Perioperatif Organ İskemisi: Erişkinden Öğrendiklerimiz

Prof.Dr.Fevzi Toraman
Acıbadem Üniversitesi Anesteziyoloji ve Reanimasyon AD

GKDA ve YB 2015

Perioperatif organ iskemisi (Hasarı)

1. Durum tespiti
2. Serebral iskemi (Stroke: İnme)
3. Akut Böbrek hasarı
4. Akut Gastrointestinal sistem hasarı

A STUDY OF THE DEATHS ASSOCIATED WITH ANESTHESIA AND SURGERY*

BASED ON A STUDY OF 599,548 ANESTHESIAS IN TEN INSTITUTIONS 1948–1952, INCLUSIVE

HENRY K. BEECHER, M.D., AND DONALD P. TODD, M.D.

FROM THE ANESTHESIA DEPARTMENT OF THE HARVARD MEDICAL SCHOOL AT THE MASSACHUSETTS GENERAL HOSPITAL, BOSTON

Ann Surg. 1954 Jul;140(1):2-35.

Henry Beecher 1948-1952 yılları arasında anesteziye bağlı ölüm oranını 1/1560 olarak bulmuş

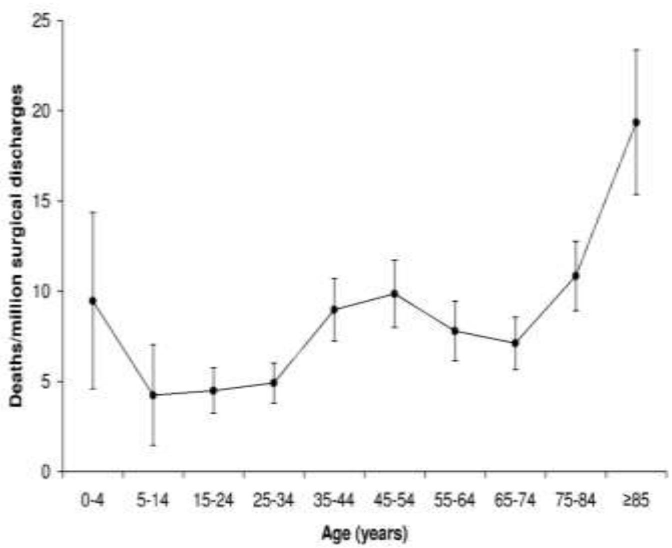
Ancak daha sonraki yıllarda yapılan çalışmalarda anesteziye bağlı ölüm insidansı daha düşük bulunmuştur

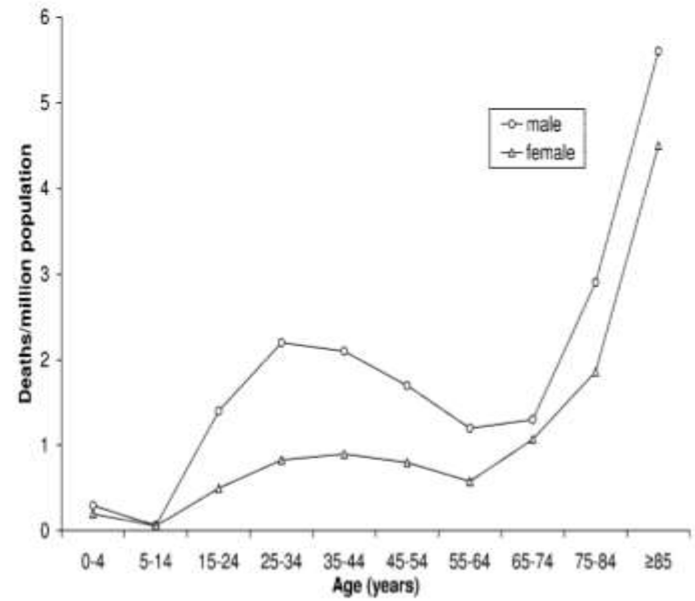
Anesthesiology 2009; 110:759-65

Epidemiology of Anesthesia-related Mortality in the United States, 1999–2005

Guohua Li, M.D., Dr.P.H., Margaret Warner, Ph.D.,† Barbara H. Lang, B.S.,‡ Lin Huang, M.S.,§ Lena S. Sun, M.D.||*

- 8,2/ Milyon





Acta Anaesthesiol Scand 2003; 47: 809–817

ACTA ANAESTHESIOLOGICA SCANDINAVICA
ISSN 0001-5172

Anesthesia-related mortality and morbidity over a 5-year period in 2,363,038 patients in Japan

- 21/milyon

Table 2A

Critical incidents other than cardiac arrest due to all etiologies during anesthesia and surgery, 1994–1998

	Serious hypotension	Serious hypoxemia	Others	Subtotal
5-year total	4696	1871	2073	8640
Incidence/ 10,000	19.17	7.67	8.35	35.19
95% CI	15.49~22.84	6.09~9.25	6.12~10.58	28.19~42.18

n = 2,363,038.
CI = confidence interval.

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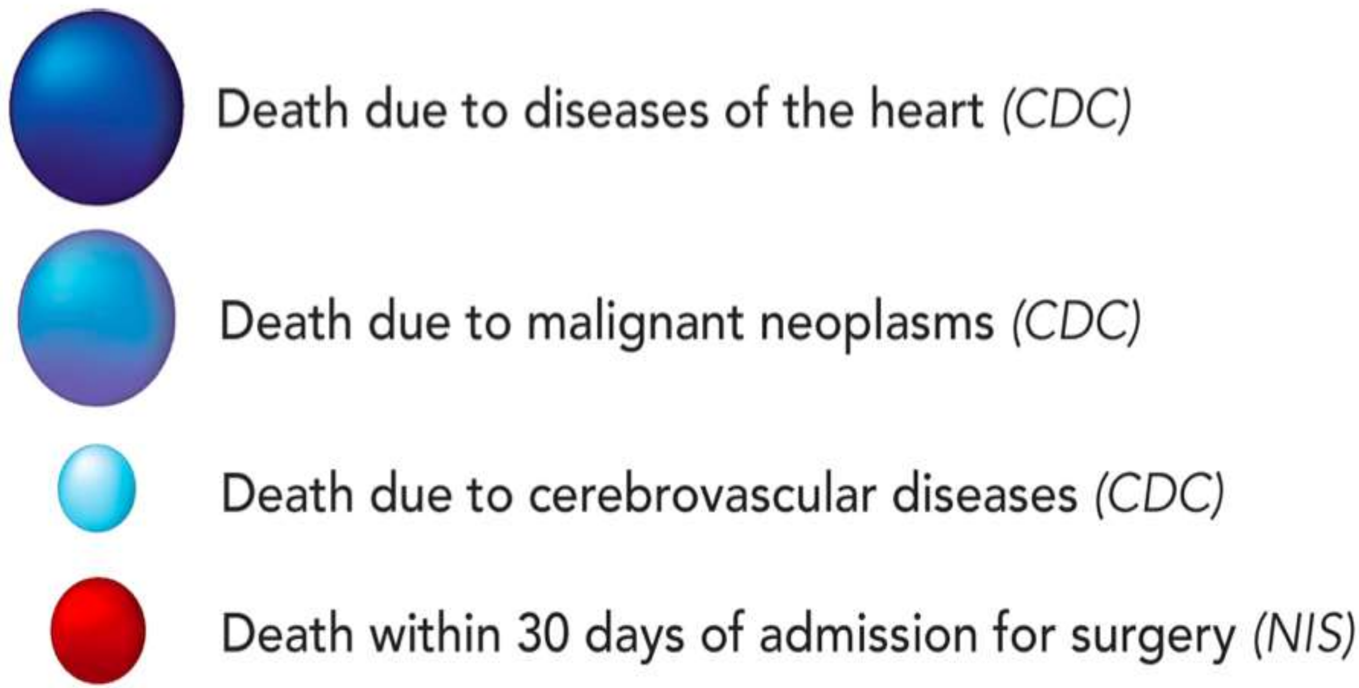

Figure 1. Magnitude of perioperative mortality

The 3 leading causes of death in the Center for Disease Control's (CDC) annual death table for the United States in 2006 were: #1. Diseases of heart (n=631,636), #2. Malignant neoplasms (n=559,888), and #3. Cerebrovascular diseases (n=137,119).* Using the Nationwide Inpatient Sample (NIS) for the same year, Gawande and colleagues reported 189,690 deaths within 30 days of admission for inpatients having a surgical procedure.[10] In magnitude, all-cause 30-day inpatient mortality following surgery approximated the third leading cause of death in the United States.

Stroke

1. Perioperatif hemodinamik instabiliteye ve

2. Cerrahi sırasındaki akut stres yanıta bağlı olarak
   - Nöroprotektif mekanizmanın bozulması ile
     - Hiperkoagülabilite
     - Nöroinflamasyon daki artama

meydana gelebilir

- Inflammation. 2012; 35:98–113.
- Anesthesiology. 2011; 115:879–890.

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

CURRENT CONCEPTS

Perioperative Stroke

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N Engl J Med 2007;356:706-13.

