Kalp Cerrahisinde Serebral Rejional Oksijen Saturasyonu Takibinin Yeri

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Acıbadem Üniversitesi Anesteziyoloji ve Reanimasyon ABD

ACIBADEM ÜNİVERSİTESİ

GKDA 2011 ANTAKYA

Non fizyolojik dolaşım (CPB)

- Açık kalp cerrahisinde;
  - Anestezi
  - Cerrahi
  - Organ koruma yöntemlerindeki gelişmeler,

- Yaşa ve eşlik eden hastalıklara bağlı mortalite oranlarının azalmasını sağlarken

- Postoperatif kognitif fonksiyon bozukluğu (POCD) oranında anlamlı bir düzelme sağlayamamıştır.

- Newman MF. N Engl J Med 344:395-402, 2001
- McKenzie ED. Am J Surg 190:289-294, 2005

POCD Ety;

Multi faktoriyel olduğu;

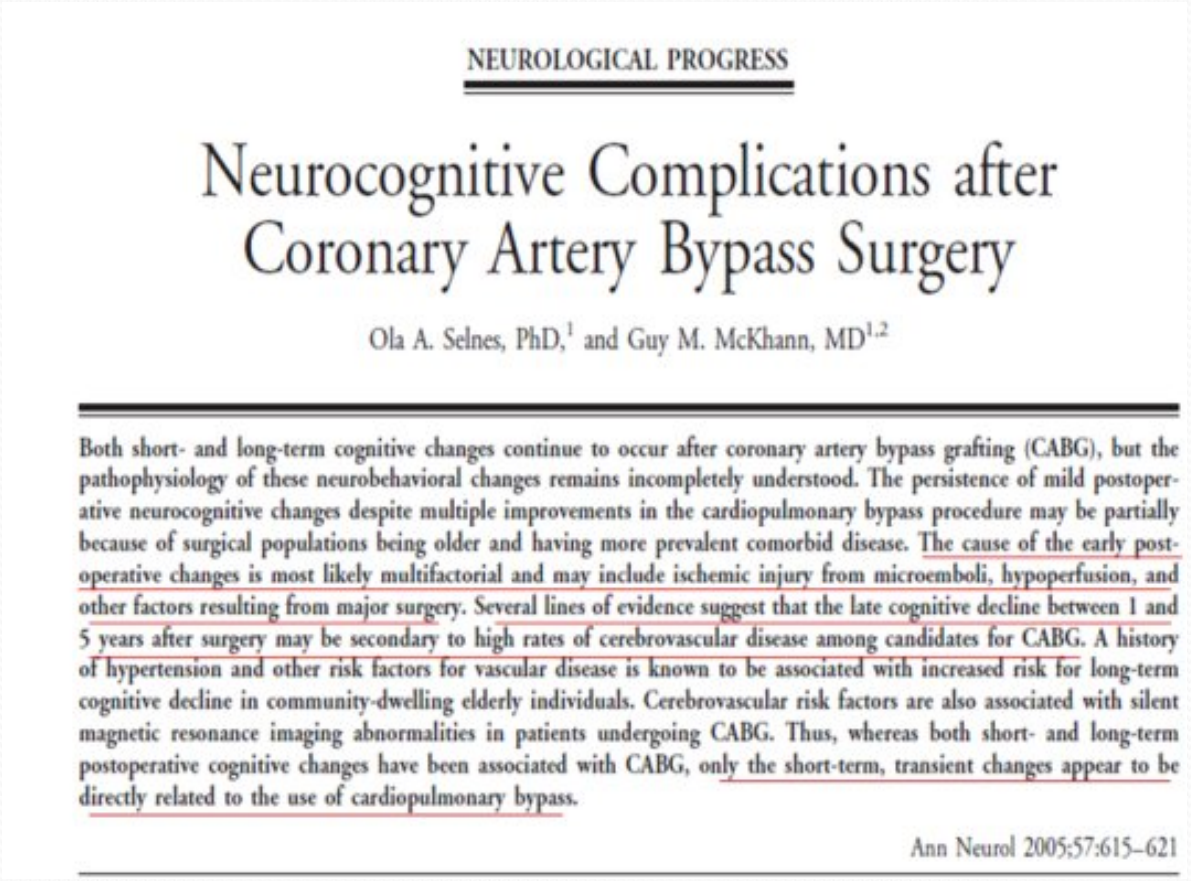
- Emboli
- Hipoperfüzyon
- Diğer nedenler
  - İnflamatuvar
  - Toksik

Geç POCD en önemli nedeninin,

- Mevcut SVH olduğu

Erken dönem POCD en önemli nedeninin,

- CPB kullanımı



NEUROLOGICAL PROGRESS

Neurocognitive Complications after Coronary Artery Bypass Surgery

Ola A. Selnes, PhD,[1] and Guy M. McKhann, MD[1,2]

Both short- and long-term cognitive changes continue to occur after coronary artery bypass grafting (CABG), but the pathophysiology of these neurobehavioral changes remains incompletely understood. The persistence of mild postoperative neurocognitive changes despite multiple improvements in the cardiopulmonary bypass procedure may be partially because of surgical populations being older and having more prevalent comorbid disease. The cause of the early postoperative changes is most likely multifactorial and may include ischemic injury from microemboli, hypoperfusion, and other factors resulting from major surgery. Several lines of evidence suggest that the late cognitive decline between 1 and 5 years after surgery may be secondary to high rates of cerebrovascular disease among candidates for CABG. A history of hypertension and other risk factors for vascular disease is known to be associated with increased risk for long-term cognitive decline in community-dwelling elderly individuals. Cerebrovascular risk factors are also associated with silent magnetic resonance imaging abnormalities in patients undergoing CABG. Thus, whereas both short- and long-term postoperative cognitive changes have been associated with CABG, only the short-term, transient changes appear to be directly related to the use of cardiopulmonary bypass.

Ann Neurol 2005;57:615–621

CABG op.sonrası SSS fonks. Boz.

- Nörolojik fonks. boz.

  Hemipleji/inme

  %2-3

  Yaş>70 de oralar daha da artmakta

- Nöropsikolojik fonks.boz.

  İnsidansın %35-83

Neuropsychologic Dysfunction After Cardiac Surgery: What Is the Problem?

Ravi Gill, BM, FRCA (UK), and John M. Murkin, MD, FRCPC

Twenty years ago Åberg published his seminal studies on the neuropsychologic consequences of cardiopulmonary bypass (CPB). Twenty years later, what is the state of current research on the problem of post-CPB neurologic injury, and what different management techniques have been employed to influence this outcome? This article reviews the definition and assessment of postoperative neuropsychologic dysfunction; epidemiologic data and associated risk factors assessing post-CPB neuropsychologic dysfunction are critically appraised.

KEY WORDS: cardiac surgery, neuropsychologic dysfunction, central nervous system

THE SUCCESSFUL application of cardiopulmonary bypass (CPB) in 1954 by Gibbon[1] led to rapid advances in cardiac surgery, anesthesia, and perfusion techniques. Today, valve replacement and coronary artery bypass (CAB) surgery are usually successful in improving symptoms from cardiac disease. Mortality directly attributable to primary cardiac failure after CPB is acceptable (0.5% to 5%)[2] despite an aging population with significant comorbid disease presenting for surgery.[3] However, the incidence of death caused by neurologic deficits has increased from 7.2% to 19.6%, as overall mortality declines.[4] Therefore, even if cardiac function is improved by surgery, the overall success of an operation may be masked by neurologic or neuropsychologic dysfunction after surgery.

Central nervous system (CNS) dysfunction after cardiac surgery can be divided functionally, ie, the method by which it is detected, into two types, either neurologic dysfunction or neuropsychologic dysfunction. Overt neurologic dysfunction, ie, hemiplegia or stroke, has an overall incidence of 2% to 3%, although age-related risk of CNS damage increases exponentially after age 70.[5] This appears to be largely secondary to cerebral macroemboli arising as a result of aortic atherosclerosis. The incidence of postoperative neuropsychologic dysfunction varies between 33% to 83%[6-9] and has been shown to persist in up to 35% of patients for at least 12 months.[10] The etiology of neuropsychologic dysfunction after CPB remains unresolved and is probably multifactorial resulting from the interactions of both intraoperative microemboli and cerebral hypoperfusion, as well as premorbid disease and postoperative events. ments may all play a part in the deterioration that is observed in patients independent of exposure to CPB.[14] A surprisingly high incidence of neuropsychologic deficits have been demonstrated in patients before CPB and major surgery and may be the result of age, chronic cardiac, and medical diseases,[6,12] and a possible genetic predisposition to intellectual deterioration.[15] Therefore, cognitive dysfunction after CPB could be an epiphenomenon within a global picture of intellectual deterioration that can be demonstrated in elderly patients with multiple medical problems who are then stressed by hospital admission and surgery.

In this review, the constraints and limitations that hamper attempts to assess cognitive performance in cardiac surgical patients are assessed. Factors influencing the selection of a specific battery of neuropsychologic tests are reviewed in order to suggest an appropriate methodology for studies in this field. Finally, the current literature is reviewed in order to ascertain the extent of neuropsychologic dysfunction after CPB and the factors that are associated with it.

NEUROPSYCHOLOGIC ASSESSMENT

Test Selection

Neuropsychologic assessment is frequently used in clinical practice to examine whether a patient with a known cerebral lesion exhibits cognitive dysfunction, and it allows a pattern of cognitive and adaptive abilities to be established.[16] This type of assessment may require several hours and uses an exhaustive battery of tests, generally between 25 and 35 in number, that has been extensively validated by

Yaş ↔stroke ve mortalite

British Journal of Anaesthesia **84** (3): 378–93 (2000)

BJA

REVIEW ARTICLE

Central nervous system complications of cardiac surgery†

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Br J Anaesth 2000; **84**: 378–93

Keywords: surgery, cardiovascular; surgery, postoperative period; heart, cardiopulmonary bypass; brain, function; psychological responses, postoperative; complications, neurological; complications, stroke

While the number of patients undergoing surgery for valvular and other types of heart disease has remained fairly constant, the number undergoing coronary revascularization procedures is increasing (Fig. 1). Because of many technological advances over the past four decades, there has been a steady decrease in the mortality and morbidity associated with these procedures. Nevertheless, neurological injury remains an important cause of postoperative morbidity[91] and is responsible for an increasing

Adverse neurological outcome from cardiac surgery is the result of damage to the brain, spinal cord and/or peripheral nerves. CNS injury ranges in severity from subtle changes in personality, behaviour and cognitive function to fatal brain injury—the 'cerebral catastrophe' (Table 1). A major neurological complication after otherwise successful surgery represents a devastating outcome for patient and their family. The social and economic impact is enormous.

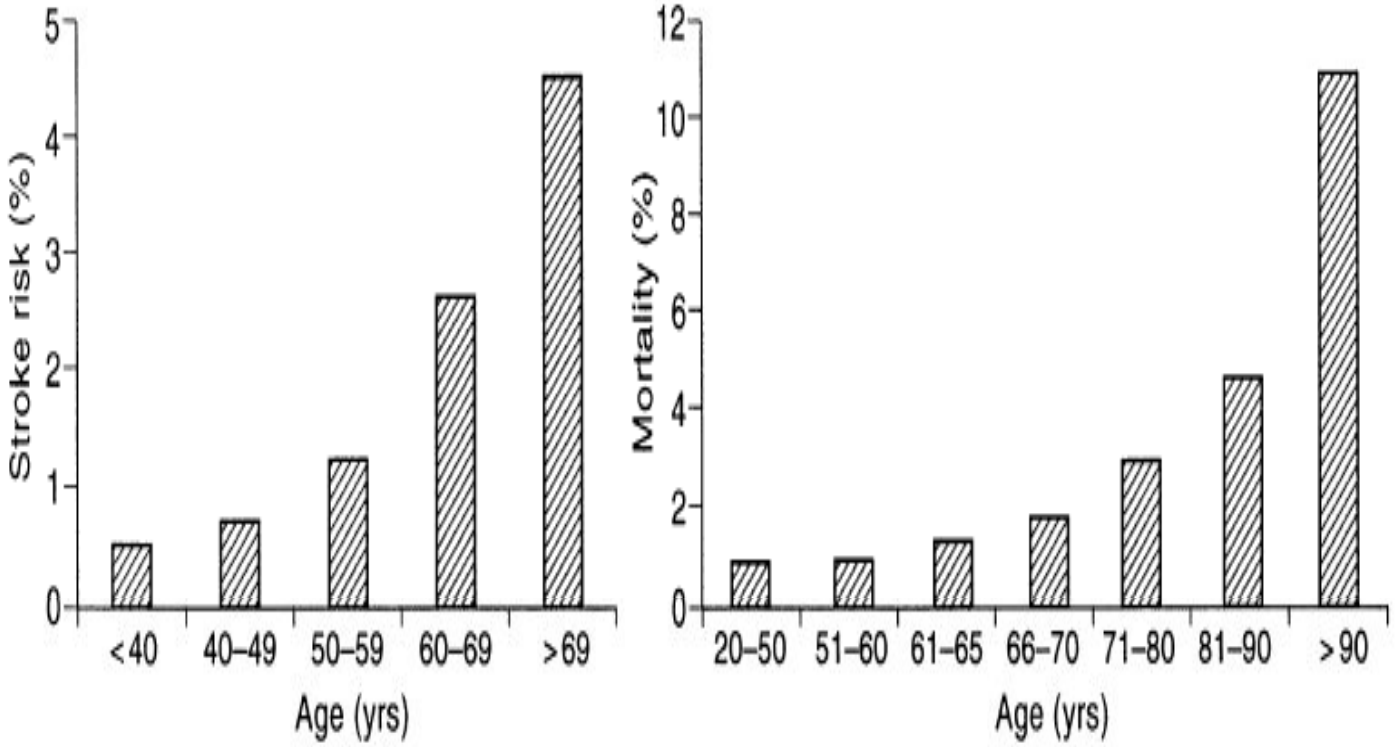

Fig 3 Effect of age on the risk of stroke (left) and mortality (right) after coronary artery bypass surgery. Reproduced with permission from Cosgrove and colleagues[32] and the US Society of Thoracic Surgeons (http://www.sts.org/graphics/sts/db/us98/gchart63.gif), respectively.

