LV CSA obliteration indicates hyperdynamic-hypovolemic ventricle |
| Fluid responsiveness (responders) |
| SVV >10% |
| PPV >13% |
| dIVC >16% |
| Accurate in controlled MV settings, synchronous with the ventilator, caution with small TV, no afib/flutter. PLR if limitations |
| Pulmonary edema |
| ELWI = 3.0–10.0 ml/kg |
| Lung US = A-line predominance |

# The bispectral index, a useful adjunct for the timely diagnosis of brain death in the comatose trauma patient

C. Michael Dunham, M.D., D. Alisa Katradis, A.P.N., Mark D. Williams, M.D.*

The American Journal of Surgery (2009) 198, 846–851

The American Journal of Surgery®

27 pt

ever, BIS monitoring seems to simplify the task. BIS monitoring appears to be useful for indicating the appropriate point for performing a comprehensive neurologic examination, apnea assessment, or ancillary test. Timely brain death evaluations in brain-dead patients enhance patient and fam-

# Bispectral Index Monitoring for Early Detection of Brain Death

M. Misis, J. Gener Raxach, H. Perez Molto, S. Martinez Vega, and P. Sanchez Rico

*Transplantation Proceedings*, 40, 1279–1281 (2008)

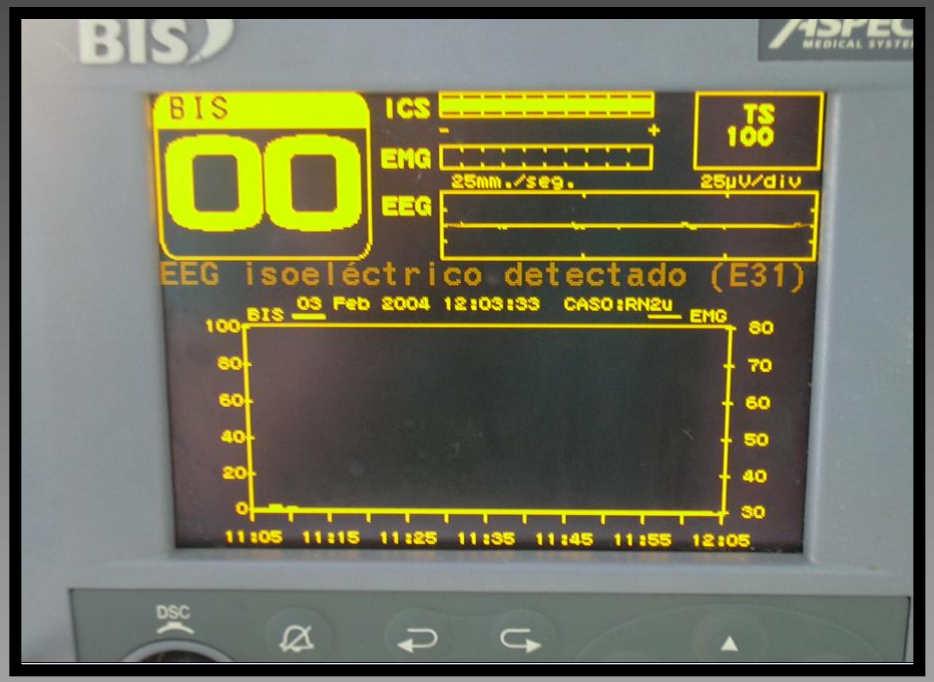
54 pt

In conclusion, BIS monitoring is a continuous, simple, easy to interpret method that helps in the clinical evaluation of BD. In our series, cerebral circulatory arrest (TCD) preceded BIS 0/SR 100 values. The BIS detected early cerebral arrest and BD.


# BRAIN DEATH ASSESMENT USING BISPECTRAL INDEX.

R BADENES, ML GARCIA-PEREZ, J CARRERA, A MARUENDA, V CHISBERT,G AGUILAR, FJ BELDA
Department of Anesthesiology and Intensive Care.
Hospital Clinico Universitario. Valencia. Spain.

2004;30:S55




AUDITORY EVOKED POTENTIALS MONITOR AND BRAIN DEATH .

R BADENES, A MARUENDA, ML GARCIA-PEREZ, V CHISBERT, FJ BELDA
Department of Anesthesiology and Intensive Care.
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EJA
European Journal of Anaesthesiology

2005;22:150

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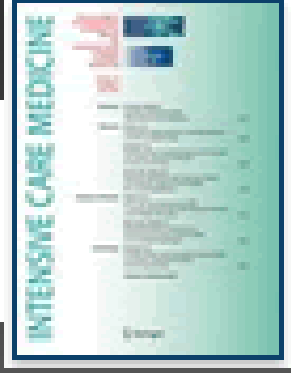
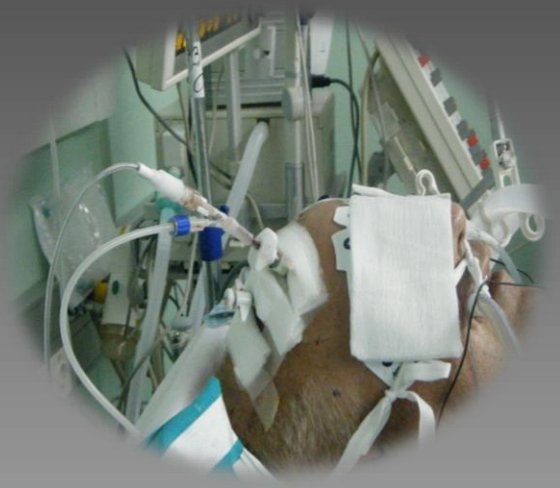