STROKE IS ONE OF THE MOST FEARED COMPLICATIONS OF SURGERY. TO PROvide adequate preventive and therapeutic measures, physicians need to be knowledgeable about the risk factors for stroke during the perioperative period. In this article, I review the pathophysiology of perioperative stroke and provide recommendations for the stratification of risk and the management of risk factors.

INCIDENCE

The incidence of perioperative stroke depends on the type and complexity of the surgical procedure. The risk of stroke after general, noncardiac procedures is very low. Cardiac and vascular surgeries — in particular, combined cardiac procedures — are associated with higher risks[1-7] (Table 1). The timing of surgery is also important. More strokes occur after urgent surgery than after elective surgery.[1]

Despite advances in surgical techniques and improvements in perioperative care, the incidence of perioperative strokes has not decreased, reflecting the aging of the population and the increased number of elderly patients with coexisting conditions who undergo surgery. Perioperative strokes result in a prolonged hospital stay and increased rates of disability, discharge to long-term care facilities, and death after surgery.[7]

Table 1. **Incidence of Stroke after Various Surgical Procedures.**

Procedure	Risk of Stroke (%)
General surgery[2]	0.08–0.7
Peripheral vascular surgery[3]	0.8–3.0
Resection of head and neck tumors[4]	4.8
Carotid endarterectomy[5]	5.5–6.1
Isolated CABG[1,7]	1.4–3.8
Combined CABG and valve surgery[1,7]	7.4
Isolated valve surgery[1]	4.8–8.8
Double- or triple-valve surgery[1]	9.7
Aortic repair[7]	8.7

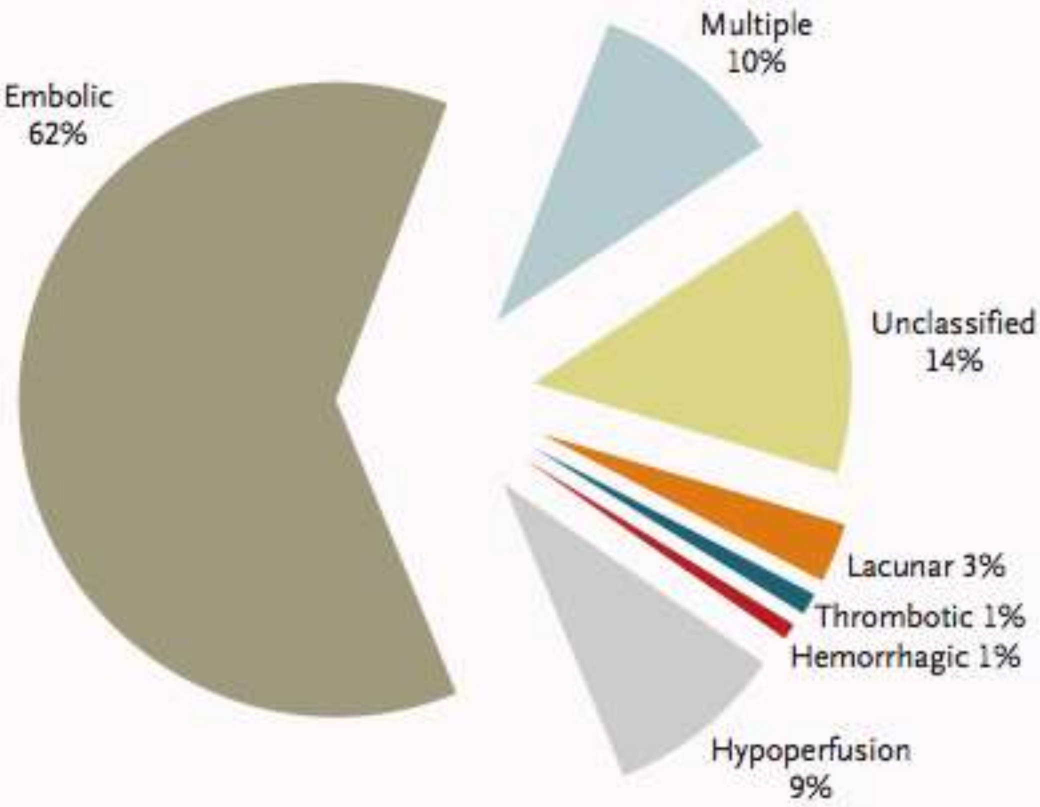

Figure 1. Mechanisms of Perioperative Stroke.
Data are from Likosky et al.[12]

Impaired Autoregulation of Cerebral Blood Flow During Rewarming from Hypothermic Cardiopulmonary Bypass and Its Potential Association with Stroke

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Charles W. Hogue, Jr., MD*

BACKGROUND: Patient rewarming after hypothermic cardiopulmonary bypass (CPB) has been linked to brain injury after cardiac surgery. In this study, we evaluated whether cooling and then rewarming of body temperature during CPB in adult patients is associated with alterations in cerebral blood flow (CBF)–blood pressure autoregulation.

METHODS: One hundred twenty-seven adult patients undergoing CPB during cardiac surgery had transcranial Doppler monitoring of the right and left middle cerebral artery blood flow velocity. Eleven patients undergoing CPB who had arterial inflow maintained at >35°C served as controls. The mean velocity index (Mx) was calculated as a moving, linear correlation coefficient between slow waves of middle cerebral artery blood flow velocity and mean arterial blood pressure. Intact CBF–blood pressure autoregulation is associated with an Mx that approaches 0. Impaired autoregulation results in an increasing Mx approaching 1.0. Comparisons of time-averaged Mx values were made between the following periods: before CPB (baseline), during the cooling and rewarming phases of CPB, and after CPB. The number of patients in each phase of CPB with an Mx >4.0, indicative of impaired CBF autoregulation, was determined.

RESULTS: During cooling, Mx (left, 0.29 ± 0.18; right, 0.28 ± 0.18 [mean ± SD]) was greater than that at baseline (left, 0.17 ± 0.21; right, 0.17 ± 0.20; $P \leq 0.0001$). Mx increased during the rewarming phase of CPB (left, 0.40 ± 0.19; right, 0.39 ± 0.19) compared with baseline ($P \leq 0.001$) and the cooling phase ($P \leq 0.0001$), indicating impaired CBF autoregulation. After CPB, Mx (left, 0.27 ± 0.20; right, 0.28 ± 0.21) was higher than at baseline (left, $P = 0.0004$; right, $P = 0.0003$), no different than during the cooling phase, but lower than during rewarming (left, $P \leq 0.0001$; right, $P \leq 0.0005$). Forty-three patients (34%) had an Mx ≥0.4 during the cooling phase of CPB and 68 (53%) had an average Mx ≥0.4 during rewarming. Nine of the 11 warm controls had an average Mx ≥0.4 during the entire CPB period. There were 7 strokes and 1 TIA after surgery. All strokes were in patients with Mx ≥ 0.4 during rewarming ($P = 0.015$). The unadjusted odds ratio for any neurologic event (stroke or transient ischemic attack) for patients with Mx ≥ 0.4 during rewarming was 6.57 (95% confidence interval, 0.79 to 55.0, $P < 0.08$).

CONCLUSIONS: Hypothermic CPB is associated with abnormal CBF–blood pressure autoregulation that is worsened with rewarming. We found a high rate of strokes in patients with evidence of impaired CBF autoregulation. Whether a pressure-passive CBF state during rewarming is associated with risk for ischemic brain injury requires further investigation.

(Anesth Analg 2010;110:321-8)

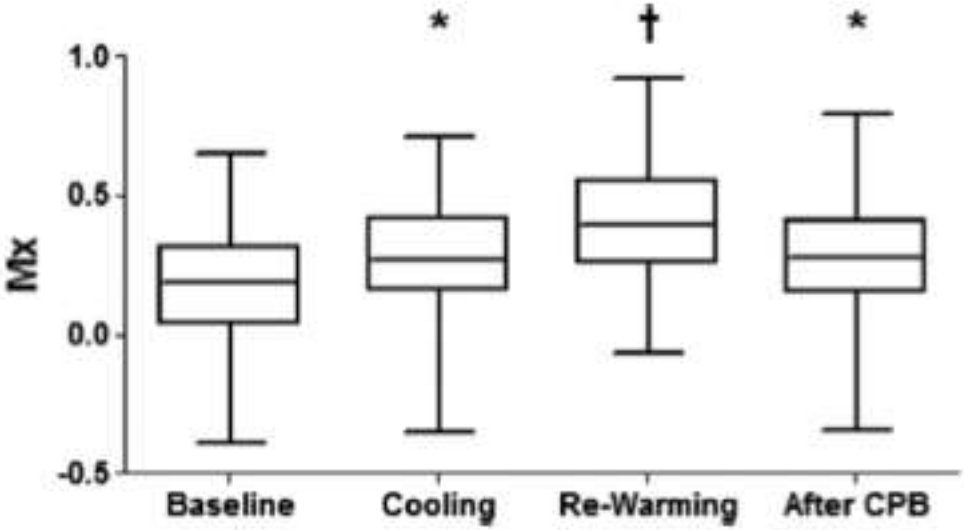

Figure 1. Mean velocity index (Mx) values obtained after anesthesia induction but before cardiopulmonary bypass (CPB) initiation (baseline) and during the cooling and re-warming phases of CPB. Mx is derived as the nonlinear correlation between cerebral blood flow (CBF) velocity of the right and left middle cerebral arteries and mean arterial blood pressure. This unitless measurement is obtained from 300-s windows of data that are updated every 10 s. Functional CBF autoregulation is indicated by values of Mx that approach 0; dysregulation is indicated by Mx values approaching 1.0. An Mx value between 0.3 and 0.5 is likely associated with autoregulation failure.[16–18] *$P \leq 0.001$ versus baseline; †$P \leq 0.0001$ versus cooling phase and baseline.