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Brain Research Reviews 50 (2005) 266 – 274

BRAIN RESEARCH REVIEWS

www.elsevier.com/locate/brainresrev

Review

Neural correlates of cognitive dysfunction after cardiac surgery

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Abstract

Patients who underwent cardiac surgery and their relatives often complain on postoperative memory impairment. Most prospective neuropsychological studies also found postoperative cognitive decline early after surgery. Nevertheless, recently several reports questioned the existence of long-term brain alterations in these patient cohorts. The present review was aimed to clear up the true cardiac surgery effects on brain and cognitive functions. The reviewed data evidence that cardiac surgery interventions induce persistent localized brain ischemic lesions along with rapidly reversing global brain swelling and decreased metabolism. A range of studies showed that left temporal region was especially prone to perioperative ischemic injury, and these findings might explain persistent verbal short-term memory decline in a considerable proportion of cardiac surgery patient cohorts. Speed/time of cognitive performance is commonly decreased early after on-pump surgery either. Nevertheless, no association between psychomotor speed slowing and intraoperative embolic load was found. The rapid recovery of the latter cognitive domain might be better explained by surgery related acute global brain metabolism changes rather than ischemic injury effects. Hence, analyses of performance on separate cognitive tests rather than summarized cognitive indexes are strongly recommended for future neuropsychological studies of cardiac surgery outcomes.

1. Introduction

Most cardiac surgery patients demonstrate mild cognitive impairment at discharge [3,42,70], and a considerable proportion of them (7–69% according to different criteria) do not recover in 1–3 months after surgery. Recently, several reports questioned the existence of long-term cognitive deterioration after cardiac surgery [38,54], whereas other research groups observed surgery related decline of cognitive functions in 5 years after on-pump [43,60]. Importantly, Stygall and colleagues [60] reported the association between cognitive decline in 5 years after coronary artery bypass grafting (CABG) and the number of intraoperative microemboli.

Table 1
Postoperative brain alterations in cardiac surgery

Brain alterations	Time of postsurgery	Recovery	Proportion of deteriorated patients (*n*)	Method	References
Brain swelling	1–3 days	7th day	71% (7), GM (10–35)	Diffusion-weighted MRI FLAIR MRI	Harris et al.; Anderson et al.; Bendszus et al. ([2,5,21])
Global and regional brain metabolism changes					
Decreased NAA/Cr ration	3rd day	10–14 days	GM (35)	MRS	Bendszus et al. [5]
Decreased global cerebral glucose metabolism	8–12 days	–	GM (18)	PET	Jacobs et al. [23]
Regional bilateral glucose metabolism changes	8–12 days	–	GM (18)	PET	Jacobs et al. [23]
Global cerebral blood flow increase	6th day	–	GM (12)	CASL-P-MRI	Floyd et al. [18]
New ischemic lesions	4–5 day	–	31–45% (13–35)	Diffusion-weighted MRI	Bendszus et al.; Restrepo et al.; Knipp et al. ([5,28,49])
	2–6 weeks	–	18–21% (27–38)	T2-weighted MRI	Vanninen et al.; Sylivris e al. ([63,72])
Brain perfusion decrease in bilateral occipital and cerebellar lobes and in left temporal lobe	3rd day	–	GM (30)	SPECT	Lee et al. [30]
Benzodiazepine receptor density decrease in bilateral frontal and left temporal lobe	3 months	–	GM (15)	SPECT	Rasmussen et al. [46]
Relative beta activity power increase	7th day	2 months	GM (62)	EEG	Toner et al. [68]
Total power decrease most prominent in alpha/beta bands, slowing of the total mean frequency (1.46–20.02 Hz)	2–3 months	–	GM (38–62)	EEG	Toner et al.; Vanninen et al. ([68,72])
Decreased amplitude of P300 component of auditory cognitive EP	7th day	2 months	GM (61)	Evoked EEG potentials	Toner et al. [67]
Prolonged latency of P300 component of auditory cognitive EP	7th day	2–4 months	GM (61)	Evoked EEG potentials	Toner et al. [67]
			GM (30–308)		Kilo et al. [26]
	4 months	–	GM (30)		Zimpfer et al. [79]

Abbreviations: *n*, number of patients in studied cohort; GM, significant difference in group means at baseline and postoperative follow-up; MRI, magnetic resonance imaging; MRS, magnetic resonance spectroscopy; NAA/Cr ratio, *N*-acetylaspartate-creatine ratio; FLAIR MRI, fluid attenuated inversion recovery magnetic resonance imaging; PET, positron emission tomography; SPECT, single photon emission computer tomography; CASL-P-MRI, continuous arterial spin labeling perfusion MRI; EEG, electroencephalography; EP, evoked potentials.

- Birçok klinisyen;
  - POCD ciddiye almaz
    - Çünkü;
      - Hastaların çoğunda bunun geçici olduğuna inanır.

- Gerçek durum böyle mi?

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LONGITUDINAL ASSESSMENT OF NEUROCOGNITIVE FUNCTION AFTER CORONARY-ARTERY BYPASS SURGERY

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ABSTRACT

Background Cognitive decline complicates early recovery after coronary-artery bypass grafting (CABG) and may be evident in as many as three quarters of patients at the time of discharge from the hospital and a third of patients after six months. We sought to determine the course of cognitive change during the five years after CABG and the effect of perioperative decline on long-term cognitive function.

Methods In 261 patients who underwent CABG, neurocognitive tests were performed preoperatively (at base line), before discharge, and six weeks, six months, and five years after CABG surgery. Decline

COGNITIVE decline has increasingly been recognized as a complication after cardiac surgery. Although important advances in techniques for perioperative anesthesia, surgery, and the protection of organs have resulted in substantial reductions in age-adjusted and risk-adjusted mortality,[1] the incidence of cognitive decline has changed little over the past 15 years. Elderly patients with multiple health problems, who are at higher risk than other groups of patients for neurologic and neurocognitive problems, are now able to undergo surgical procedures relatively late in life without serious

Methods In 261 patients who underwent CABG, neurocognitive tests were performed preoperatively (at base line), before discharge, and six weeks, six months, and five years after CABG surgery. Decline in postoperative function was defined as a drop of 1 SD or more in the scores on tests of any one of four domains of cognitive function. (A reduction of 1 SD represents a decline in function of approximately 20 percent.) Overall neurocognitive status was assessed with a composite cognitive index score representing the sum of the scores for the individual domains. Factors predicting long-term cognitive decline were determined by multivariable logistic and linear regression.

Results Among the patients studied, the incidence of cognitive decline was 53 percent at discharge, 36 percent at six weeks, 24 percent at six months, and 42 percent at five years. We investigated predictors of cognitive decline at five years and found that cognitive function at discharge was a significant predictor of long-term function ($P<0.001$).

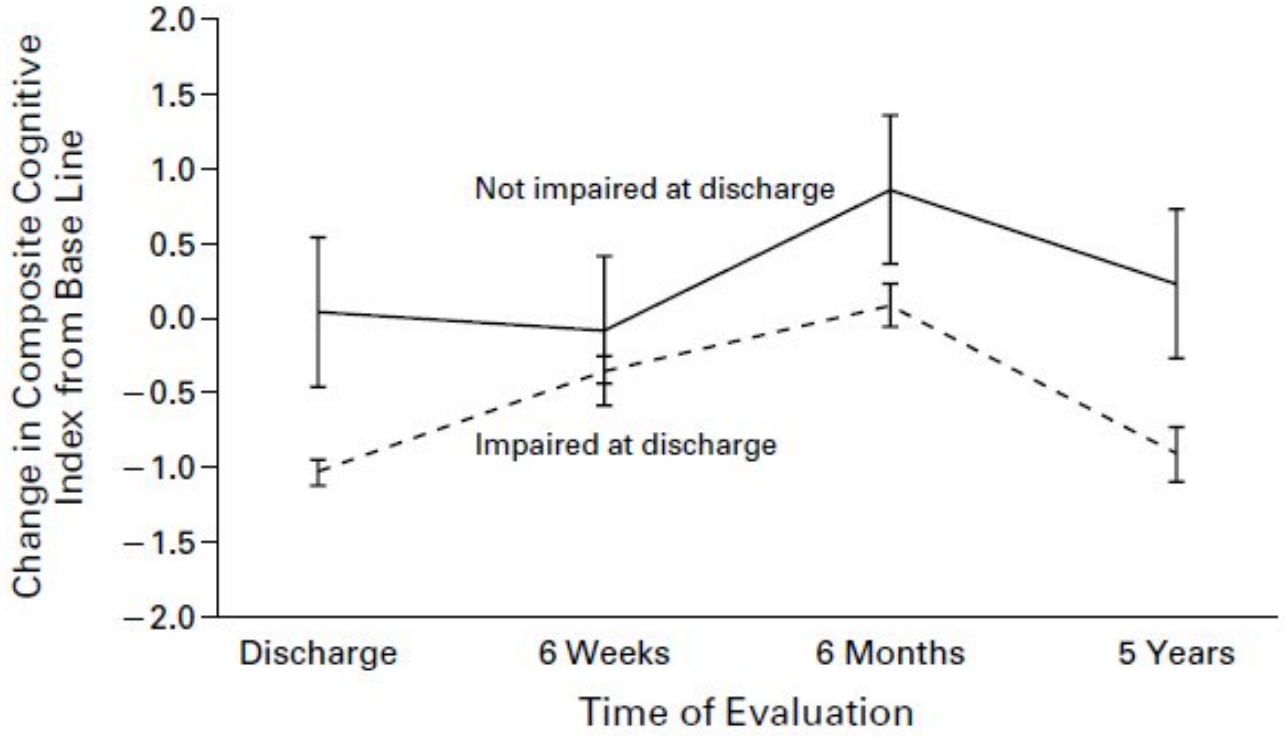

Figure 2. Composite Cognitive Index as a Function of Cognitive Impairment at Discharge.

The composite cognitive index is the sum of the scores for the four domains and includes cognitive decline as well as increases in scores as a result of learning. Positive change represents an overall improvement (learning), whereas negative values indicate overall decline. The I bars represent the standard error.

TABLE 3. UNIVARIABLE AND MULTIVARIABLE PREDICTORS OF COGNITIVE DECLINE AND CHANGE IN THE COMPOSITE COGNITIVE INDEX AT FIVE YEARS.*

VARIABLE	PREDICTORS OF DICHOTOMOUS OUTCOME		PREDICTORS OF CHANGE IN THE COMPOSITE INDEX	
	UNIVARIABLE P VALUE	MULTIVARIABLE P VALUE	UNIVARIABLE P VALUE	MULTIVARIABLE P VALUE
Cognitive decline at discharge	0.006	0.03	0.002	<0.001
Lower left ventricular ejection fraction	0.97	—	0.03	—
Longer duration of aortic cross-clamping	0.82	—	0.85	—
History of hypertension	0.18	—	0.95	—
Longer duration of cardiopulmonary bypass	0.97	—	0.49	—
Female sex	0.28	—	0.67	—
CCS class IV angina	0.17	—	0.50	—
Previous symptomatic neurologic event	0.12	—	0.44	—
Previous myocardial infarction	0.31	—	0.04	—
Diabetes	0.38	—	0.83	—
Higher composite base-line neurocognitive score	0.74	0.07	<0.001	<0.001
Older age	0.02	0.01	<0.001	<0.001
Fewer years of education	0.02	0.003	<0.001	0.003

Association of Neurocognitive Function and Quality of Life 1 Year After Coronary Artery Bypass Graft (CABG) Surgery

Barbara Phillips-Bute, PhD, Joseph P. Mathew, MD, James A. Blumenthal, PhD, Hilary P. Grocott, MD, FRCPC, Daniel T. Laskowitz, MD, Robert H. Jones, MD, Daniel B. Mark, MD, and Mark F. Newman, MD

Objective: Although coronary artery bypass grafting (CABG) has been shown to improve quality of life and functional capacity for many patients, recent studies have demonstrated that a significant number of patients exhibit impairment in cognitive function immediately following surgery and beyond. We sought to determine the impact of this postoperative cognitive dysfunction on quality of life (QOL) and to characterize the dysfunction from the patient's perspective. **Methods:** With Institutional Review Board (IRB) approval and written informed consent, 732 patients at Duke University Hospital undergoing CABG were enrolled. Five hundred fifty-one (75%) completed baseline, 6-week, and 1-year neurocognitive tests and psychometric measures designed to assess QOL. Neurocognitive status was assessed by a composite cognitive index score representing the mean of the scores in four cognitive domains. Change in QOL was assessed by subtracting baseline from 1-year scores for each of 10 QOL measures. The association between QOL and cognitive dysfunction was investigated using multivariable linear regression analysis. **Results:** Cognitive decline limited improvement in QOL, with substantial correlation between change in cognition and change in QOL. One-year QOL measures are associated with both 6-week and 1-year change in cognition (Instrumental Activities of Daily Living, $p < .0001$; Duke Activity Status Index, $p < .02$; Cognitive Difficulties, $p < .0001$; Symptom Limitations, $p = .0001$; Center for Epidemiologic Study Depression, $p < .0001$; General Health Perception, $p = .0001$). **Conclusions:** Postoperative cognitive decline may diminish improvements in QOL. Strategies to reduce cognitive decline may allow patients to achieve the maximum improvement in QOL afforded by CABG, as even short-term cognitive dysfunction has implications for QOL 1 year later.
Key words: neurocognitive function, CABG surgery, quality of life.

TABLE 2. Binary Outcomes Measures in Each Domain[a]

Domain	6 Week, $N = 581$, % of Patients With Deficit	1 Year, $N = 551$, % of Patients With Deficit
Verbal memory and language comprehension	12.02	12.57
Visuoconstruction	11.61	6.26
Abstract recall	4.64	3.69
Complex attention, executive function	7.65	7.51
Overall deficit	41.31	36.84

[a] Deficit in a domain is defined as a standard deviation decline; overall deficit is defined as a standard deviation decline in one or more domains.

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ADVERSE CEREBRAL OUTCOMES AFTER CORONARY BYPASS SURGERY

GARY W. ROACH, M.D., MARC KANCHUGER, M.D., CHRISTINA MORA MANGANO, M.D., MARK NEWMAN, M.D., NANCY NUSSMEIER, M.D., RICHARD WOLMAN, M.D., ANIL AGGARWAL, M.D., KATHERINE MARSCHALL, M.D., STEVEN H. GRAHAM, M.D., PH.D., CATHERINE LEY, PH.D., GERARD OZANNE, M.D., AND DENNIS T. MANGANO, PH.D., M.D., FOR THE MULTICENTER STUDY OF PERIOPERATIVE ISCHEMIA RESEARCH GROUP AND THE ISCHEMIA RESEARCH AND EDUCATION FOUNDATION INVESTIGATORS*

ABSTRACT

Background Acute changes in cerebral function after elective coronary bypass surgery are a difficult clinical problem. We carried out a multicenter study to determine the incidence and predictors of — and the use of resources associated with — perioperative adverse neurologic events, including cerebral injury.