# CEREBRAL BLOOD FLOW BY THERMAL DIFFUSION DURING ASSESSMENT OF BRAIN DEATH

R. Badenes Quiles[1], J. Moreno[1], M.L. Garcia-Pérez[1], E. Pastor[1], A. Maruenda[1], F.J. Belda[1]
[1]Hospital Clinico Universitario Valencia, Anesthesiology and Critical Care, Valencia, Spain

2011;37.S252

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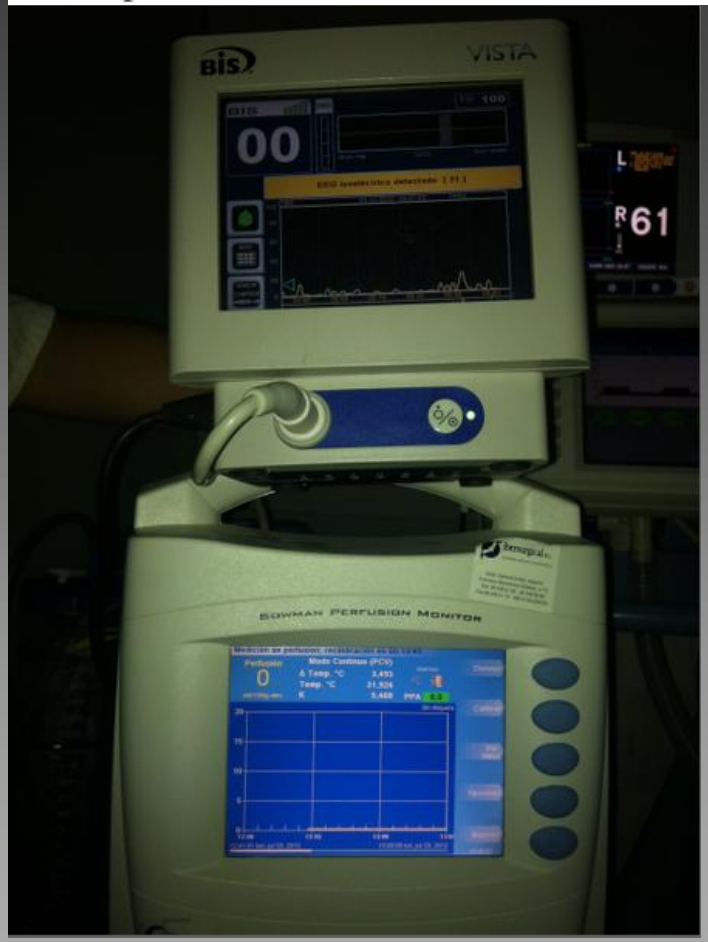


2011;37.S252



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Valencia 2 de Diciembre de 2013

**XXX Congreso Nacional**
**XIV Hispano-Luso**
de la Sociedad Española de Anestesiología, Reanimación y Terapéutica del dolor

SEDAR 2011 MADRID del 5 al 7 de mayo

El XXX Congreso Nacional y XIV Hispano-Luso
de la Sociedad Española de Anestesiologia, Reanimación y Terapéutica del Dolor

una vez evaluada la calidad científica de las Comunicaciones Orales presentadas, tiene a bien otorgar el

PREMIO DEL COMITÉ ORGANIZADOR A LA MEJOR COMUNICACIÓN ORAL

Flujo sanguíneo cerebral por difusión térmica en traumatismos craneoencefálicos graves.

MJ MONTERO HERNANDEZ; DE FEZ M.; MARUENDA PAULINO A.; GARCIA PEREZ ML.; BODENTES QUILES R.
BELDA NACHER FJ; Hospital Clínico Universitario, Valencia.

En Madrid a 7 de Mayo de 2011

Profesor F. Gilsanz Rodríguez
Presidente de la SEDAR

Dr. A. Planas Roca
Presidente del Comité Organizador

CONSORCIO HOSPITAL GENERAL UNIVERSITARIO DE VALENCIA
SERVICIO DE ANESTESIOLOGIA, REANIMACION Y TERAPEUTICA DEL DOLOR
SARTD

**SARTD-CHGUV Sesión de Formación Continuada**
**Valencia 2 de Diciembre de 2013**

1. Organ Crosstalk.
   Brain Heart Connection
   Brain Lung Connection

2. Brain Death Diagnosis.

3. Brain Death Time

4. Neuromonitoring