Table 5. Neurological Outcomes for Patients with and Without Impaired Cerebral Blood Flow Autoregulation During Rewarming on Cardiopulmonary Bypass

Outcome	No impairment ($n = 60$)	Impairment ($n = 67$)	P
Perioperative stroke	0	7 (10.4%)	0.015
Transient ischemic attack	1 (1.7%)	0	0.463

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Duration and magnitude of blood pressure below cerebral autoregulation threshold during cardiopulmonary bypass is associated with major morbidity and operative mortality

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Abstract

Objectives—Optimizing blood pressure using near-infrared spectroscopy monitoring has been suggested to ensure organ perfusion during cardiac surgery. Near-infrared spectroscopy is a reliable surrogate for cerebral blood flow in clinical cerebral autoregulation monitoring and might provide an earlier warning of malperfusion than indicators of cerebral ischemia. We hypothesized that blood pressure below the limits of cerebral autoregulation during cardiopulmonary bypass would be associated with major morbidity and operative mortality after cardiac surgery.

Methods—Autoregulation was monitored during cardiopulmonary bypass in 450 patients undergoing coronary artery bypass grafting and/or valve surgery. A continuous, moving Pearson's correlation coefficient was calculated between the arterial pressure and low-frequency near-infrared spectroscopy signals and displayed continuously during surgery using a laptop computer. The area under the curve of the product of the duration and magnitude of blood pressure below the limits of autoregulation was compared between patients with and without major morbidity (eg, stroke, renal failure, mechanical lung ventilation >48 hours, inotrope use >24 hours, or intra-aortic balloon pump insertion) or operative mortality.

Results—Of the 450 patients, 83 experienced major morbidity or operative mortality. The area under the curve of the product of the duration and magnitude of blood pressure below the limits of autoregulation was independently associated with major morbidity or operative mortality after cardiac surgery (odds ratio, 1.36; 95% confidence interval, 1.08–1.71; *P* = .008).

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BJA

Arterial pressure above the upper cerebral autoregulation limit during cardiopulmonary bypass is associated with postoperative delirium

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Editor's key points

- Cerebral hyperperfusion attributable to arterial pressure above an upper limit of autoregulation could contribute to delirium after cardiopulmonary bypass (CPB).
- Cerebral autoregulation was measured using cerebral oximetry, and postoperative delirium was prospectively assessed in cardiac surgery patients.
- Mean arterial pressure above the upper limit of cerebral autoregulation during CPB was associated with increased risk of delirium.

Background. Mean arterial pressure (MAP) below the lower limit of cerebral autoregulation during cardiopulmonary bypass (CPB) is associated with complications after cardiac surgery. However, simply raising empiric MAP targets during CPB might result in MAP above the upper limit of autoregulation (ULA), causing cerebral hyperperfusion in some patients and predisposing them to cerebral dysfunction after surgery. We hypothesized that MAP above an ULA during CPB is associated with postoperative delirium.

Methods. Autoregulation during CPB was monitored continuously in 491 patients with the cerebral oximetry index (COx) in this prospective observational study. COx represents Pearson's correlation coefficient between low-frequency changes in regional cerebral oxygen saturation (measured with near-infrared spectroscopy) and MAP. Delirium was defined throughout the postoperative hospitalization based on clinical detection with prospectively defined methods.

Results. Delirium was observed in 45 (9.2%) patients. Mechanical ventilation for >48 h [odds ratio (OR), 3.94; 95% confidence interval (CI), 1.72–9.03], preoperative antidepressant use (OR, 3.0; 95% CI, 1.29–6.96), prior stroke (OR, 2.79; 95% CI, 1.12–6.96), congestive heart failure (OR, 2.68; 95% CI, 1.28–5.62), the product of the magnitude and duration of MAP above an ULA (mm Hg h; OR, 1.09; 95% CI, 1.03–1.15), and age (per year of age; OR, 1.01; 95% CI, 1.01–1.07) were independently associated with postoperative delirium.

Conclusions. Excursions of MAP above the upper limit of cerebral autoregulation during CPB are associated with risk for delirium. Optimizing MAP during CPB to remain within the cerebral autoregulation range might reduce risk of delirium.

Clinical trial registration. clinicaltrials.gov NCT00769691 and NCT00981474.

Keywords: cardiac surgery; cardiopulmonary bypass; cerebral autoregulation; delirium

Accepted for publication: 25 June 2014

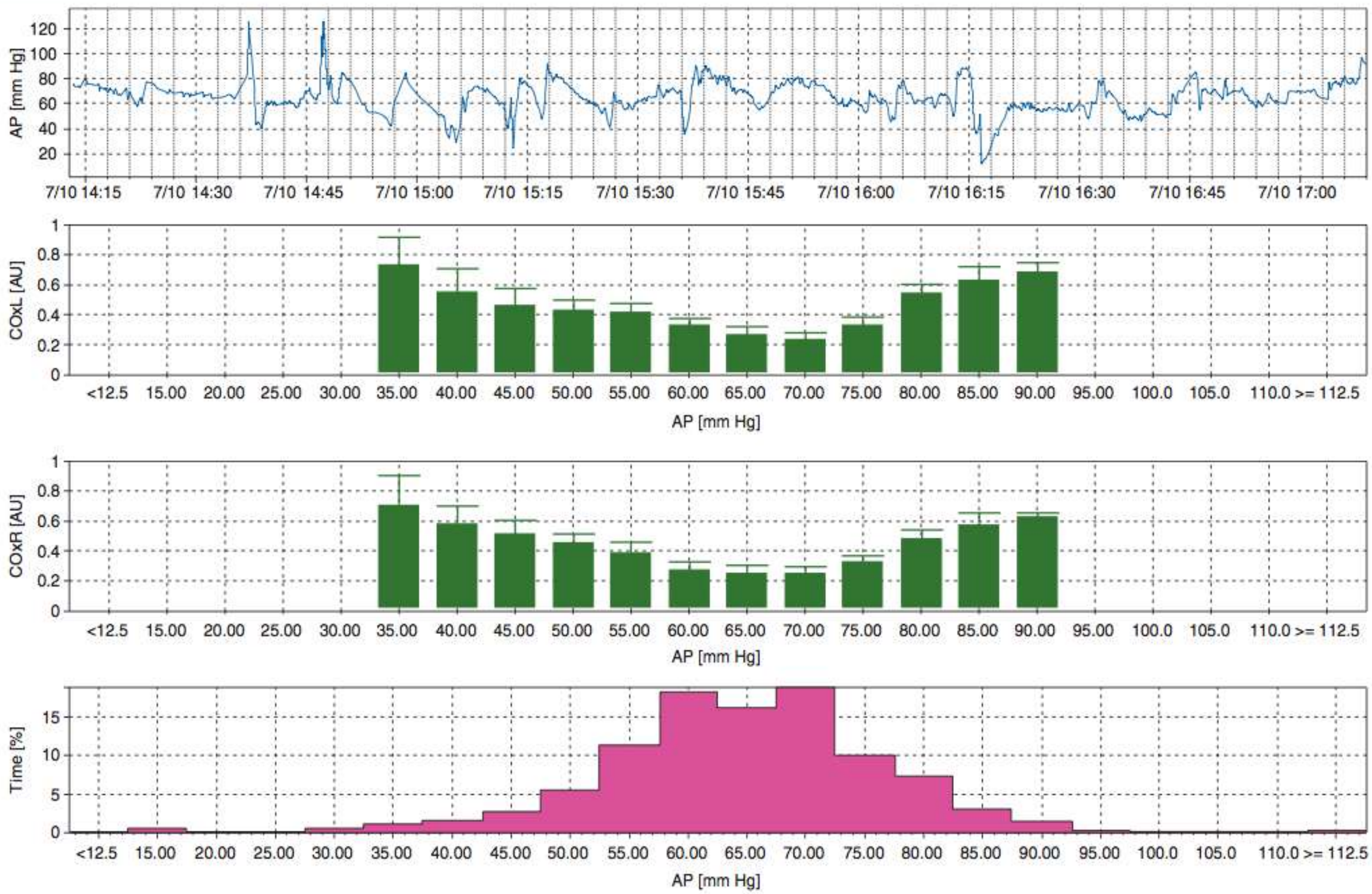

Fig 1 The representative graph of autoregulation monitoring during CPB. The COx represents the correlation coefficient between low-frequency regional cerebral oxygen saturation and MAP. When arterial pressure is above or below the autoregulation threshold, COx approaches 1, but when autoregulation is functional, COx is near zero. In this example, the lower limit of autoregulation based on the MAP at which COx $\geq$0.3 is ~55 mm Hg, and an ULA is at a MAP of 75 mm Hg. AP, arterial pressure.