Methods In a prospective study, we evaluated 2108 patients from 24 U.S. institutions for two general categories of neurologic outcome: type I (focal injury, or stupor or coma at discharge) and type II (deterioration in intellectual function, memory deficit, or seizures).

Results Adverse cerebral outcomes occurred in 129 patients (6.1 percent). A total of 3.1 percent had type I neurologic outcomes (8 died of cerebral injury, 55 had nonfatal strokes, 2 had transient ischemic attacks, and 1 had stupor), and 3.0 percent had type II outcomes (55 had deterioration of intellectual function and 8 had seizures). Patients with adverse cerebral outcomes had higher in-hospital mortality (21 percent of patients with type I outcomes died, vs. 10 percent of those with type II and 2 percent of those with no adverse cerebral outcome; P<0.001 for all

STROKE, the third leading cause of death in the United States, will continue to be a challenging problem as the population ages. Patients who undergo myocardial revascularization procedures, now more than 800,000 a year throughout the world, are particularly prone to stroke, encephalopathy, and other neurologic dysfunction, because they are relatively old and have atherosclerotic disease. They are also subject to marked hemodynamic fluctuations; cerebral embolization of atherosclerotic plaque, air, fat, and platelet aggregates; cerebral hyperthermia after the discontinuation of cardiopulmonary bypass; and other inflammatory and neurohumoral derangements associated with surgery.[1-5]

Although cerebral complications are responsible for an increasing proportion of perioperative deaths,[6,7] their incidence and effects have not been rigorously investigated. The majority of studies have been performed only at one center, have enrolled a limited number of patients, or have been retrospective (all of

ADVERSE CEREBRAL OUTCOMES AFTER CORONARY BYPASS SURGERY. GARY W. The New England Journal of Medicine 1996;335:1857-63

- ABD'de 24 merkez 2108 hasta
  - Grup I: Fokal hasar, Stupor, Koma
  - Grup II: Entellektuel aktivitede, hafıza /bilinç de azalma
- 129/2108=% 6,1
- Grup I: %3,1
- Grup II: %3

TABLE 3. MORTALITY AND POSTOPERATIVE RESOURCE USE, ACCORDING TO CEREBRAL OUTCOME.*

VARIABLE	TYPE I OUTCOME (N=66)	TYPE II OUTCOME (N=63)	NO ADVERSE CEREBRAL EVENT (N=1979)
Death during hospitalization — no. (%)	14 (21)	6 (10)	38 (2)
Duration of postoperative hospital stay — days			
Mean ±SD	25.3±22.2	20.5±25.2	9.5±12.4
Median	17.6	10.9	7.7
Duration of ICU stay — days			
Mean ±SD	11.1±15.4	6.6±7.9	2.6±3.5
Median	5.8	3.2	1.9
Discharged to home — no. (%)†	21 (32)	38 (60)	1773 (90)

*P<0.001 for all comparisons among the groups. ICU denotes intensive care unit.

†Patients not discharged to their homes either died or were discharged to intermediate- or long-term care facilities.

POCD insidansını azaltmalıyız

- POCD nasıl azaltabiliriz?

1. CPB idaresi ve
2. Monitörizasyon sistemlerimizi gözden geçirerek
3. End organ perfüzyon yeterliliğini takip ederek

Table 5 Potentially neuroprotective physical interventions in cardiac surgery

General considerations	Expeditious surgery
	Attention to myocardial preservation and haemostasis
Maintaining cerebral perfusion	Avoid prolonged/profound arterial hypotension
	Avoid prolonged systemic hypoperfusion
	Avoid prolonged superior vena caval obstruction
	Consider retrograde cerebral perfusion during DHCA
Reducing cerebral embolization	Adequate anticoagulation
	Minimize aortic manipulation/instrumentation
	Careful (de)cannulation of the aorta
	Avoid venous air entrainment
	Use of arterial line filter
	Avoid CPB altogether (i.e. 'beating heart' procedures)
	Use of exhaustive deairing/debridement procedures
	Avoid or reduce use of cardiotomy suction
	Consider heparin-bonded circuits
Temperature management	Moderate hypothermia (i.e. 32°C)
	Avoid rapid/excessive rewarming
Acid–base management	Alpha-stat regimen (pH-stat during cooling before DHCA and in patients with significant aorto-pulmonary anastomoses)
	Avoid hypercapnia and hypocapnia
Other	Avoid/treat hyperglycaemia
Advanced neurological monitoring	Jugular venous oxygen saturation
	Near infrared spectroscopy (NIRS)
	Electroencephalography (EEG)
	Transcranial Doppler sonography

CPB idaresi

- 60 yıllık geçmişe rağmen, CPB sırasındaki izlem parametrelerinin ideal değerleri hala tanımlanamamıştır

CPB sırasında hangi parametreleri kontrol ediyoruz

- **Akım**
  - Erişkinde **30-70** ml/kg
  - **1.6 -3.2** L/m^2
- **Basınç**
  - 30-100 mmHg
- **Hematocrit**
  - %18-30
- **Kan gazı**
  - **pH, α stat strateji**
  - $PaO_2$100-600 mmHg
  - $PaCO_2$ 30-50 mmHg
- Hipotermi

CPB ve Hipotermi

- Beyin met.↓
  - Bu etki, global hipoksiye karşı metabolik etki ile bütünlüğü (beyni) korumakta
  - Nörolojik outcome'ı düzeltmekte
  - Bu değişiklik postoperatif dönemde de devam etektedir

- Greeley WJ. Ann Thorac Surg 1993;56:1464-1466
- Mezrow CK. Ann Thorac Surg 1994; 57:532-539
- Cook DJ. J Thorac Cardiovasc Surg 1994;107:1020-1028
- Ehrlich MP. Ann Thorac Surg 2002;73:191-197

CPB ve Hemodilüsyon

- Cerebral hiperemi (kan akımında artma)
- Eğer hemodilüsyon ciddi ise, OER da ↑ ?????

CPB, Hipotermi ve hemodilüsyon

- Serebrovasküler direnç
- Sunum/tüketim dengesi üzerine olan etkileri

  zıtlık göstermekte
- Hipotermi ve hemodilüsyonun neden oldukları bu oto regülasyondaki bozulma;
  - Postop. dönemde serebrovasküler direnç artışına neden olmaktadır

Cook DJ. Ann Thorac Surg 1995;60:1671-1677
Abdul-Khaliq H. Perfusion 2002;17:179-185

CPB ve pH Stat strateji

- Serebral kan akımını artırır;
- Ancak;
- Artan cerebral kan akımına karşı bir ADAPTASYON mekanizması gelişir
  - CPB sonrası/CPB CO2'in verilmediği dönemde serebrovasküler direncin yüksek kalır ↔ kan akımının azalması neden olur

- Sonuç; pH stat stratejisinin, CPB ve sonrası dönemde etkisi kompleks (karışık)

- Daubeney PE. Eur J Cardiothorac Surg 1998;13:370-377
- Hoffman GM. J Thorac Cardiovasc Surg 2004;127:223-233

Sonuç

- CPB idaresi oldukça karmaşık
- Hemodinamik/kan gazı parametreler için standart değerler vermek olanaksız.

CPB

- Monitorizasyon
  - Genel monitörizasyon

Monitorizasyon parametreleri

- Venöz PvO_2 ve SvO_2
- Laktat
- Dv-aPCO_2
- BE
  - Her parametreye ait belli eksikliklerin varlığının bilinmesine rağmen, değerli parametreler
  - Ancak;
    - Sistemik doku perfüzyonu hakkında bilgi verir,
    - Bölgesel değişikliği yansıtmazlar

CPB

- Monitorizasyon
  - Nöromonitörizasyon

CPB sonrası serebral hasar

1. Hipo/ Hiper perfüzyon
2. Embolizasyon
3. Sistemik inflamatuvar cevap

- **Nörofizyolojik monitörizasyon (multimodal) sayesinde**
  - TCD (2 MHz)
  - EEG (4 kanal)
  - NIRS
    1. Hipo/ Hiper perfüzyon
    2. Embolizasyon tespit edilebilmektedir

TCD (2 MHz)

- Hipoperfüzyon;
  - Orta serebral arter kan akım hızı (BFV) başlangıç değerinin %20 altında ise
- Hiperperfüzyon;
  - Orta serebral arter kan akım hızı (BFV) başlangıç değerinin iki katını (% 200) geçerse
- Emboli;
  - High-intensity transient signals (HITS) indicative of particulate or gaseous emboli.
-

TCD

1. CPB sırasındaki;
   - Hemodilüzyon,
   - Hipotermi ve
   - Asid-baz idaresinin alfa-stat ile yapılması
- Serebral kan akım miktarını ve hızını etkileyeceğinden yeni threshold değerlerinin belirlenmesi gerekir

2. CPB sırasında hemodinami her an değişebileceğinden devamlı olmayan monitörizasyonun yeri ???
3. Eğitim

EEG

- Serebral kortikal sinaptik aktiviteyi ölçmekte

- Eğer,
- Aktivite başlangıç değerinin %50 altına düşer ise

- Bu durum iskemi olarak tanımlanmakta

EEG

- Quantitative (nicel) EEG monitörizasyonunun serebral iskemiyi belirlemedeki
  - Sensitivitesi yüksek,
- Fakat;
  - Spesifitesi düşüktür (derin anestezi ve hipotermi)

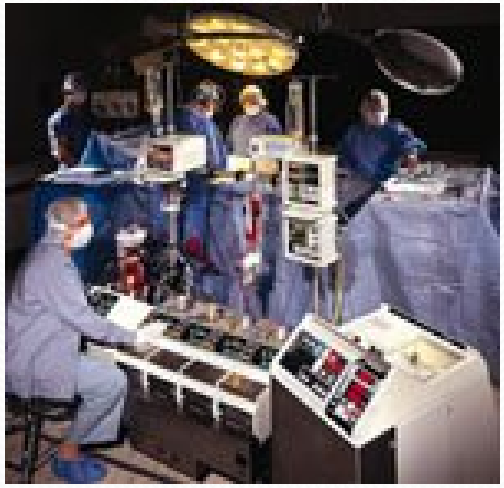
Non-anesthetic Factors Affecting EEG

Miller et al.

- Surgical
  - Cardiopulmonary bypass
  - Occlusion of major cerebral vessel (carotid cross-clamping, aneurysm clipping)
  - Retraction on cerebral cortex
  - Surgically induced emboli to brain
- Pathophysiologic Factors
  - Hypoxemia
  - Hypotension
  - Hypothermia
  - Hypercarbia and hypocarbia

CPB sırasında EEG kullanımı

- Teorik olarak
  - Kanulasyona bağlı embolik olaylarda
  - Yüksek riskli karotis hastalarında faydalı
  - Ancak;
    - PCO2
    - OAB değişikliği
    - Hipotermi
    - Hemodilüsyon



varlığında EEG değişikliğinin yorumlanması güçtür

Gelişmiş monitorizasyon

- Near Infrared Spectroscopy (NIRS)

ACIBADEM
ÜNİVERSİTESİ

INVOS 5100 C System

OxyAlert™ NIRSensor

Pediatric, Adult

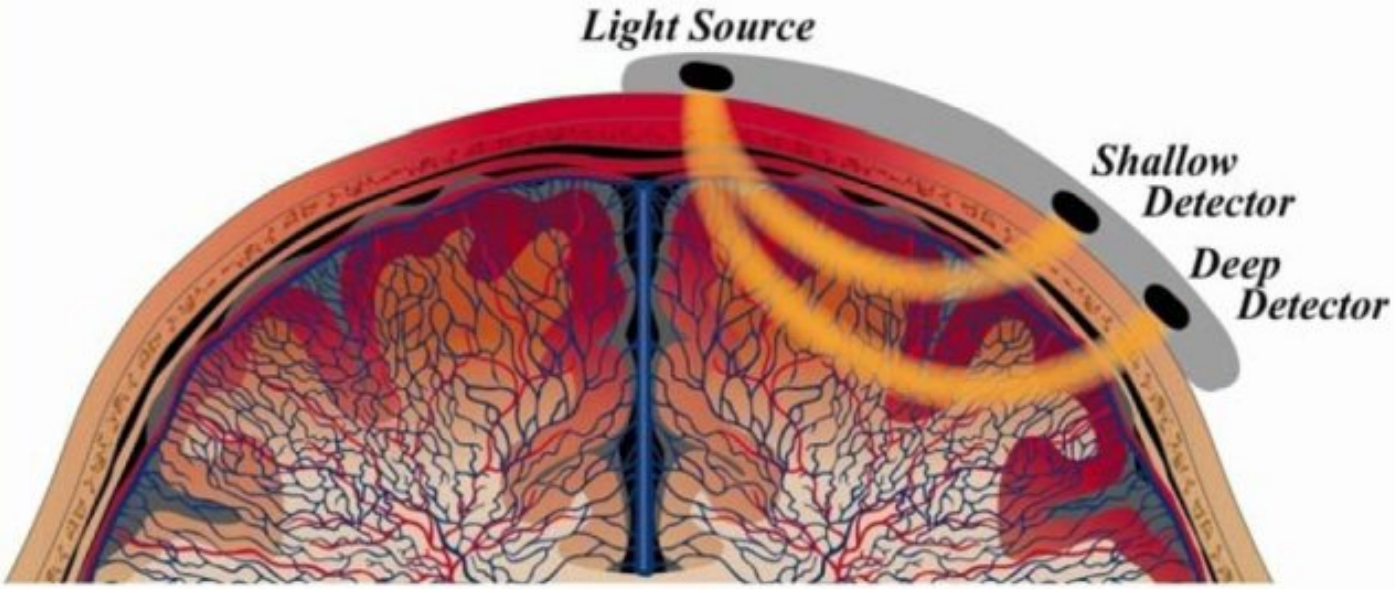
Infant/Neonate

Serebral rejional oksijen saturasyonu (rSO_2)

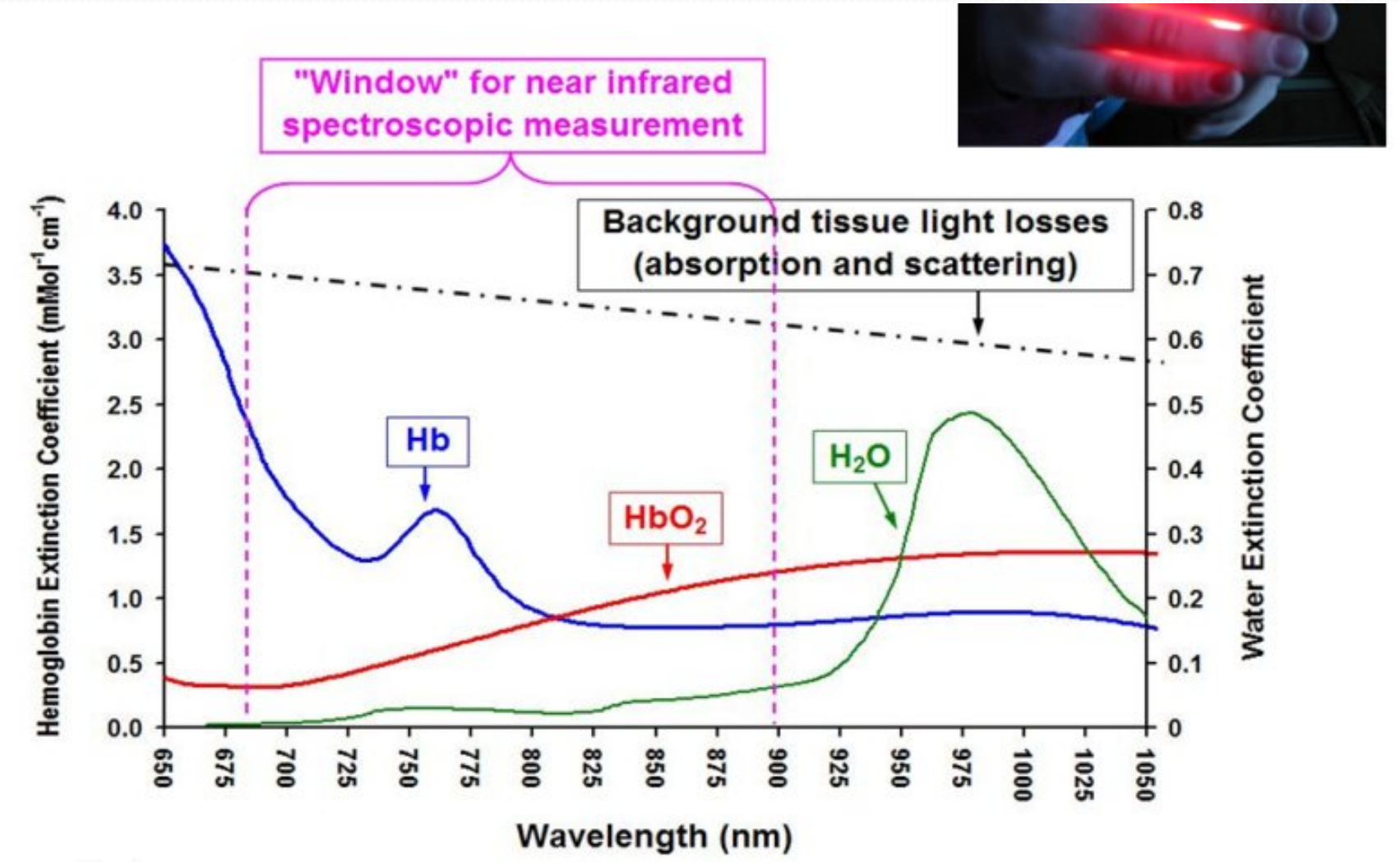
- Spektroskobik olarak bakıldığında ;
  - Cihaz, 660-940 nm dalga aralığında bir ışık göndermekte (LED)
  - Bu dalga boyundaki ışığı absorbe eden bileşiklerden,
    - Oksi Hb, Deoksi Hb →ışığı güçlü bir şekilde absorbe etmekte
    - Yağ, Su, cilt, kemik →ışığı çok az absorbe etmektedir.
- İnfrared ışığın bu özelliği bize;
  - Hb/HbO hakkında bilgi vermektedir.

ACIBADEM
ÜNİVERSİTESİ

How the INVOS System can Help



The INVOS System uses two depths of light penetration to subtract out surface data, resulting in a regional oxygenation value for deeper tissues.



ACIBADEM
UNIVERSITESI

Hb Extinction Coefficient ($mMol^{-1}cm^{-1}$)

- Belli bir dalga boyundaki ışığı, kütlelerin absorbe etme dereceleri

	680 nm	900 nm
Hb	2,4	1,2
HbO_2	0,4	0,8

ACIBADEM
ÜNİVERSİTESİ

rSO2'deki anlamlı değişim

- Bazal değerden %20 den fazla düşme veya
- %50- ölçülen değer (%) x saniye >3000 % sn
  - 50-35 x 300 =4500 % sn

ACIBADEM
ÜNİVERSİTESİ

Müdahaleler

1. Baş ve kanül pozisyonunu kontrol et
2. PCO2 >40 mmHg sağla (taze gaz akımını azalt)
3. Debi
4. OAB
5. Fio2
6. Anestezi derinliğini artır
7. Serebral vazodilatatör kullan
8. RBC transf. düşün

ACIBADEM
ÜNİVERSİTESİ

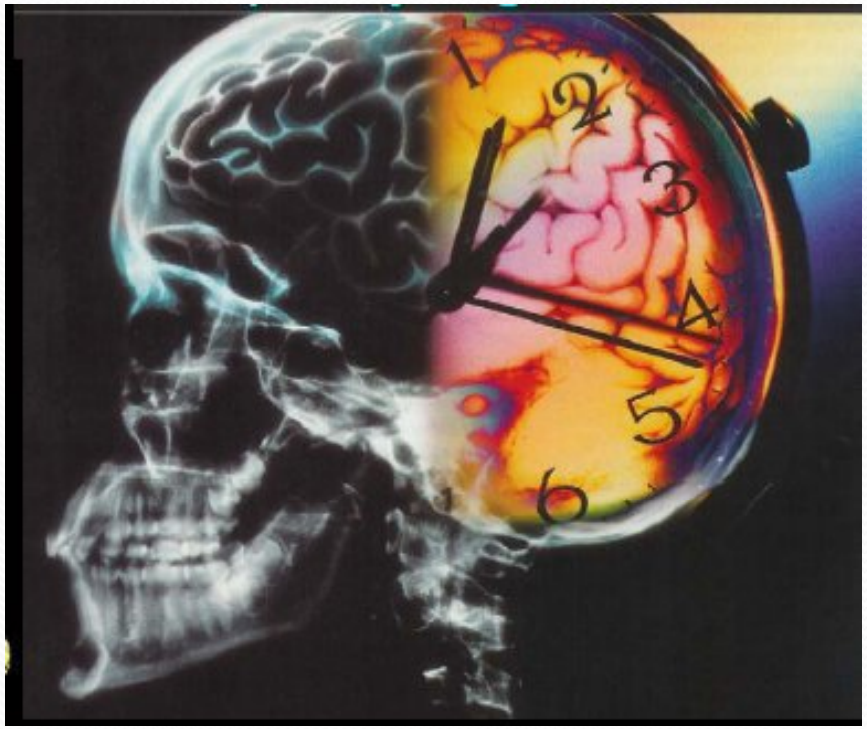
Serebral oksimetre

- Serebral kan akımı oto regülasyonuna

- Serebral arterioler CO2 reaktivitesine

- Vaso-nöronal (flow-metabolizma) etkiye bağlı olarak

  - An ve an değişebilen serebral akımı devamlı olarak ölçmektedir.



Doğru uygulama ne ?

- Sadece;
  - Kan akım hızı
  - Kan basıncı ve
  - Kan gaz analizi ile

  - Yeterli doku perfüzyonunun sağlandığından emin olunamaz.

- Akım/basınç dışı parametrelerin değerlendirilmesi gerekir

Akım/basınç dışındaki parametreler

- Oksijen sunumu (Hb,Hct, PO2)
- Oksijen tüketimi (**VO_2**)
  - Hastanın ısısı
  - Anestezi derinliği
- Perfüzyona duyarlı organ fonksiyonları,
  - kalp, Böbrekler, Beyin
- Sistemik hastalıklar (DM)
- Periferik arter hastalığı
- Hemoliz
- 3. boşluklara kayıp-ödem (kapiller blok)

End organ perfüzyon yeterliliğini takibi

- Beyin perfüzyonu ↔ NIRS

Fudickar et al. BMC Anesthesiology 2011, 11:7
http://www.biomedcentral.com/1471-2253/11/7

BMC Anesthesiology

RESEARCH ARTICLE Open Access

Postoperative cognitive deficit after cardiopulmonary bypass with preserved cerebral oxygenation: a prospective observational pilot study

Axel Fudickar*, Sönke Peters, Claudia Stapelfeldt, Götz Serocki, Jörn Leiendecker, Patrick Meybohm, Markus Steinfath, Berthold Bein

Abstract

Background: Neurologic deficits after cardiac surgery are common complications. Aim of this prospective observational pilot study was to investigate the incidence of postoperative cognitive deficit (POCD) after cardiac surgery, provided that relevant decrease of cerebral oxygen saturation (cSO2) is avoided during cardiopulmonary bypass.

Methods: cSO2 was measured by near infrared spectroscopy in 35 patients during cardiopulmonary bypass. cSO2 was kept above 80% of baseline and above 55% during anesthesia including cardiopulmonary bypass. POCD was tested by trail making test, digit symbol substitution test, Ray's auditorial verbal learning test, digit span test and verbal fluency test the day before and 5 days after surgery. POCD was defined as a decline in test performance that exceeded - 20% from baseline in two tests or more. Correlation of POCD with lowest cSO2 and cSO2 - threshold were determined explorative.

Results: POCD was observed in 43% of patients. Lowest cSO2 during cardiopulmonary bypass was significantly correlated with POCD ($p = 0.015$, $r2 = 0.44$, without Bonferroni correction). A threshold of 65% for cSO2 was able to predict POCD with a sensitivity of 86.7% and a specificity of 65.0% ($p = 0.03$, without Bonferroni correction).

Conclusions: Despite a relevant decrease of cerebral oxygen saturation was avoided in our pilot study during cardiopulmonary bypass, incidence of POCD was comparable to that reported in patients without monitoring. A higher threshold for cSO2 may be needed to reduce the incidence of POCD.

Fudickar et al. BMC Anesthesiology 2011, 11:7

- CPB uygulana 35 hasa çalışmaya alınmış
- Tüm hastaların perioperatif dönem süresince
  - rSo2 değerlerini, başlangıç (bazal) değerinin %80 den fazla veya
  - rSO2 > %55 den fazla olacak şekilde tutmaya çalışmışlar
  - Ameliyat öncesi ve postop 2. gün değerlendirme testleri uygulanmış
  - 6 ayrı test kullanılmış

Sonuçlar

1. rSo2> bazal değerin %80 den veya %55 tutulmasın rağmen

   POCD oranı %43

2. CPB sırasındaki düşük rSO2 ile POCD korele olduğu

   - $r^2 = 0.44$, $p = 0.015$.

3. POCD açısında CPB sırasındaki kritik rSO2 değerinin bazal değerin % 65 olduğunu ifade etmişler ve

Buradaki ;

sensivitenin % 86.7 ve

spesifitenin % 65 ($p = 0.03$).

EEG
TCD
NIRS ile nöromonitörizasyon yaptıkları hastalarında

- %59 oranında serebral oksijen sunum/tüketim imbalansı tespit ederek gerekli müdahalede bulunmuşlar

- % 2 hastada yapılan müdahaleye rağmen düzelme olmamış

- Nöromonitörizasyon yapılanlarda ciddi beyin hasarı insidansı %3

- Nöromonitörizasyon yapılmayanlarda ciddi beyin hasarı insidansı %6,1, p=0,03

Protective Effect of Neuromonitoring during Cardiac Surgery

HARVEY L. EDMONDS, JR.

Neuromonitoring Associates, Incorporated, Louisville, Kentucky, USA

ABSTRACT: This study was a retrospective examination of the influence of multimodality neuromonitoring on the incidence of serious brain injury associated with a common type of adult cardiac surgery, coronary artery bypass grafting (CABG). Multichannel EEG, cerebral oximetry, and transcranial Doppler ultrasound were used to detect and correct imbalances in cerebral perfusion and oxygenation. Imbalances were detected in 59% of the cases and successfully corrected in all but 2%. In the absence of neuromonitoring, the expected incidence of serious brain injury is 6.1%. With neuromonitoring, the actual observed incidence was 3.0% (P = 0.03). The apparent improvement can be attributed primarily to a reduction in the number of nonembolic diffuse injuries.