Table 3 The product of the magnitude and duration of MAP above selected cutoffs (mm Hg h). *The cutoffs represent raw MAP irrespective of the upper limits of autoregulation. Data are presented as median (inter-quartile range)

MAP cutoff*	Delirium (n=45)	No delirium (n=446)	P-value
MAP >80 mm Hg	8.71 (3.66–15.27)	5.99 (2.90–11.50)	0.120
MAP >85 mm Hg	4.96 (1.45–9.69)	3.01 (1.18–6.76)	0.143
MAP >90 mm Hg	2.23 (0.44–4.80)	1.36 (0.44–3.39)	0.196
MAP >95 mm Hg	0.89 (0.13–2.40)	0.58 (0.10–1.66)	0.298
MAP >100 mm Hg	0.30 (0.05–0.86)	0.24 (0–0.75)	0.321
MAP >105 mm Hg	0.08 (0–0.28)	0.07 (0–0.31)	0.634

Sonuç

- Daha önce yapılan çalışmalarda serebral otoregülasyon alt sınırının altındaki bir OAB nın sonuç parametrelerini olumsuz etkilediği gösterilmişti

- Bu çalışmada da otoregülasyon üst sınırının üstündeki bir OAB nında benzer etkiler oluşturuğu görülmüştür.

- Bu nedenle serebral otoregülasyonun NIRS ile izlenerek uygun OAB nın sağlanması çok önemlidir.

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BJA

Influence of variations in systemic blood flow and pressure on cerebral and systemic oxygen saturation in cardiopulmonary bypass patients

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Editor's key points

- Maintenance of adequate tissue perfusion and oxygenation is important during anaesthesia.
- In patients undergoing cardiopulmonary bypass, the authors independently manipulated blood flow and systemic arterial pressure.
- Cerebral and systemic oxygenation were positively correlated with flow but not with pressure.

Background. Although both pressure and flow are considered important determinants of regional organ perfusion, the relative importance of each is less established. The aim of the present study was to evaluate the impact of variations in flow, pressure, or both on cerebral and whole-body oxygen saturation.

Methods. Thirty-four consenting patients undergoing elective cardiac surgery on cardiopulmonary bypass were included. Using a randomized cross-over design, four different haemodynamic states were simulated: (i) 20% flow decrease, (ii) 20% flow decrease with phenylephrine to restore baseline pressure, (iii) 20% pressure decrease with sodium nitroprusside (SNP) under baseline flow, and (iv) increased flow with baseline pressure. The effect of these changes was evaluated on cerebral (Sc_{O_2}) and systemic (Sv_{O_2}) oxygen saturation, and on systemic oxygen extraction ratio (OER). Data were assessed by within- and between-group comparisons.

Results. Decrease in flow was associated with a decrease in Sc_{O_2} [from 63.5 (7.4) to 62.0 (8.5) %, $P<0.001$]. When arterial pressure was restored with phenylephrine during low flow, Sc_{O_2} further decreased from 61.0 (9.7) to 59.2 (10.2) %, $P<0.001$. Increase in flow was associated with an increase in Sc_{O_2} from 62.6 (7.7) to 63.6 (8.9) %, $P=0.03$, while decreases in pressure with the use of SNP did not affect Sc_{O_2}. Sv_{O_2} was significantly lower ($P<0.001$) and OER was significantly higher ($P<0.001$) in the low flow arms.

Conclusions. In the present elective cardiac surgery population, Sc_{O_2} and Sv_{O_2} were significantly lower with lower flow, regardless of systemic arterial pressure. Moreover, phenylephrine administration was associated with a reduced cerebral and systemic oxygen saturation.

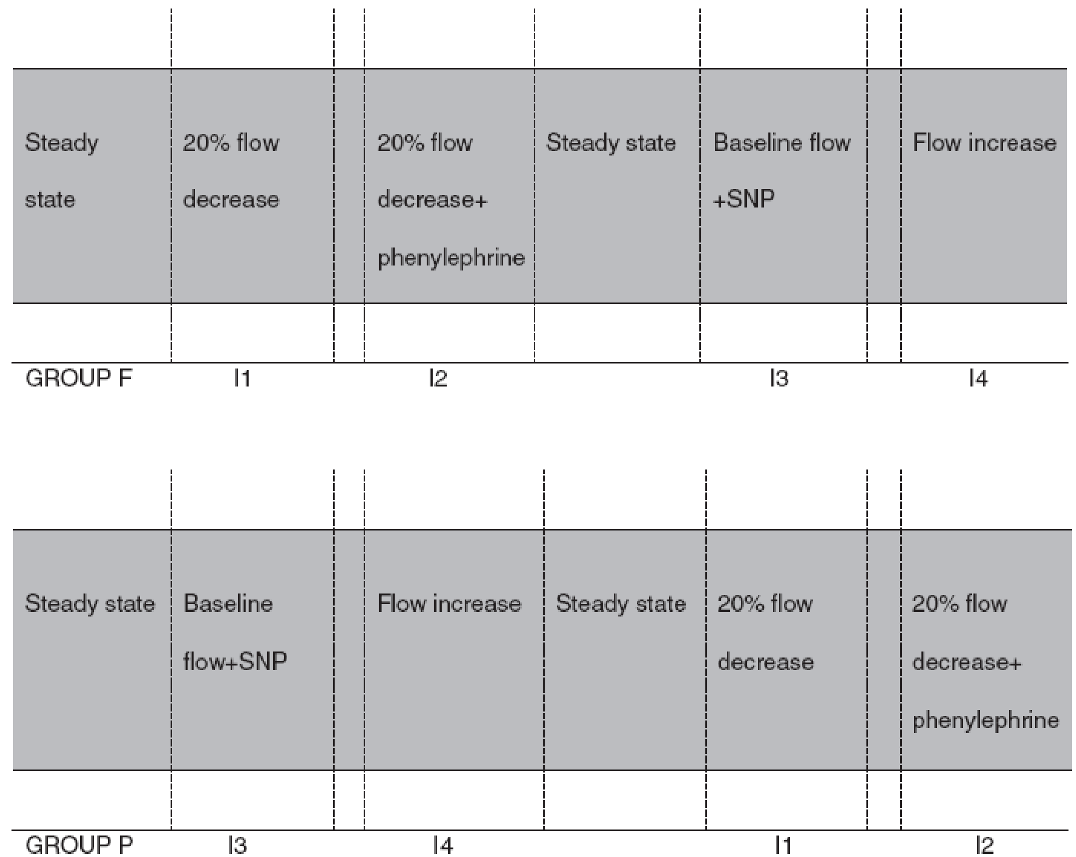

Fig 1 Graphic presentation of the study design. Sequence of interventions in Group F (first the flow related interventions) and Group P (first the pressure related interventions).