KEYWORDS: cardiac surgery; brain injury; neuroprotection; EEG; cerebral oximetry; transcranial Doppler ultrasound; outcome

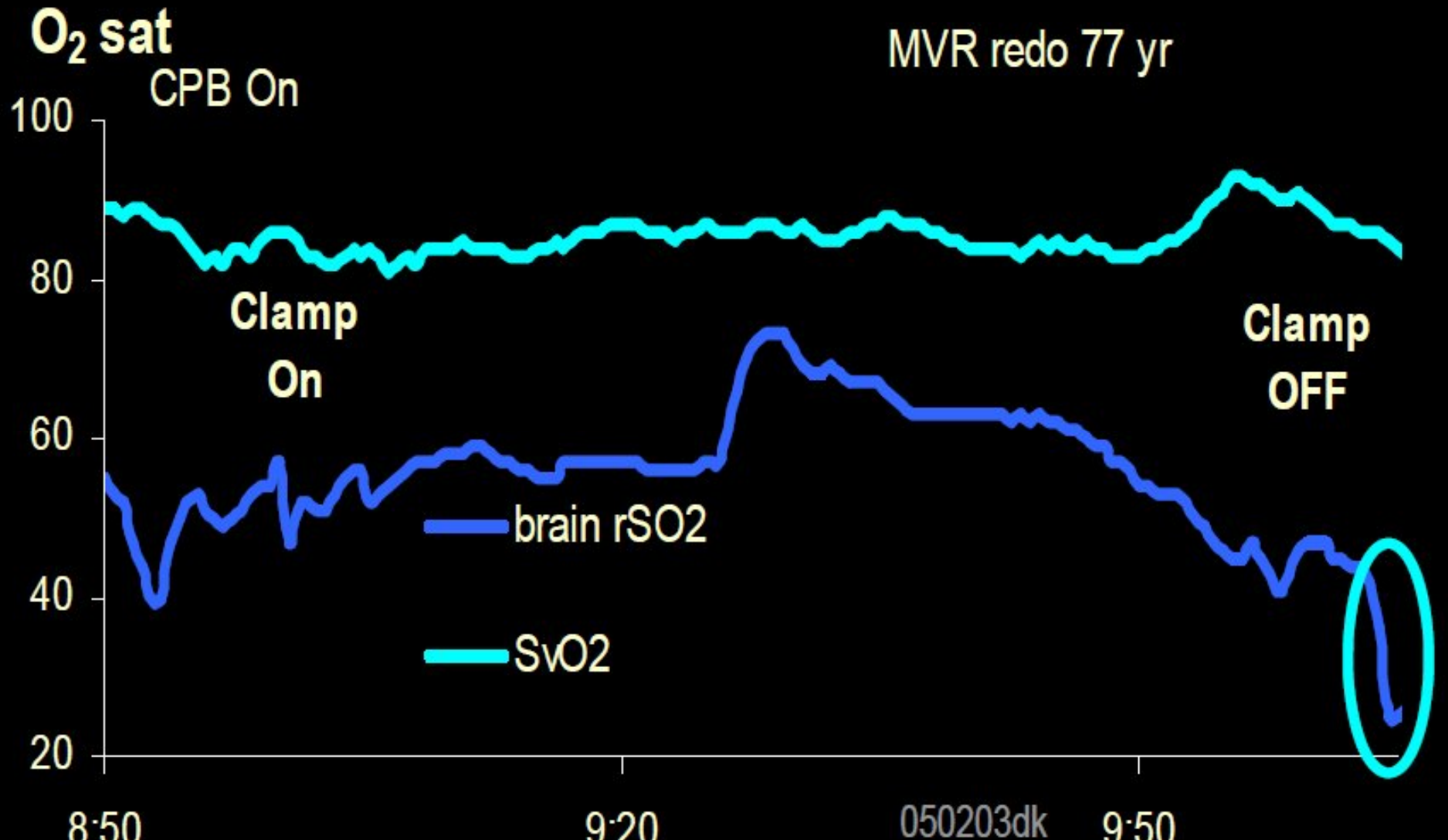
Ann. N.Y. Acad. Sci. 2005;1053: 12–19

TABLE 1. Physiologic and anesthetic imbalance detected by neuromonitoring

Modality	Imbalance	Incidence
Cerebral oximetry	Desaturation > 20%	42%
Transcranial Doppler	Velocity < 20% baseline	13%
	Velocity > twice baseline	2%
	Air leak	1%
EEG	New focal abnormality	1%

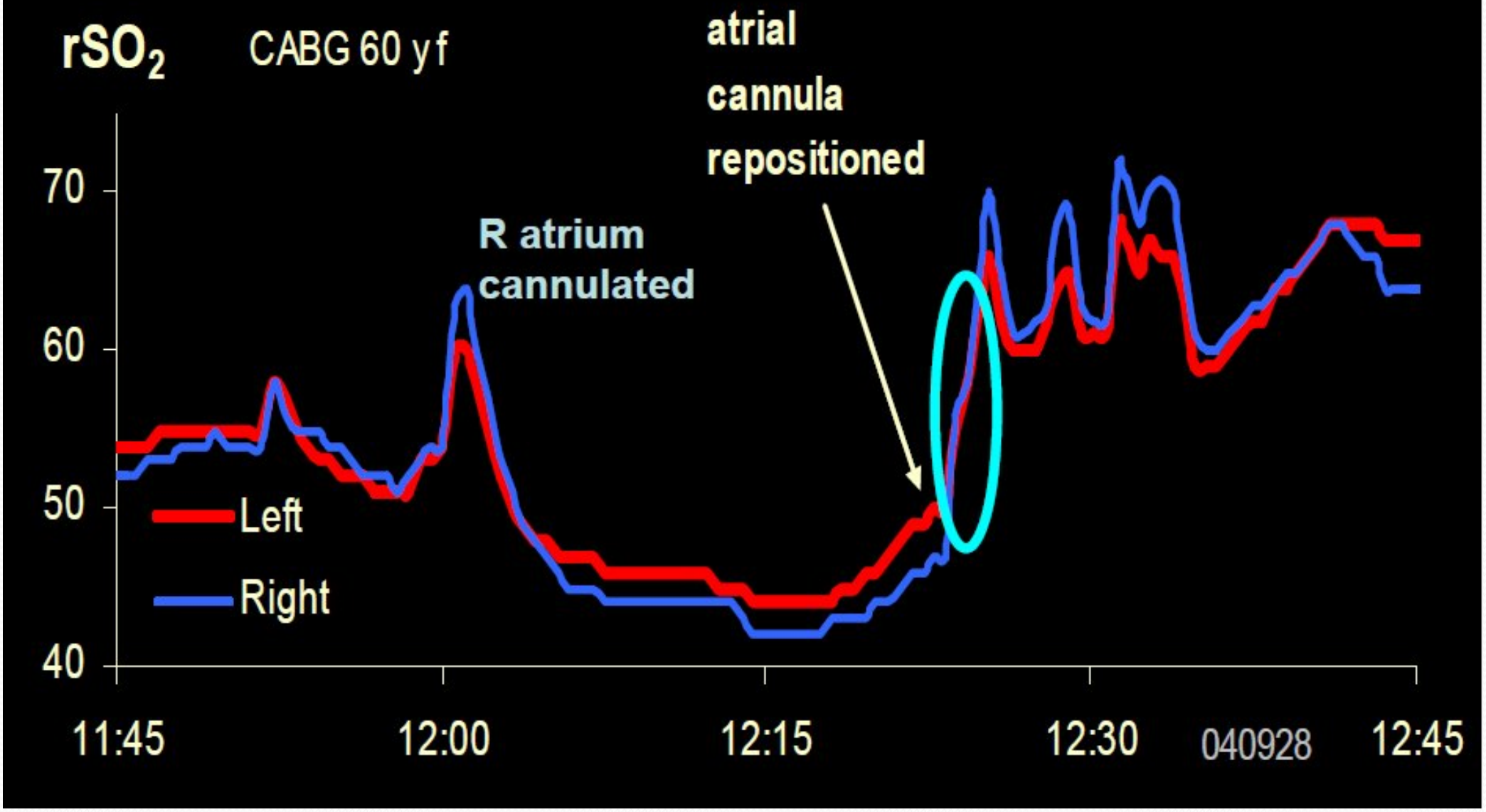
Brain rSO_2 detects oxygen imbalance invisible to SvO_2.

Yeh TJ Jr et al. 2001 J Thorac Cardiovasc Surg 122:192
Hoffman GM 2006 Ann Thorac Surg 81:S2373
Dullenkopf A et al. 2007 J Cardiothorac Vasc Anes 21:535



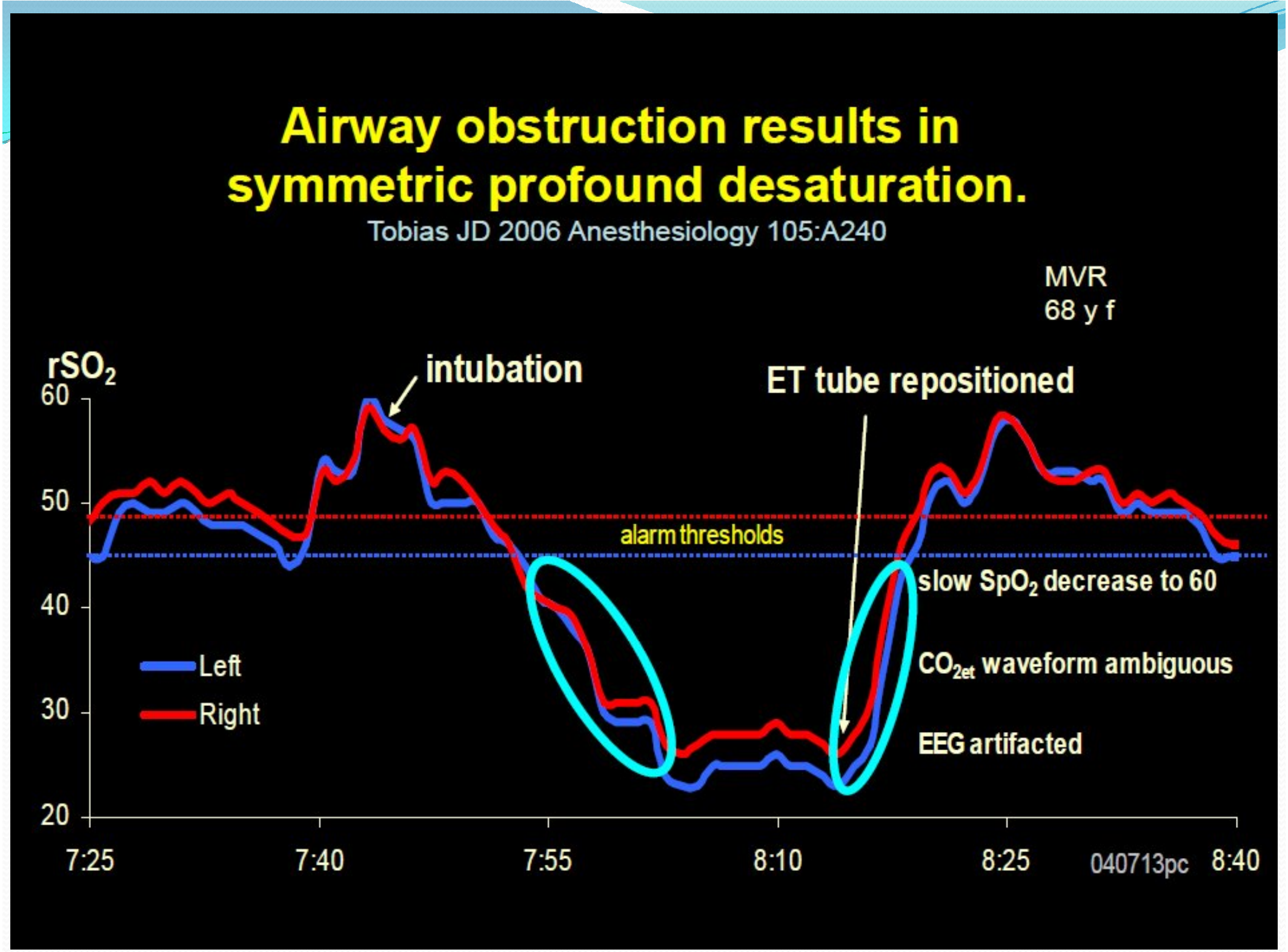
Atrial cannula malposition may cause symmetric desaturation due to cerebral venous outflow obstruction.

Han SH 2005 J Cardiothorac Vasc Anesth 19:420



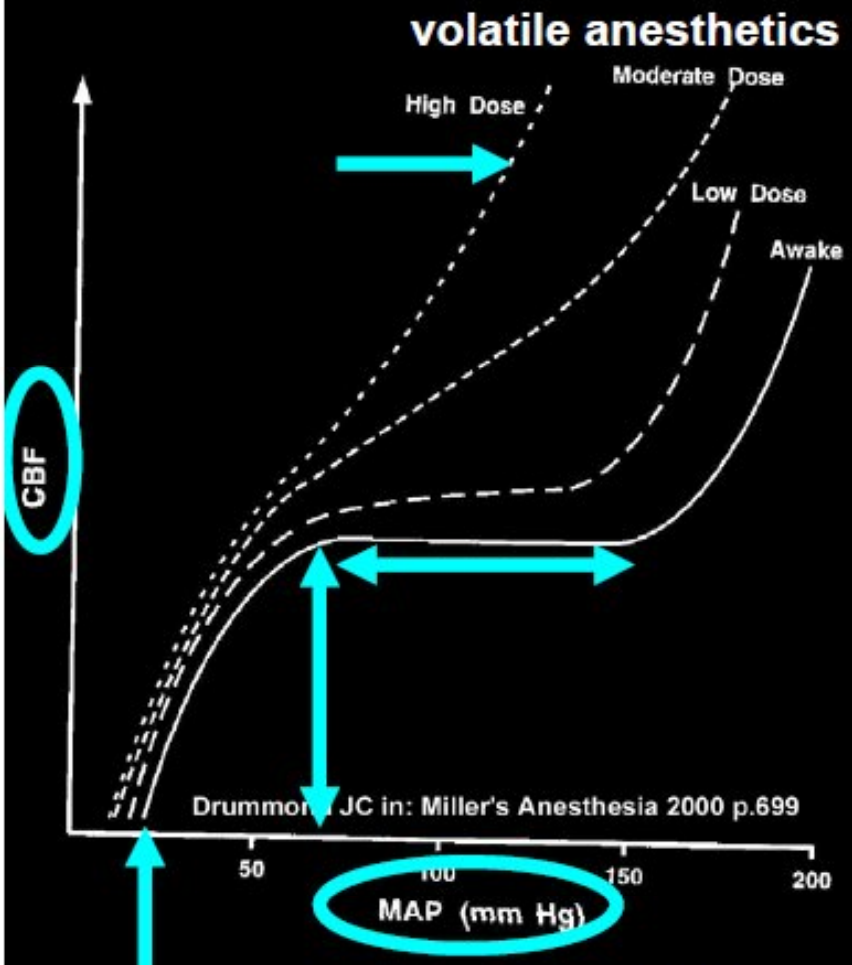
Airway obstruction results in symmetric profound desaturation.