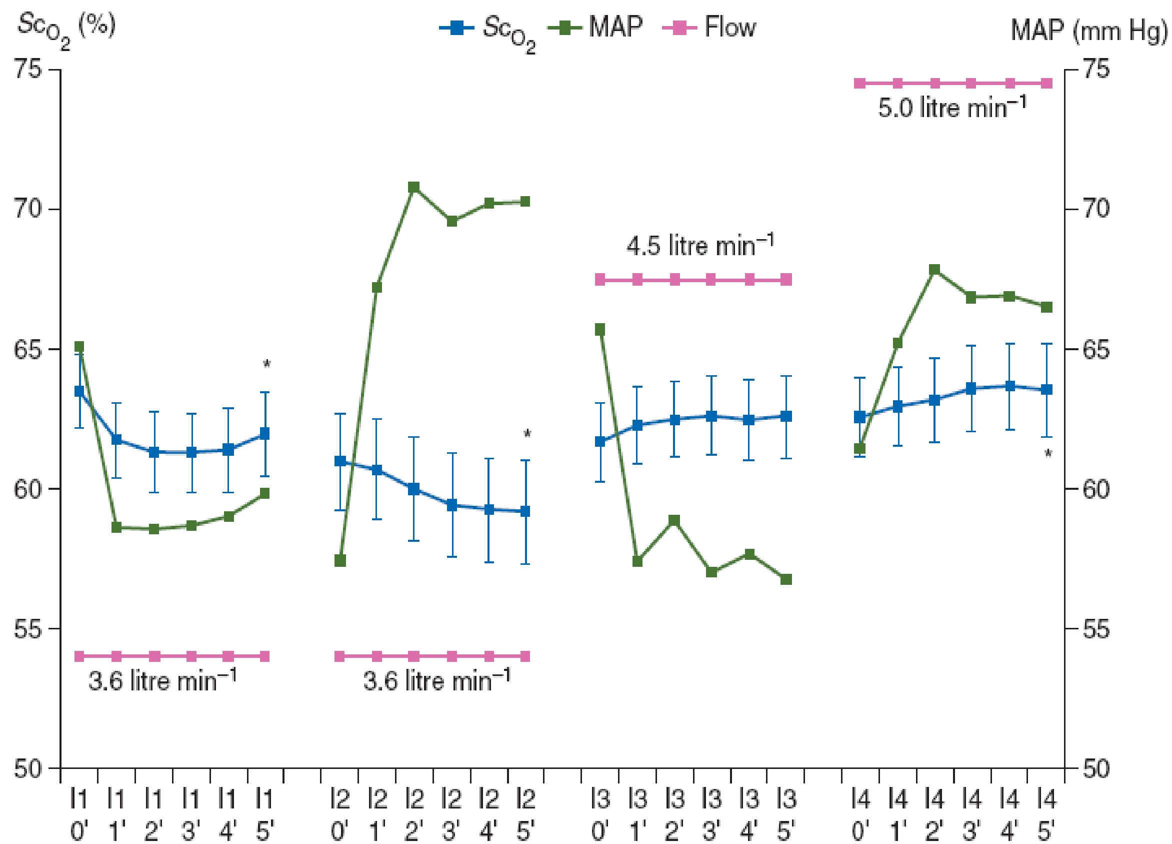

Fig 3 Mean changes in MAP and flow with their concomitant effects on Sc_{O_2} for each intervention: I1, 20% flow decrease; I2, 20% flow decrease with administration of phenylephrine to restore baseline MAP; I3, baseline flow with administration of SNP until 20% MAP decrease; I4, restoration of baseline MAP by increasing pump flow. MAP and flow data are presented as mean. Sc_{O_2} data are presented as mean and standard error. *$P<0.05$ between pre- and post-intervention Sc_{O_2}.

In conclusion, in the elective cardiac surgery population used in this study, changes in flow affected cerebral and systemic oxygen balance more than changes in pressure. Moreover, arterial pressure increase with phenylephrine elicited reduced cerebral and systemic oxygen saturation.

Blood Pressure Excursions Below the Cerebral Autoregulation Threshold During Cardiac Surgery are Associated With Acute Kidney Injury*

Objectives: To determine whether mean arterial blood pressure excursions below the lower limit of cerebral blood flow autoregulation during cardiopulmonary bypass are associated with acute kidney injury after surgery.

Setting: Tertiary care medical center.

Patients: Four hundred ten patients undergoing cardiac surgery with cardiopulmonary bypass.

Design: Prospective observational study.

Interventions: None.

Measurements and Main Results: Autoregulation was monitored during cardiopulmonary bypass by calculating a continuous, moving Pearson's correlation coefficient between mean arterial blood pressure and processed near-infrared spectroscopy signals to generate the variable cerebral oximetry index. When mean arterial blood pressure is below the lower limit of autoregulation, cerebral oximetry index approaches 1, because cerebral blood flow is pressure passive. An identifiable lower limit of autoregulation was ascertained in 348 patients. Based on the RIFLE criteria (Risk, Injury, Failure, Loss of kidney function, End-stage renal disease), acute kidney injury developed within 7 days of surgery in 121 (34.8%) of these patients. Although the average mean arterial blood pressure during cardiopulmonary bypass did not differ, the mean arterial blood pressure at the limit of autoregulation and the duration and degree to which mean arterial blood pressure was below the autoregulation threshold (mm Hg × min/hr of cardiopulmonary bypass) were both higher in patients with acute kidney injury than in those without acute kidney injury. Excursions of mean arterial blood pressure below the lower limit of autoregulation (relative risk 1.02; 95% confidence interval 1.01 to 1.03; $p < 0.0001$) and diabetes (relative risk 1.78; 95% confidence interval 1.27 to 2.50; $p = 0.001$) were independently associated with for acute kidney injury.

Conclusions: Excursions of mean arterial blood pressure below the limit of autoregulation and not absolute mean arterial blood pressure are independently associated with for acute kidney injury. Monitoring cerebral oximetry index may provide a novel method for precisely guiding mean arterial blood pressure targets during cardiopulmonary bypass. (*Crit Care Med* 2013; 41:464–471)

Key Words: acute kidney injury; blood pressure; cardiac surgery; cerebral autoregulation

TABLE 3. Near-Infrared Spectroscopy and Cerebral Autoregulation Data For Patients With and Without Acute Kidney Injury After Surgery[a]

	AKI ($n = 121$)	No AKI ($n = 227$)	p
Average regional cerebral oxygen saturation	53 ± 11 (50 to 55)	54 ± 11 (53 to 56)	0.298
Average cerebral oximetry index	0.26 ± 0.17 (0.23 to 0.30)	0.26 ± 0.19 (0.23 to 0.28)	0.820
Average MAP during cardiopulmonary bypass (mm Hg)	75 ± 7 (74 to 76)	74 ± 8 (73 to 75)	0.103
Lower limit of autoregulation (mm Hg)	69 ± 16 (66 to 72)	63 ± 15 (61 to 65)	0.001
Magnitude of MAP ≤ lower limit of autoregulation (mm Hg × min/hr)	11.2 ± 12.4 (7.8 to 13.0)	6.6 ± 7.2 (5.7 to 7.9)	0.014
pH	7.39 ± 0.03	7.39 ± 0.03	0.9179
Pa_{CO_2} (mm Hg)	40 ± 3	41 ± 3	0.2670
Pa_{O_2} (mm Hg)	262 ± 44	261 ± 47	0.7746
Hemoglobin (g/dL)	8.9 ± 1.2	9.3 ± 1.8	0.0369
Average temperature (mean ± SD)	33.8 ± 1.5°C	33.8 ± 2.5°C	0.6170
Peak temperature during rewarming	34.5 ± 2.0°C	34.5 ± 2.0°C	0.8758

AKI = acute kidney injury; MAP = mean arterial pressure.

[a]Values are given as means ± SD with 95% confidence intervals in parenthesis.

TABLE 4. Variables Independently Associated With Acute Kidney Injury Based on the Generalized Linear Model With Poisson Distribution and Robust Standard Errors

Variable	Relative Risk	95% Confidence Interval	*p*
Magnitude of mean arterial pressure ≤ lower limit of autoregulation (mm Hg × min/hr)	1.02	1.01 to 1.03	<0.0001
Diabetes	1.78	1.27 to 2.51	0.001
Pulse pressure > 60 mm Hg	1.33	0.89 to 1.99	0.158

CONCLUSIONS

Excursions of MAP below the lower limit of CBF autoregulation during CPB were independently associated with AKI. Monitoring autoregulation with processed NIRS signals may provide a novel method for precisely determining MAP targets during CPB.

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

CURRENT CONCEPTS

Normotensive Ischemic Acute Renal Failure

J. Gary Abuelo, M.D.

N Engl J Med 2007;357:797-805.

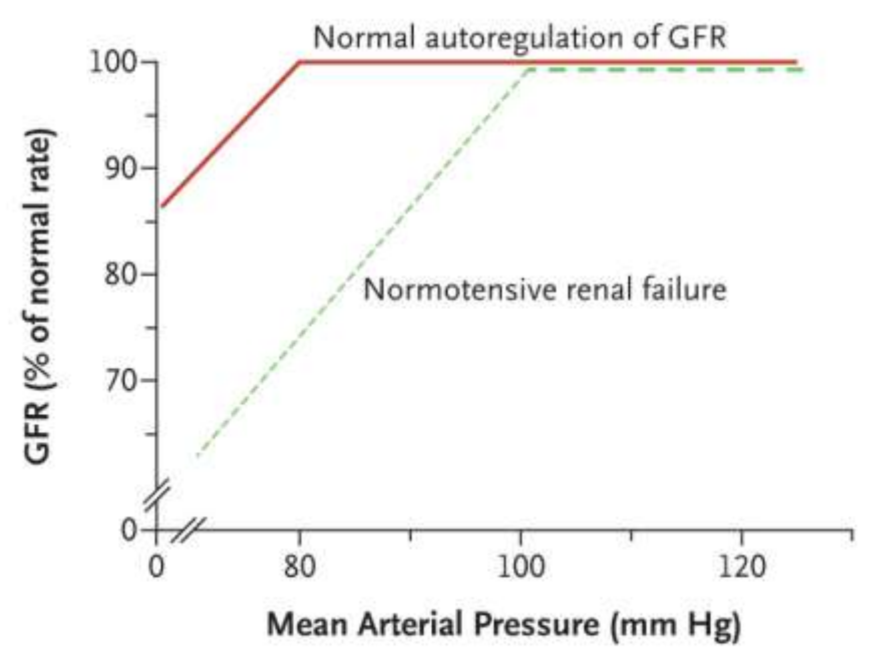

Figure 1. **Normal and Impaired Autoregulation of the Glomerular Filtration Rate during Reduction of Mean Arterial Pressure.**

Perfusion
Perfusion
2014, Vol. 29(6) 496–504

DOI: 10.1177/0267659114527331
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Increasing mean arterial pressure during cardiac surgery does not reduce the rate of postoperative acute kidney injury

A Azau,[1] P Markowicz,[1] JJ Corbeau,[1] C Cottineau,[1] X Moreau,[1] C Baufreton[2] and L Beydon[1]