Tobias JD 2006 Anesthesiology 105:A240



Autoregulation:

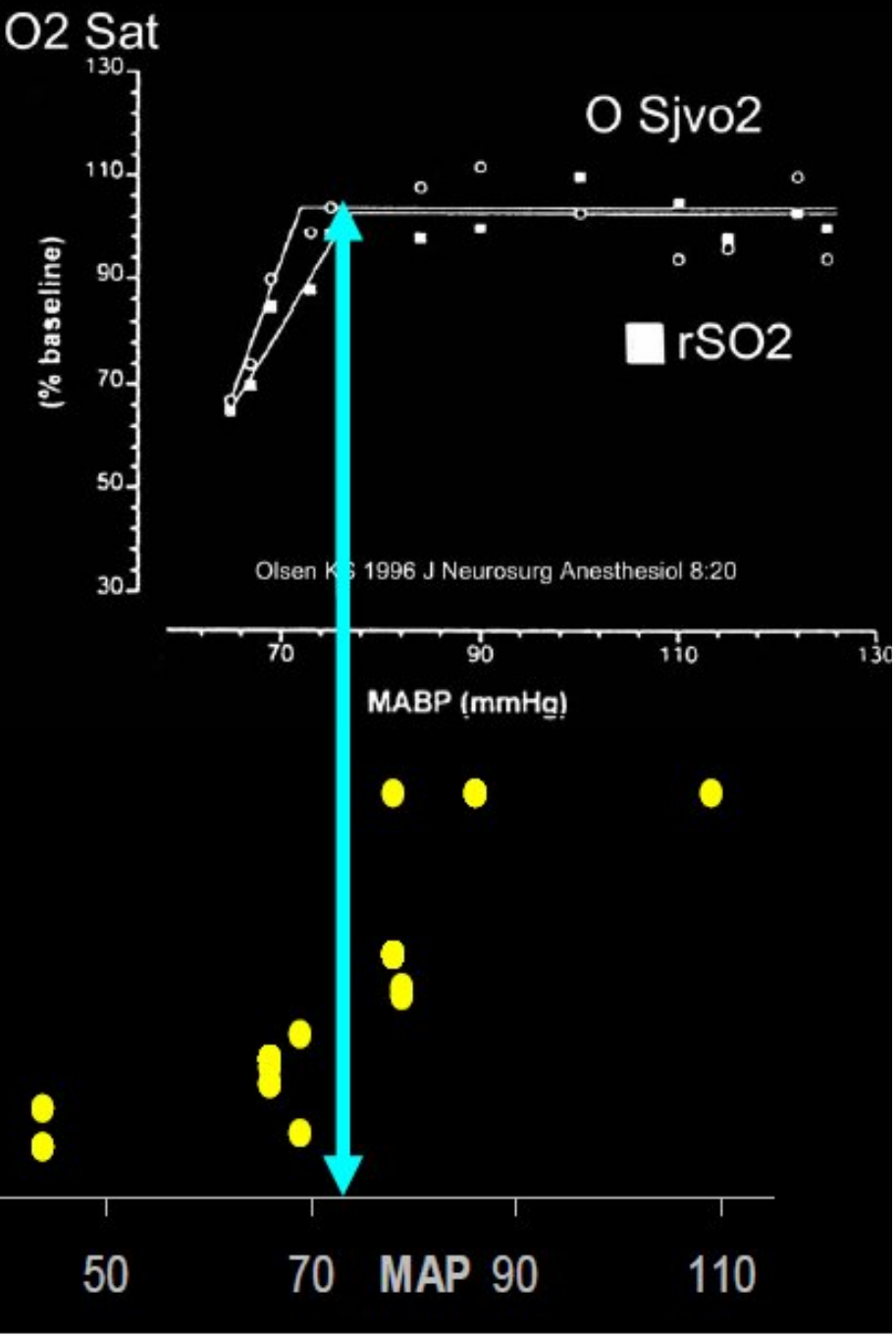
cerebral blood flow (CBF) independent of mean arterial pressure (MAP)



Zero Flow Pressure (ZFP)

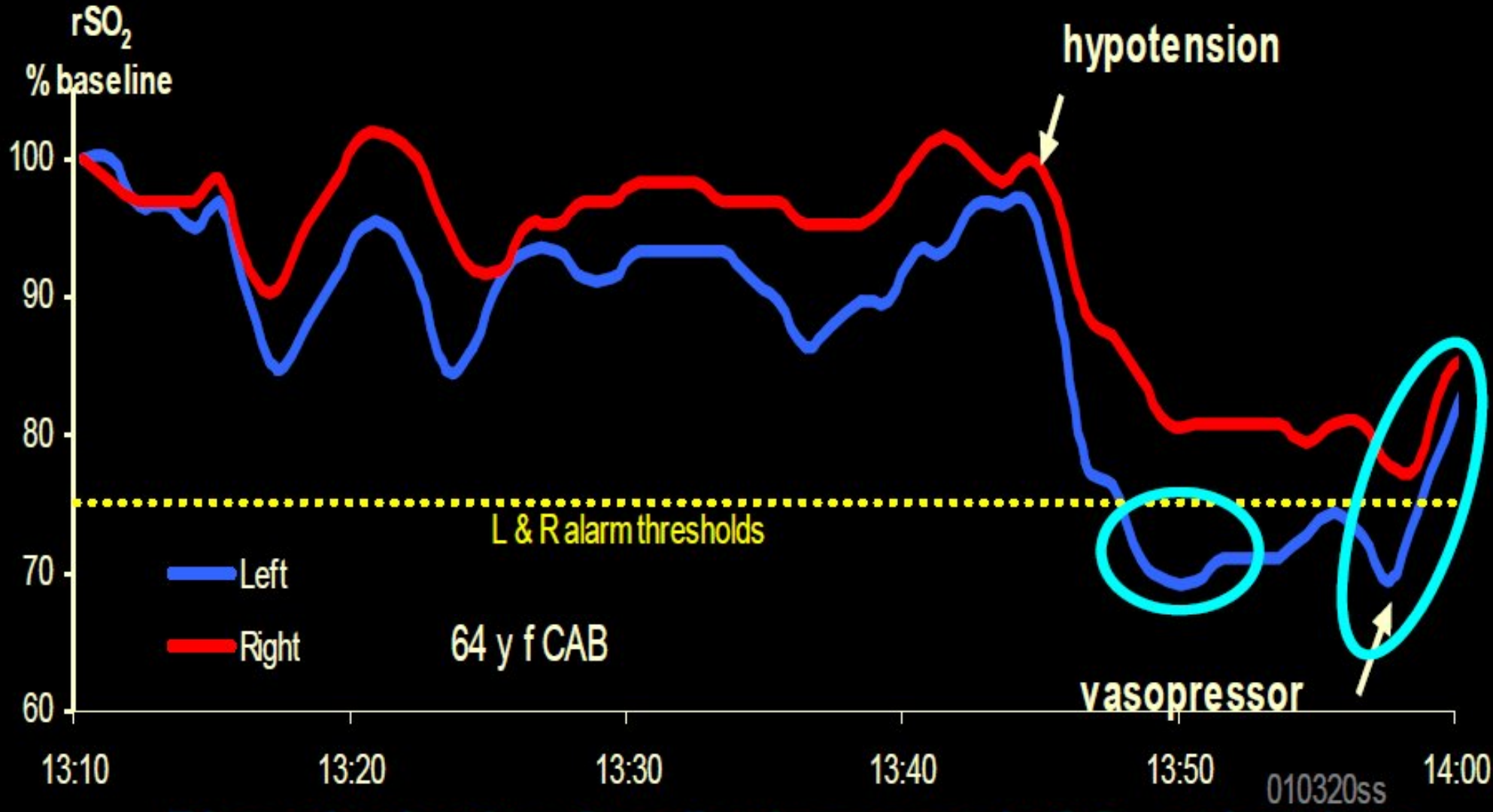
Norepi increases both ZFP & MAP.

Moppett IK 2008 Br J Anaesth 100:240



~25% rSO_2 interventions involve blood pressure increase.

Edmonds HL Jr 2005 Ann NY Acad Sci 1053:12



Phenylepherine 2 µg/kg increased rSO_2 only when MAP below the lower limit of autoregulation.

Yao FSF 2002 Anesthesiol 96:A157

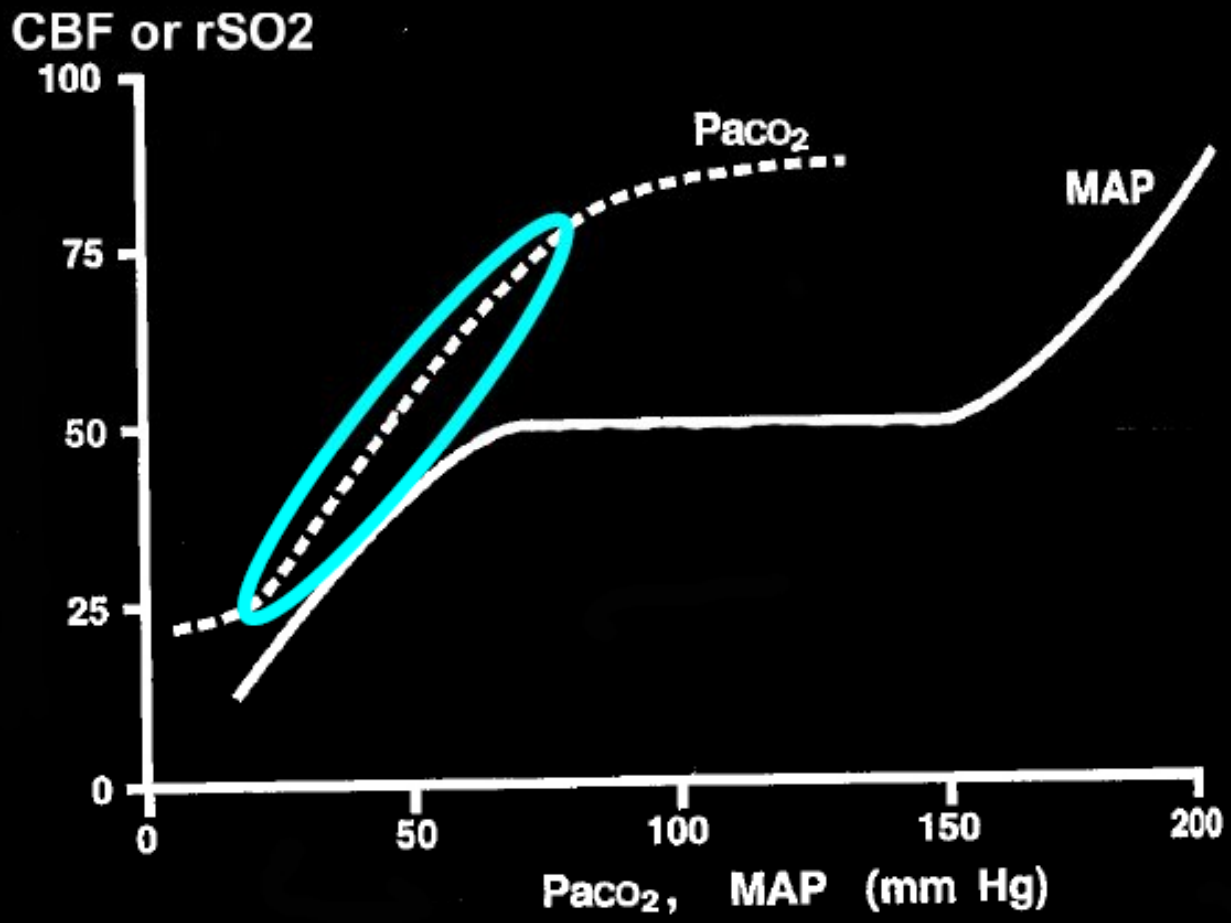
Cerebral arteriolar CO_2 reactivity is necessary, but insufficient, precondition for autoregulation.

Unreactivity = Dysautoregulation

Normal CO_2 reactivity

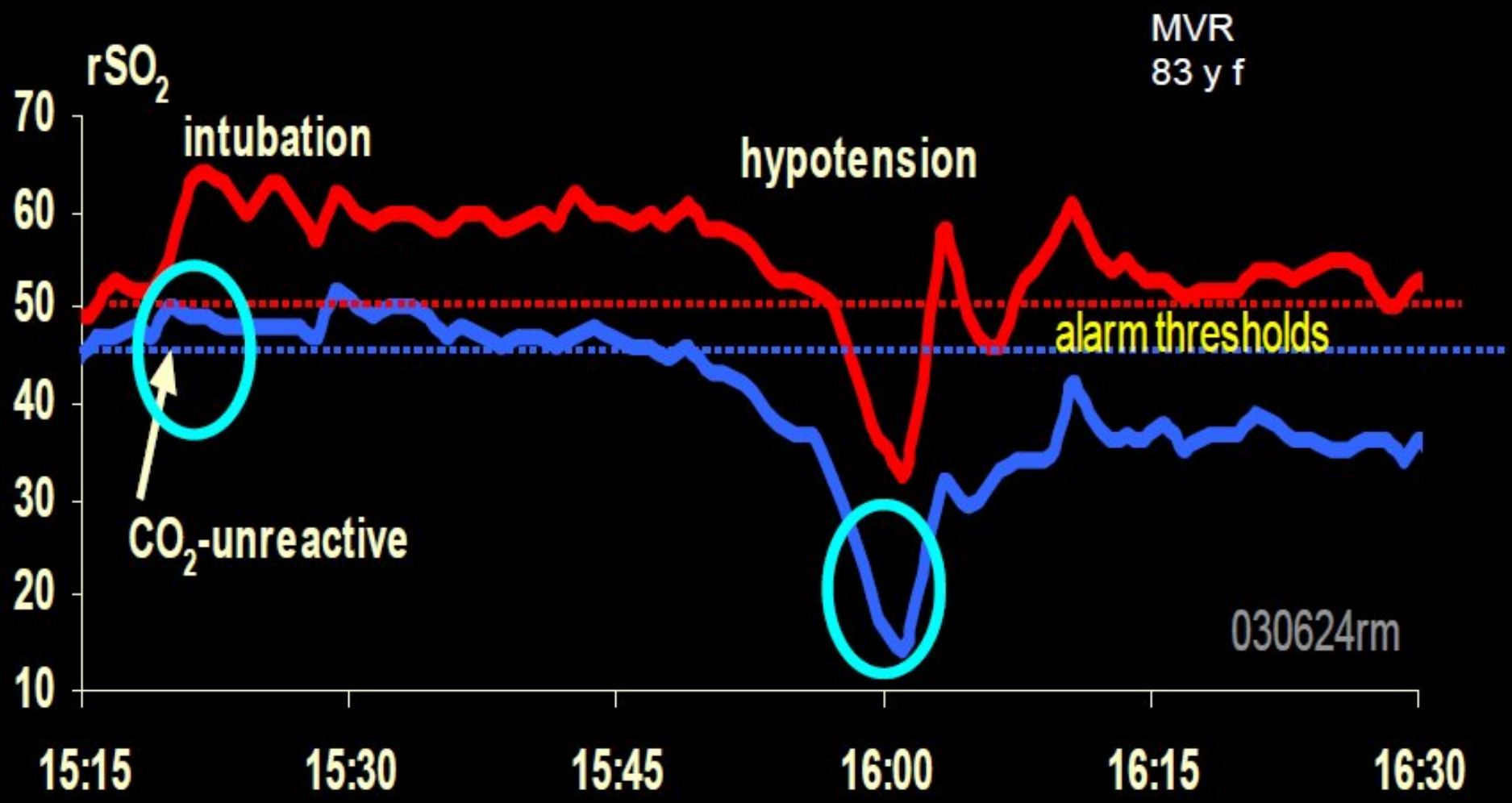
$2\%_{rSO2} / mmHg_{CO2}$

Pollard V 1996 Anesth Analg 82:278



Profound desaturation may occur in CO_2-unreactive brain regions during hypotensive events.

Edmonds HL Jr 2004 Semin Cardiothorac Vasc Anesth 8:147



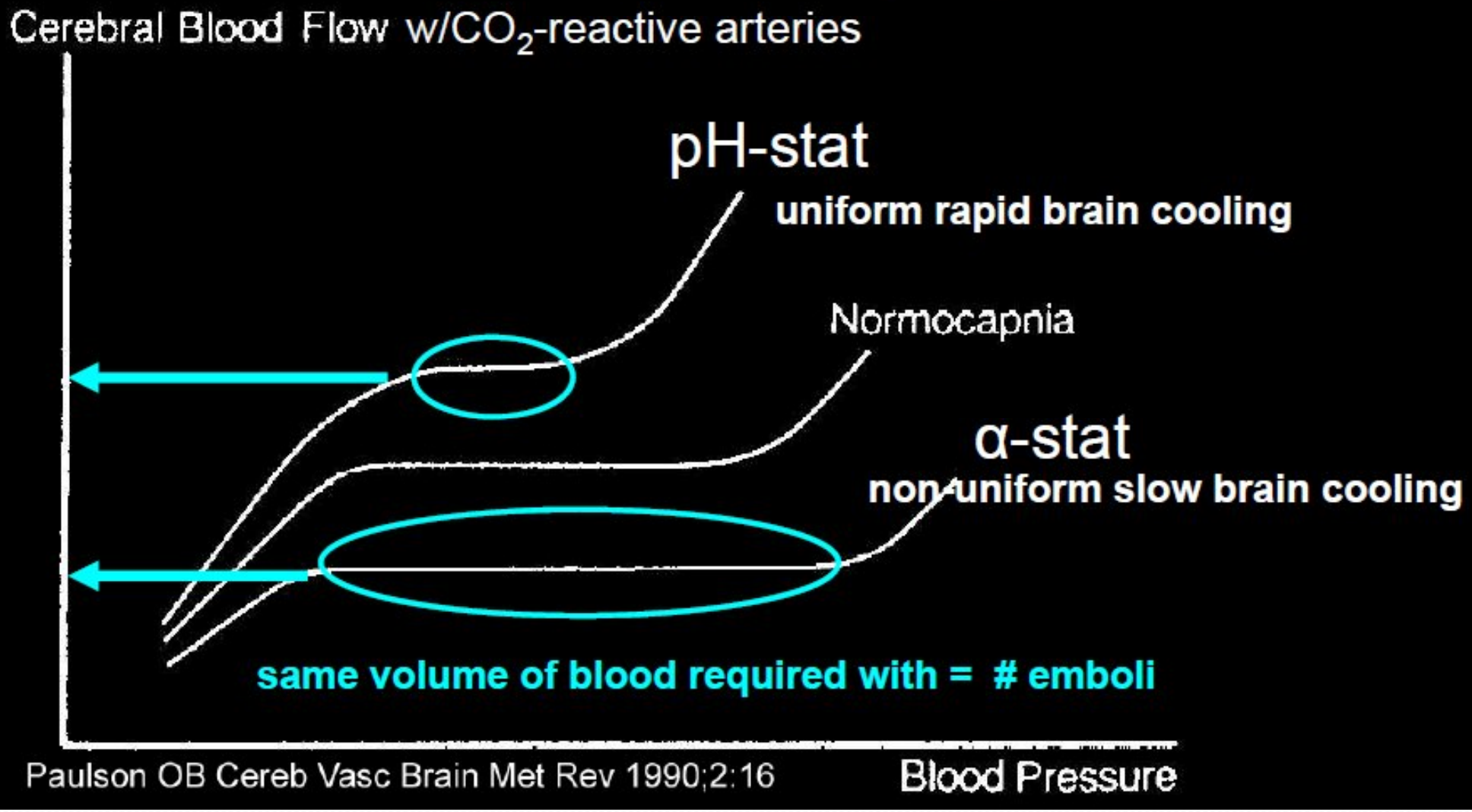
With CO_2-reactive arterioles, extent of cerebral hyperoxia with deep cooling depends on acid-base management.

Pearl JM et al. Ann Thorac Surg 2000; 70:751

Jonas RA: Comprehensive Surgical Management of Congenital Heart Disease, 2004, pp. 151-60

Randomized Clinical Trial (RCT) found improved outcome with pH-stat.

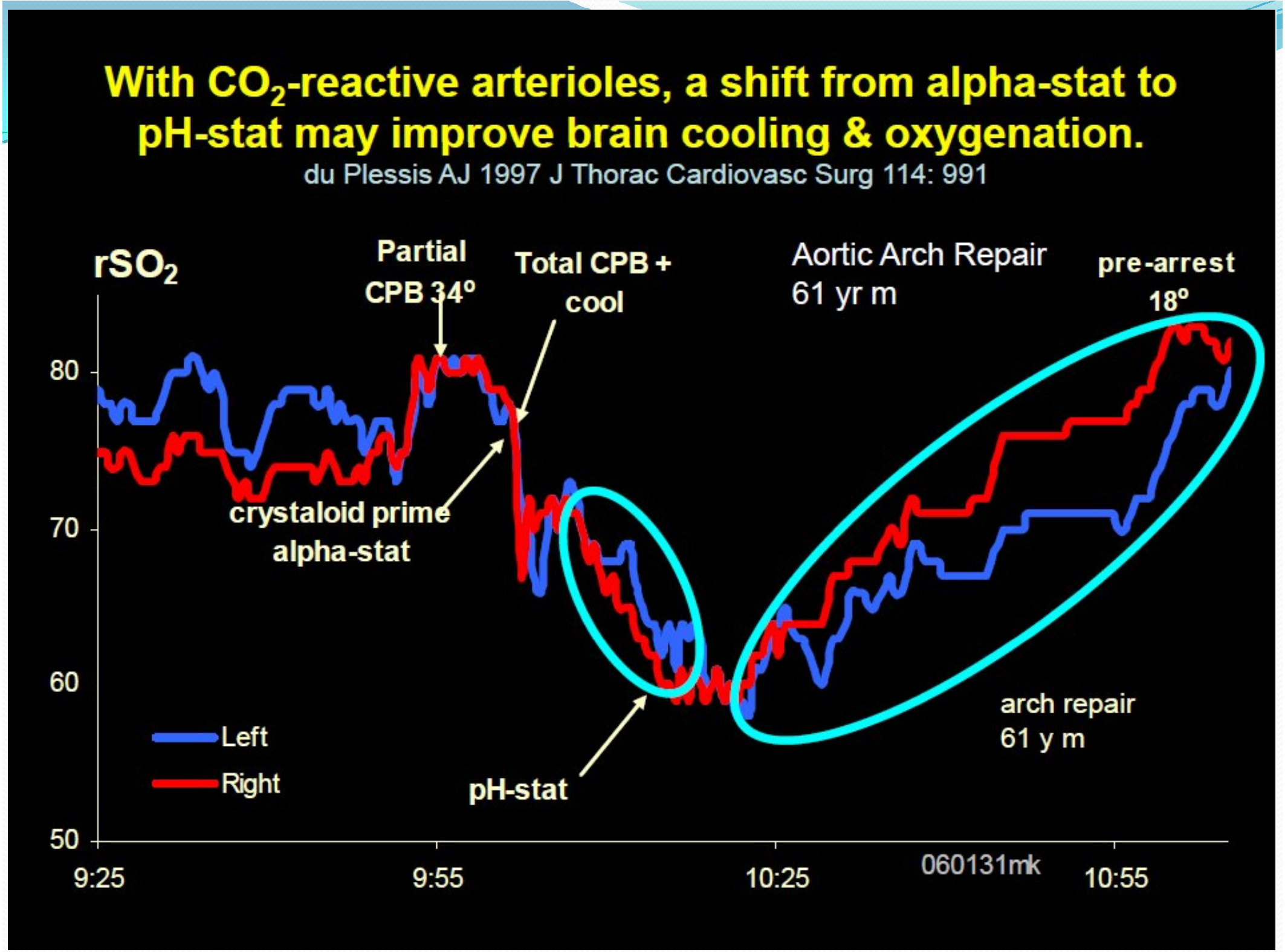
du Plessis AJ et al. J Thorac Cv Surg 1997;114: 991



Paulson OB Cereb Vasc Brain Met Rev 1990;2:16

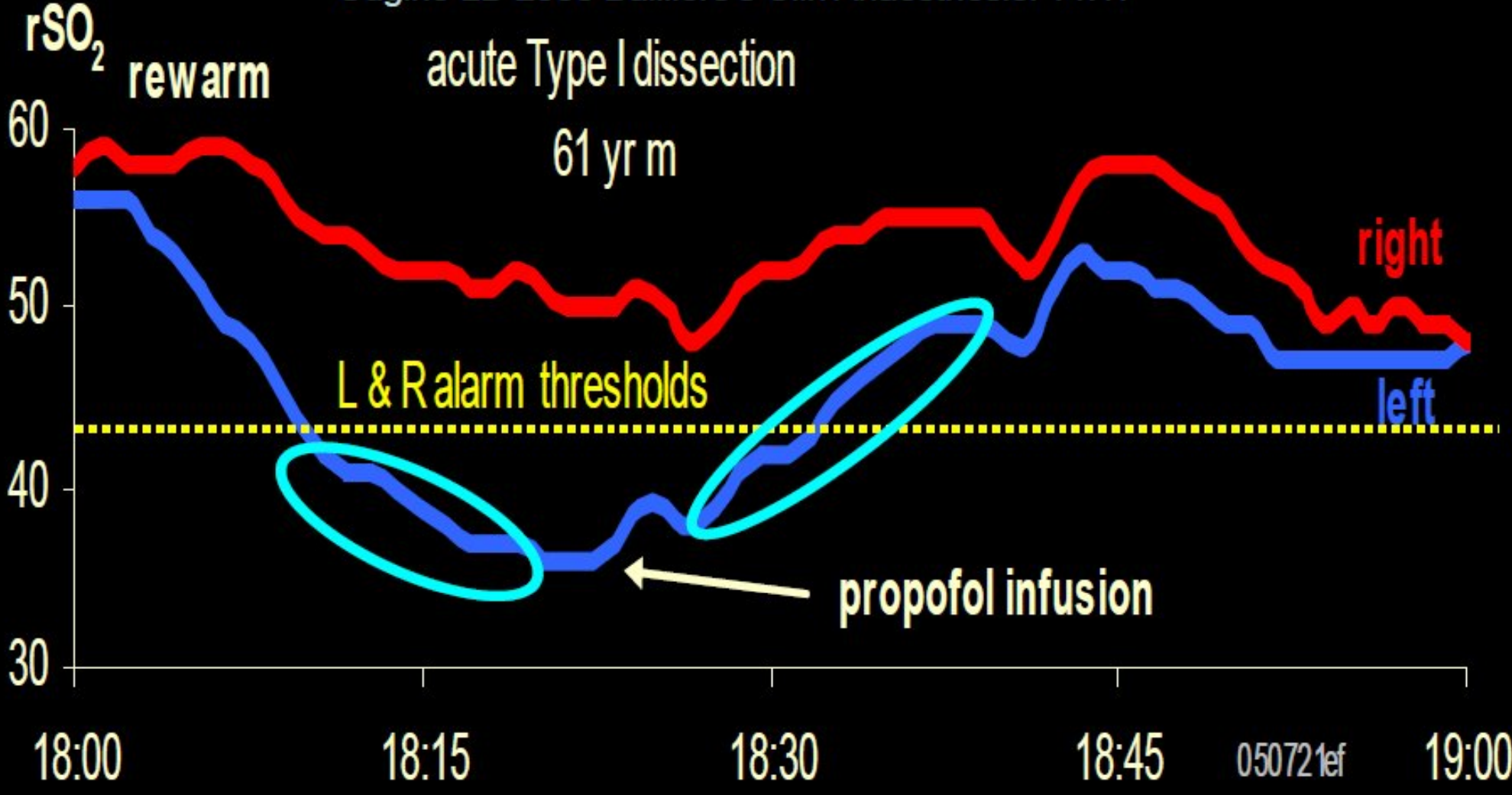
With CO_2-reactive arterioles, a shift from alpha-stat to pH-stat may improve brain cooling & oxygenation.

du Plessis AJ 1997 J Thorac Cardiovasc Surg 114: 991



Hypothermic arrest may cause vasoneural uncoupling & result in ischemic rewarming.