Abstract

Introduction: We hypothesized that the optimization of renal haemodynamics by maintaining a high level of mean arterial blood pressure (MAP) during cardiopulmonary bypass (CPB) could reduce the rate of acute kidney injury (AKI) in high-risk patients.
Methods: In this randomized, controlled study, we enrolled 300 patients scheduled for elective cardiac surgery under cardiopulmonary bypass. All had known risk factors of AKI: serum creatinine clearance between 30 and 60 ml/min for $1.73m^2$ or two factors among the following: age >60 years, diabetes mellitus, diffuse atherosclerosis. After a standardized fluid loading, the MAP was maintained between 75-85 mmHg during CPB with norepinephrine (High Pressure, n=147) versus 50-60 mmHg in the Control (n=145). AKI was defined by a 30% increased of serum creatinine (sCr). We further tested others definitions for AKI: RIFLE classification, 50% rise of sCr and the need for haemodialysis.
Results: The pressure endpoints were achieved in both the High Pressure (79 ± 6 mmHg) and the Control groups (60 ± 6 mmHg; p<0.001). The rate of AKI did not differ by group (17% vs. 17%; p=1), whatever the criteria used for AKI. The length of stay in hospital (9.5 days [7.9-11.2] vs. 8.2 [7.1-9.4]) and the rate of death at day 28 (2.1% vs. 3.4%) and at six months (3.4% vs. 4.8%) did not differ between the groups.
Conclusion: Maintaining a high level of MAP (on average) during normothermic CPB does not reduce the risk of postoperative AKI. It does not alter the length of hospital stay or the mortality rate.

Table 4. Renal function.

	Control (n=145)	High-MAP (n=147)	p value
Preoperative renal function			
Creatinine (μmol/L)	95 ± 28	93 ± 25	0.53
Indexed creatinine clearance (ml/min/1.73m²)	61 ± 21	62 ± 23	0.91
BUN (mmol/l)	8.6 ± 3.5	8.5 ± 3.3	0.85
Postoperative renal function			
Renal resistivity index	0.69 ± 0.06	0.70 ± 0.06	0.40
Renal resistivity index > 0.74 n (%)	21 (21.6)	23 (25.6)	0.53
Mean hourly diuresis (ml/kg/h)	0.95 ± 0.87	0.84 ± 0.52	0.20
Serum creatinine peak (μmolL)	108 ± 52	105 ± 50	0.73
Time of occurrence for the serum creatinine peak (h)	13.6 ± 19.1	14.2 ± 20.6	0.8
Administration of diuretics, n [%]	29 [20]	25 [17]	0.6
AKI according to 30% rise in serum creatinine, n [%]	24 [16.6]	25 [17.0]	1
AKI according to 50% rise in serum creatinine, n [%]	13 [9]	13 [8.8]	1
Classified as RIFLE "risk", n [%]	80 [55]	92 [63]	0.2
Classified as RIFLE "injury", n [%]	15 [10]	14 [10]	0.8
Number of patients requiring haemodialysis, n [%]	4 [2.8]	6 [4.1]	0.8
Number of dialysis/patient in ICU when required	3.0 ± 2.8	3.2 ± 1.2	0.90

BUN: blood urea nitrogen; ICU: intensive care unit; AKI: acute kidney injury; RIFLE classification: Risk, Injury, Failure, Loss, End-stage kidney disease.

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DOI 10.1007/s00246-010-9839-x

ORIGINAL ARTICLE

Low Renal Oximetry Correlates With Acute Kidney Injury After Infant Cardiac Surgery

Gabe E. Owens · Karen King · James G. Gurney ·
John R. Charpie

Table 3 Incidence of acute kidney injury (AKI) and evaluation of secondary clinical variables between low and normal oximetry

	Low oximetry ($n = 8$)	Normal oximetry ($n = 32$)	p value[a]
AKI (pRIFLE): n (%)	4 (50)	1 (3.1)	0.003
AKI (ΔCrt >0.4 and >50%): n (%)	5 (63)	5 (16)	0.002
Renal replacement therapy: n (%)	0 (20)	0	0.12
Mechanical ventilation (days)	7.6 ± 3.6	4.2 ±2.9	0.008
Hospital length of stay (days)	15.4 ± 5.7	12.7 ± 11	0.51
Peak creatinine	0.83 ± 0.4	0.52 ± 0.2	0.003
Peak lactate	4.7 ± 4.2	2.9 ± 2.0	0.08
Average lactate	3.0 ± 2.5	1.5 ± 0.7	0.004
VIS peak	23.6 ± 17	13.8 ± 8.8	0.03

VIS vasoactive inotropic score

[a] p values are based on Student's t-test for continuous variables and Fisher's exact test for dichotomous variables. Continuous variables are reported as mean ± standard deviation, whereas dichotomous variables are displayed as number of patients and percentage

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DOI 10.1186/s13054-015-0760-9

CRITICAL CARE

RESEARCH Open Access

Intraoperative renal near-infrared spectroscopy indicates developing acute kidney injury in infants undergoing cardiac surgery with cardiopulmonary bypass: a case–control study

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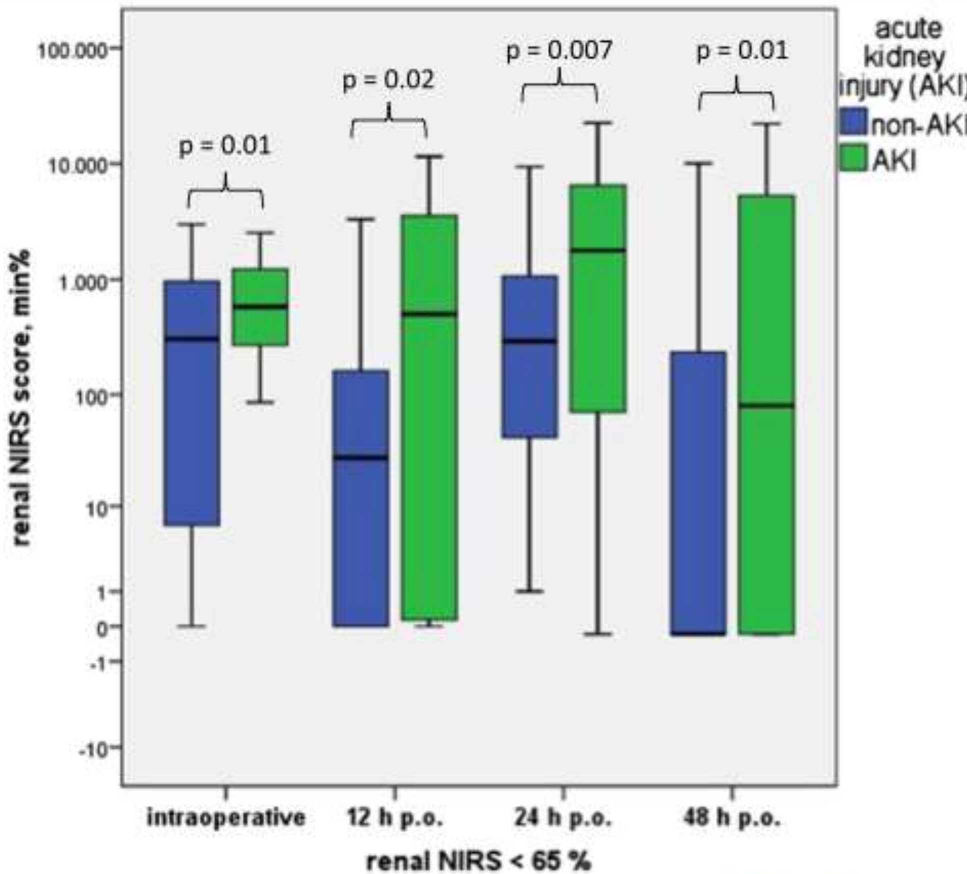

Figure 4 Renal near-infrared spectroscopy score in minute percent intraoperatively and 12, 24 and 48 hours postoperatively below the set baseline of regional oximetry <65% in infants with versus without acute kidney injury undergoing cardiopulmonary bypass operations. AKI, Acute kidney injury; NIRS, Near-infrared spectroscopy; p.o., Postoperatively.

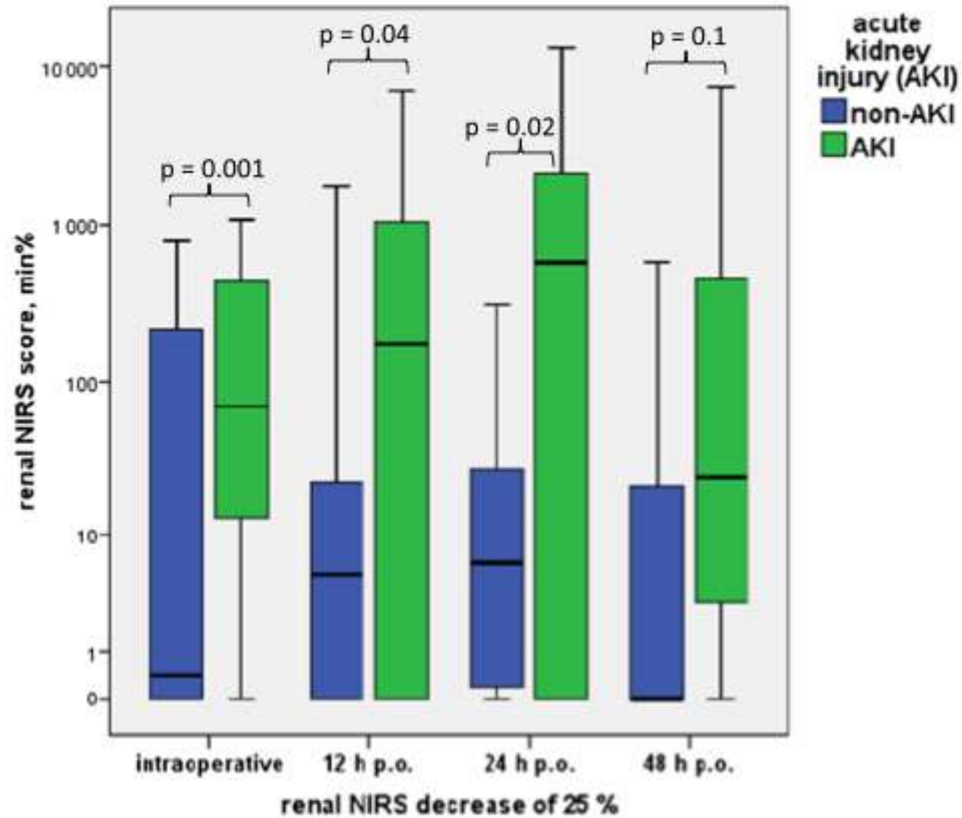

Figure 5 Renal near-infrared spectroscopy score in minute percent intraoperatively and at 12, 24 and 48 hours postoperatively below the set threshold regional oximetry decrease >25% of the preoperative averaged baseline value in infants with versus without acute kidney injury undergoing cardiopulmonary bypass operations. AKI, Acute kidney injury; NIRS, Near-infrared spectroscopy; p.o., Postoperatively.

Conclusion

Renal NIRS, when performed during and after infant cardiac surgery, is a promising tool to detect early haemodynamic compromise and predict development of AKI. Renal NIRS monitoring therefore should be included in prognostic models for early identification of renal injury risk in infants during and after CPB operation and may allow the development of therapeutic strategies to avoid kidney injury during cardiac surgery in infants.

The Role of Intraoperative Regional Oxygen Saturation Using Near Infrared Spectroscopy in the Prediction of Low Output Syndrome After Pediatric Heart Surgery

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Demetrio Pittarello, M.D.,* and Giovanni Stellin, M.D.†

ABSTRACT *Background:* We report on the applicability of intraoperative regional oxygen saturation (rSO_2) desaturation score by near-infrared spectroscopy in the early detection of postoperative low output state (LOS) in infants with congenital heart disease who underwent cardiac surgery. *Materials and Methods:* Between July and October 2011 the intra- and immediate postoperative courses of 22 patients undergoing elective cardiac surgery for congenital heart disease were analyzed. The intraoperative cerebral and somatic rSO_2 were measured and a rSO_2 desaturation score calculated (by multiplying the rSO_2 below 50% of the threshold by seconds). The aim of the study was to evaluate the applicability of intraoperative rSO_2 de-saturation score in the early detection of postoperative LOS. *Results:* Thirteen of 22 patients (62%) had an intraoperative cerebral rSO_2 desaturation score >3000% per second. Patients with a rSO_2 de-saturation score >3000% per second had a significantly lower intraoperative central venous saturation (SvO_2, $p = 0.002$), cardiac index (CI, $p = 0.004$), oxygen availability indexed (DO_2I, $p = 0.0004$), and a significantly higher extraction of oxygen (ERO_2, $p = 0.0005$) when compared to patients with a rSO_2 desaturation score <3000% per second. Nine patients had postoperative LOS; all of them had an intraoperative rSO_2 de-saturation score >3000% per second (9/13 patients, 69%; $p = 0.001$) requiring prompt treatment with major inotropic support, surface hypothermia, and extracorporeal membrane oxygenation (ECMO) support ($n = 4$). Twenty-one patients survived. One patient died from ventricular failure and inability to wean from ECMO support. *Conclusion:* The intraoperative use of NIRS provided an early warning sign of hemodynamic or metabolic compromise, enabling early and rapid intervention to prevent or reduce the severity of potentially life-threatening complications. doi: 10.1111/jocs.12122 *(J Card Surg 2013;28:446–452)*

TABLE 1
Preoperative Patient Profile and Hemodynamics

Patient's Characteristics, Vital Signs and Arterial Blood Gas Values at Anesthetic Induction	n = 22	rSO_2 De-Sat Score <3000%/sec (n = 9)	rSO_2 De-Sat Score >3000%/sec (n = 13)	P
Gender (F:M)	10:12	4:5	6:7	n/s
Age (months)	2.7 ± 3.6	1.4 ± 2.1	3.6 ± 4	n/s
Weight (kg)	3.8 ± 2.6	3.5 ± 1.2	3.9 ± 1.3	n/s
Height (cm)	54.6 ± 1.4	52.2 ± 6.3	55.2 ± 8,1	n/s
Body surface area (m^2)	0.23 ± 0	0.2 ± 0	0.2 ± 0	n/s
Body mass index (kg/m^2)	12.5 ± 1.4	12.7 ± 1.5	12.3 ± 1.4	n/s
Mean arterial pressure (mmHg)	55.6 ± 7.6	55.2 ± 7.8	55.8 ± 7.9	n/s
Heart rate (beats/min)	139 ± 18.4	140 ± 15.8	137 ± 20.6	n/s
Central venous pressure (mmHg)	8.1 ± 1.9	8.4 ± 1.7	8.3 ± 1.7	n/s
Rectal temperature (°C)	35.6 ± 0.9	35.4 ± 0.9	35.7 ± 0.8	n/s
SaO_2 (%)	94.7 ± 5.6	95.2 ± 3.2	94.2 ± 6.8	n/s
SvO_2 (%)	64.6 ± 5.6	65.2 ± 3.2	64.3 ± 6.9	n/s
PaO_2 (mmHg)	94.5 ± 64	80.9 ± 32.8	103.9 ± 78.9	n/s
$PaCO_2$ (mmHg)	36 ± 9.9	35.8 ± 11.1	36.1 ± 9.3	n/s
Hemoglobin level (g/dL)	10.8 ± 1.9	10.9 ± 1.5	10.7 ± 2.2	n/s
Lactate level (mg/dL)	18.9 ± 23.4	15.3 ± 8.1	20.7 ± 30.6	n/s
K^+ level (mg/dL)	13.2 ± 3.1	13.1 ± 3.1	13.6 ± 3.0	n/s
Glucose level (mg/dL)	95.4 ± 37.8	93.6 ± 34.2	95.4 ± 39.6	n/s

All values are mean ± SD. n/s, not significant; SD, standard deviation.