Gugino LD 2000 Baillière's Clin Anaesthesiol 14:17

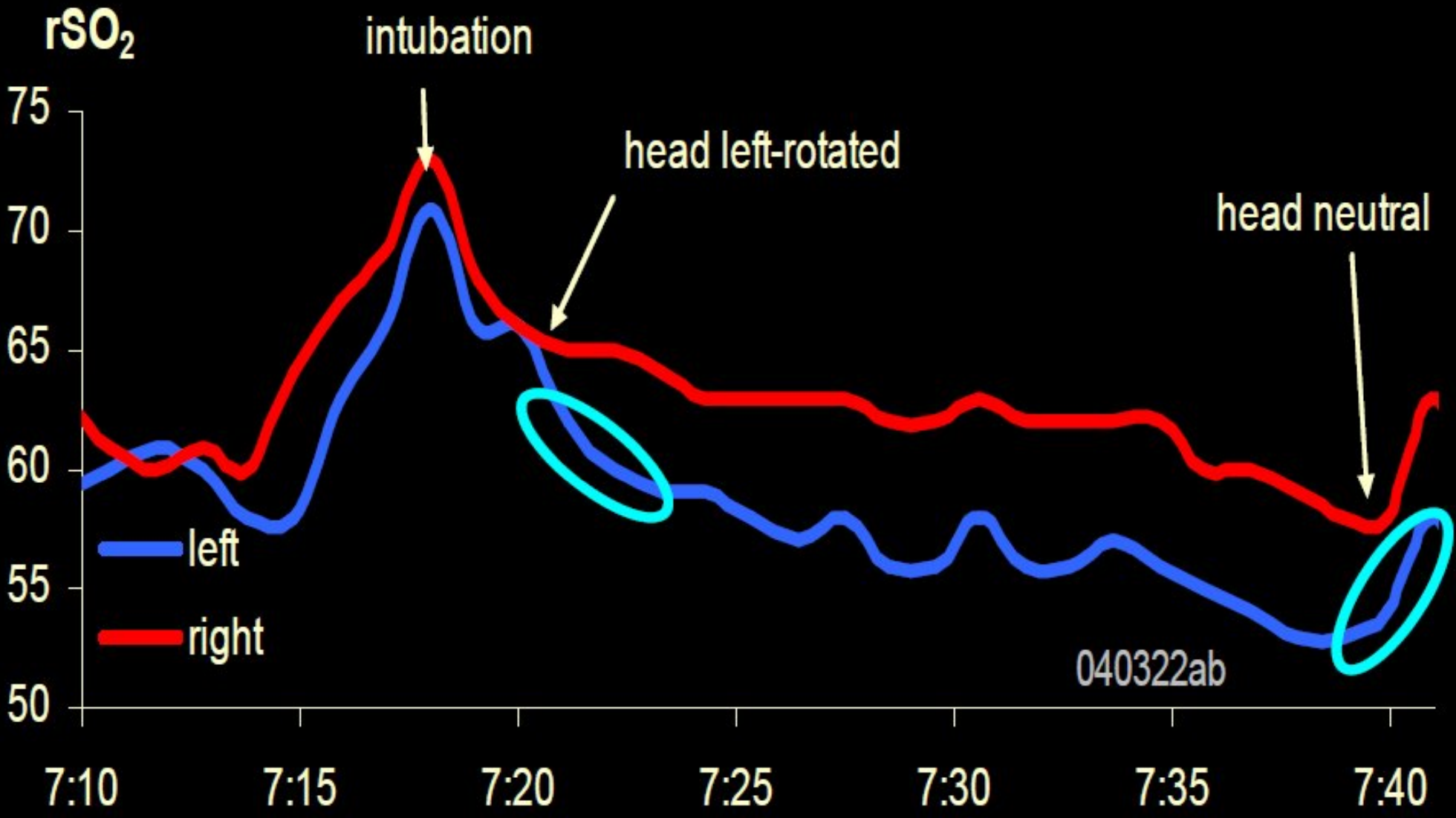


Propofol increases rSO_2 by suppressing $CMRO_2$.

McCulloch TJ 2007 Anesthesiology 106:56

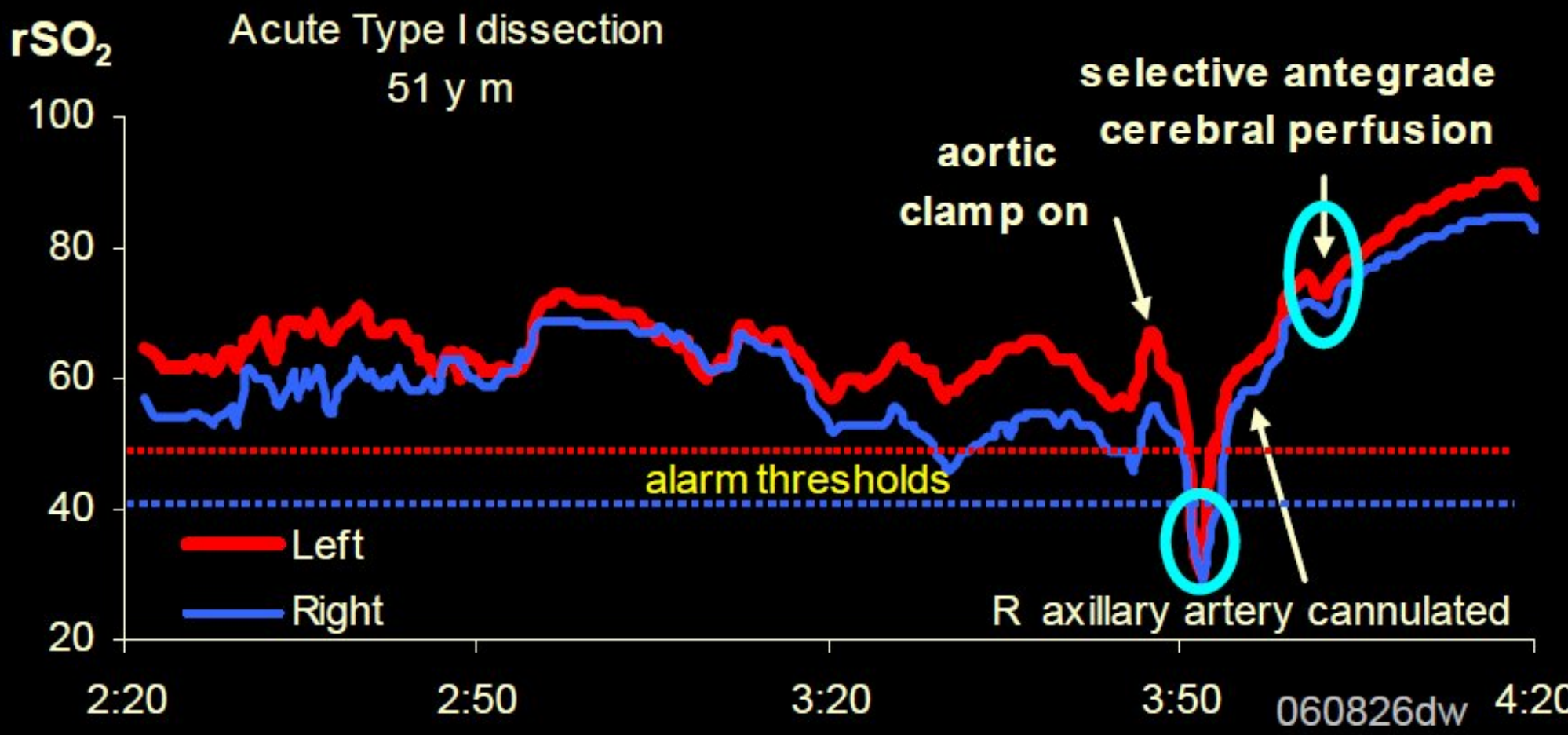
Patient head malposition may result in asymmetric desaturation.

Edmonds HL Jr 2004 Semin Cardiothorac Vasc Anesth 8:147



During acute aortic dissection repair, rSO_2 detects malperfusion at aortic clamping.

Fukada J 2003 Ann Thorac Surg 75:266



Evidenced-based Medicine:
Randomized Clinical Trial (RCT)
RCT is only method to prove cause & effect.

General Surgery

Casati A 2005 Anesth Analg 101:74

Cardiac Surgery

Baker RA 2006 J Extracorp Technol 8:77

Murkin JM 2007 Anesth Analg 104:51

A. Casati
Eur J Anaesthesiol. 2007 ;24:59-65

- Non vasküler abdominal cerrahi geçiren yaş >65 olan 60 hastada serebral desaturasyonu INVOS la izlemişler

- İntaoperatif;
  - 16/60 (%26) hastada desaturasyon gelişmiş,

- İntraoperatif rSO2 düşmesi ile POCD arasındaki ilişkiyi anlamlı bulmuşlar. OR:4,22, %95 CI:1,1-16, P=0,05

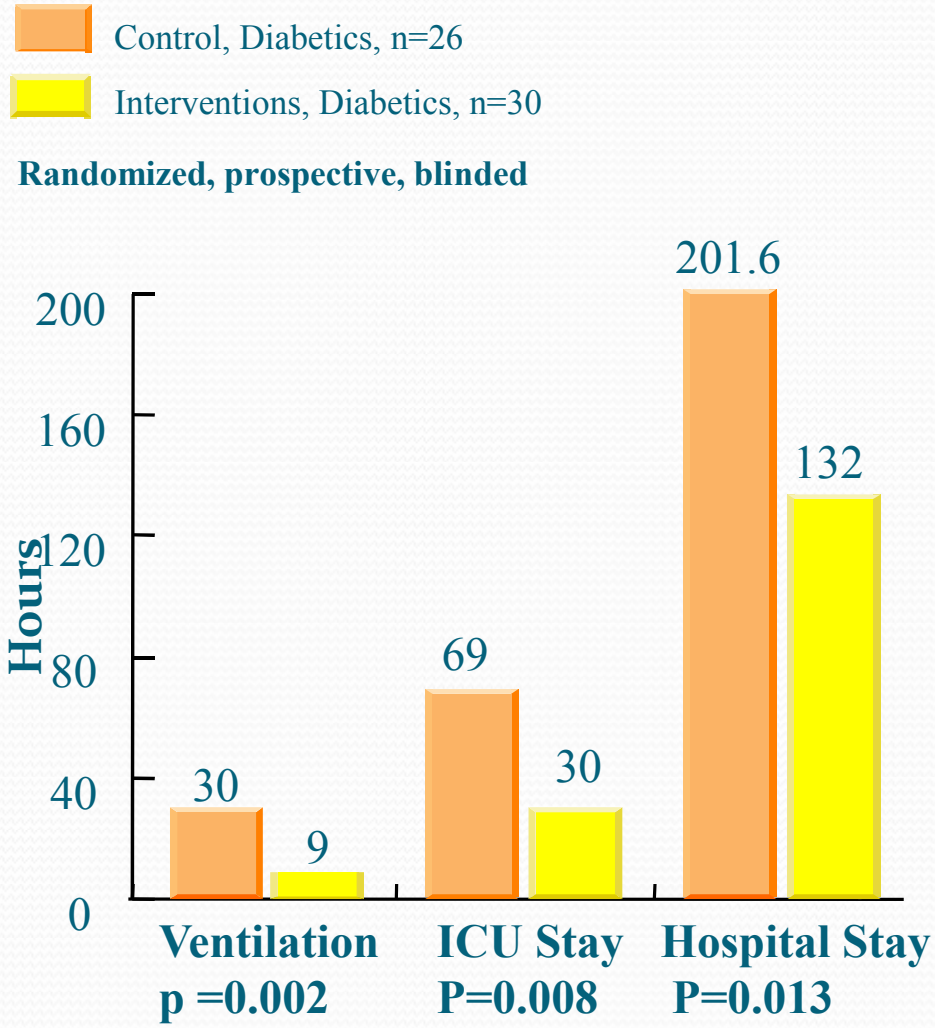
Murkin JM. Anesth Analg 2007;104:51-8 CABG

- Grup I: 100 rSO2 takibi yapılan (MAP,Fio2,PCO2,Flow,ES ,anestezi derinliği ve SVR ayarı yapılarak rSO2 değerinin bazal değerin en za %75 olması sağlanmış)
- Grup II: rSO2 takibi yapılmayan (kontrol grubu)

- Kontrol grubunda
  - Daha uzun ICU kalış (p<0,029)
  - Mojor Organ Morbidite Mortalitesinin daha fazla olduğu (p=0,017) tespit edilmiştir

ACIBADEM
ÜNİVERSİTESİ

Leveling the Playing Field for Diabetics

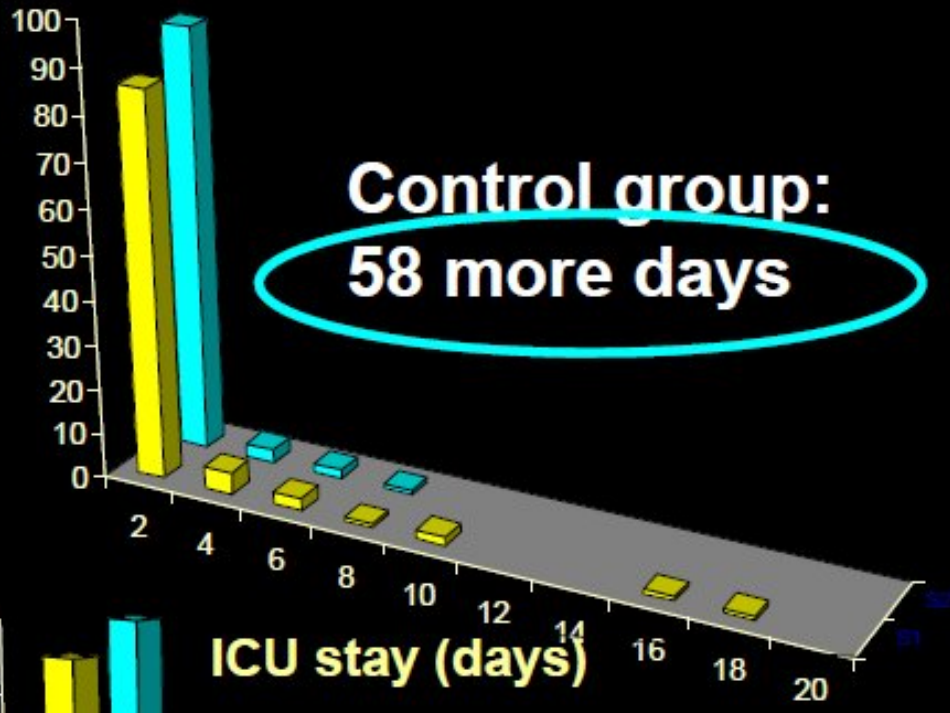


Diabetic cardiac surgery patients monitored with the INVOS System showed statistically-significant improvements over unmonitored diabetic patients.

Murkin JM; Iglesias I; Bainbridge D; Adams S; Schaffer B; et al. Anesth Analg 2005;100:SCA101.

Benefit of brain O_2 monitoring was <u>not</u> limited to stroke reduction.

Control group:
340 more hours



Control group:
58 more days

Control group:
82 more days

The greatest impact is on reduction in the # and severity of outliers.

Murkin JM et al. Anesth Analg 2007;104:51; Anesth Analg 2007;105:537

Correction of Brain O_2 Imbalance Decreased Hospital Cost of Care

post-op mechanical ventilation:

$63/hr

Dasta JF et al. 2005 Crit Care Med 33:1266

ICU without mechanical ventilation:

$3,250/day

Dasta JE et al. 2005 Crit Care Med 33:1266

hospital stay:

$539/day

AHA Hospital Statistics 2005

* $32,000 monitor cost amortized over 200 cases/yr x 5 yr

Oximetry Cost Reduction

	$/patient
Ventilation	215
ICU	1,885
Hospital stay	442
subtotal	2,542
- sensors	280
- hardware*	32
Net Savings	**$2,230**

Özet

1. Açık kalp cerrahisi sırasında beyin oksijen sunum/tüketim dengesizliği (İMBALANCE) yaygındır

2. rSo2 takibi ile beyindeki oksijen sunum/tüketim dengesizliği tespit edilir ve uygun tedaviye yönlendirilir

3. Uygun tedavi;

   Sonuç parametrelerini

   Hastanede kalış ve maliyeti olumlu yönde etkiler.