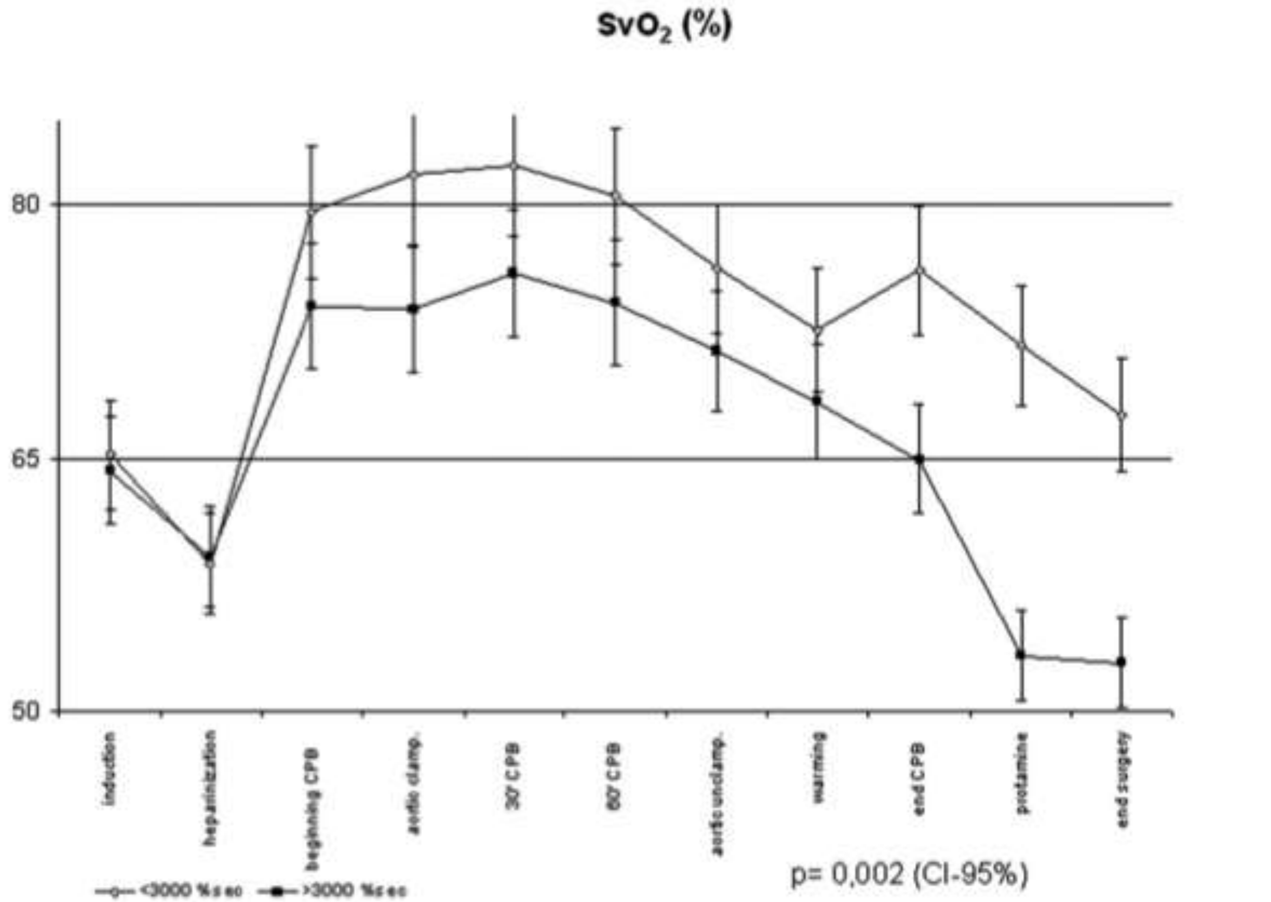

Figure 1. *Image showing the variation of intraoperative mixed venous saturation (SvO_2) between the two groups (rSO_2 de-saturation score < and >3000%/sec; see Table 2).*

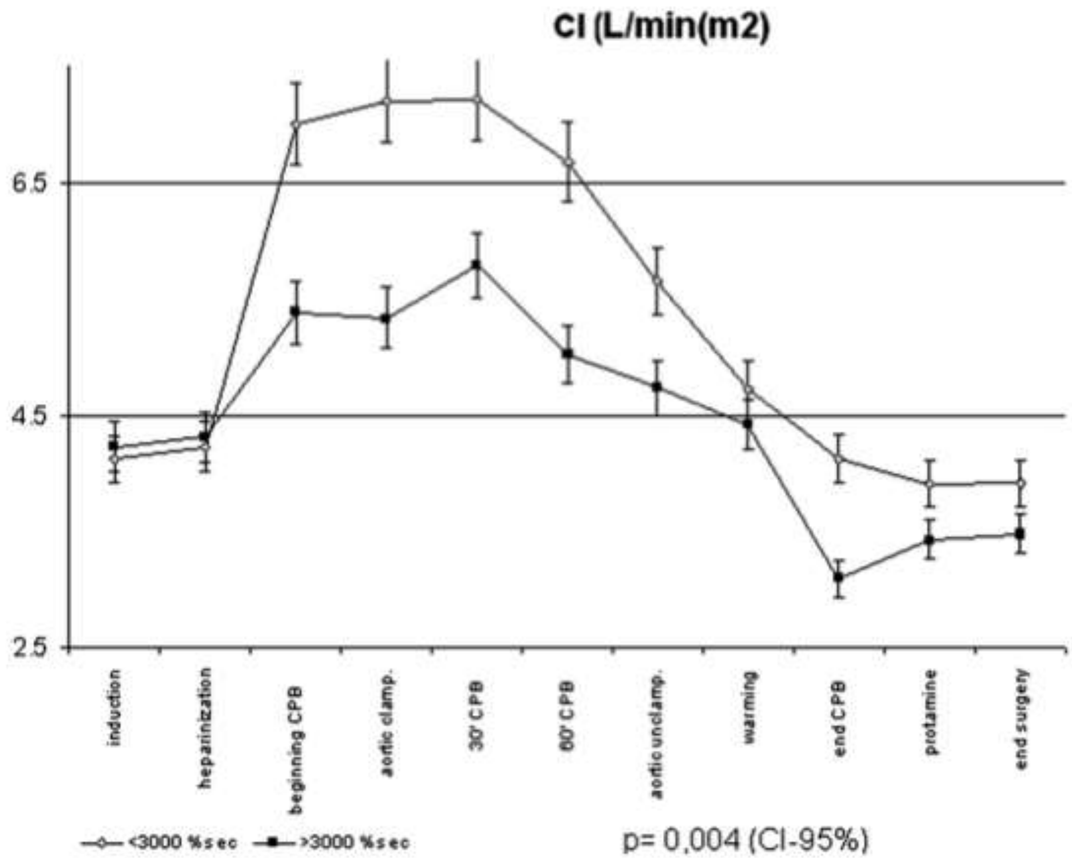

Figure 2. *Image showing the variation of intraoperative cardiac index (CI) between the two groups (rSO_2 de-saturation score < and >3000%/sec; see Table 2).*

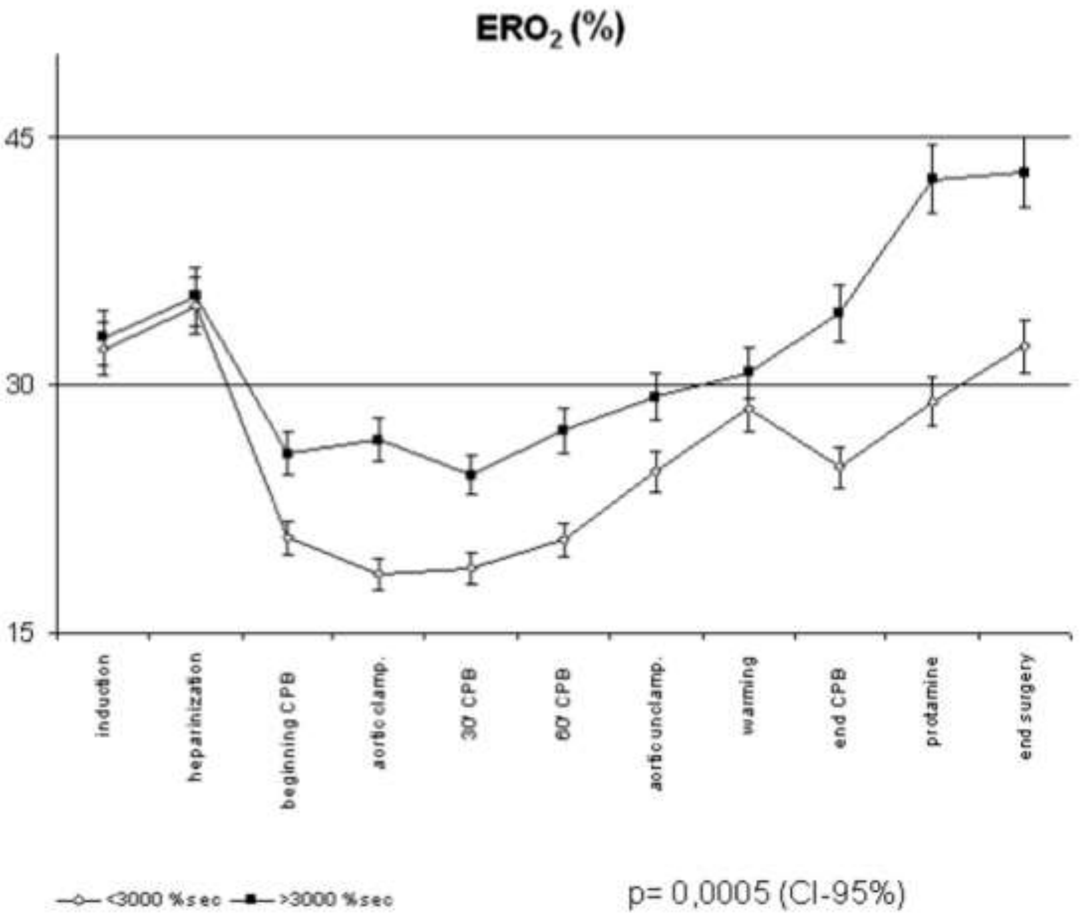

Figure 4. *Image showing the variation of intraoperative oxygen extraction (ERO_2) between the two groups (rSO_2 de-saturation score < and >3000%/sec; see Table 2).*

In conclusion, the use of NIRS may play an important role in the operative and postoperative management of patients with CHD and it represents another marker of the LOS state in addition to the SvO_2 or the lactate level. It provides an early warning sign of hemodynamic or metabolic compromise, enabling early and rapid intervention to prevent or reduce the severity of potentially life-threatening complications.

We believe that the cerebral NIRS monitoring alone will provide the best cost-effective solution in predicting LOS after cardiac surgery. The use of two probes (cerebral and somatic) is particularly indicated in selected procedures such as: (1) off-CPB procedure (i.e., aortic coartectomy), (2) surgery of the aortic arch/isthmus (i.e., aortic coartectomy with the aid of left ventricular assist device, aortic arch repairs, etc), and (3) surgery with the use of peripheral vessel cannulation and remote CPB (i.e., minimally invasive techniques for atrial septal defect closure, etc).

Near-infrared spectroscopy cerebral and somatic (renal) oxygen saturation correlation to continuous venous oxygen saturation via intravenous oximetry catheter☆

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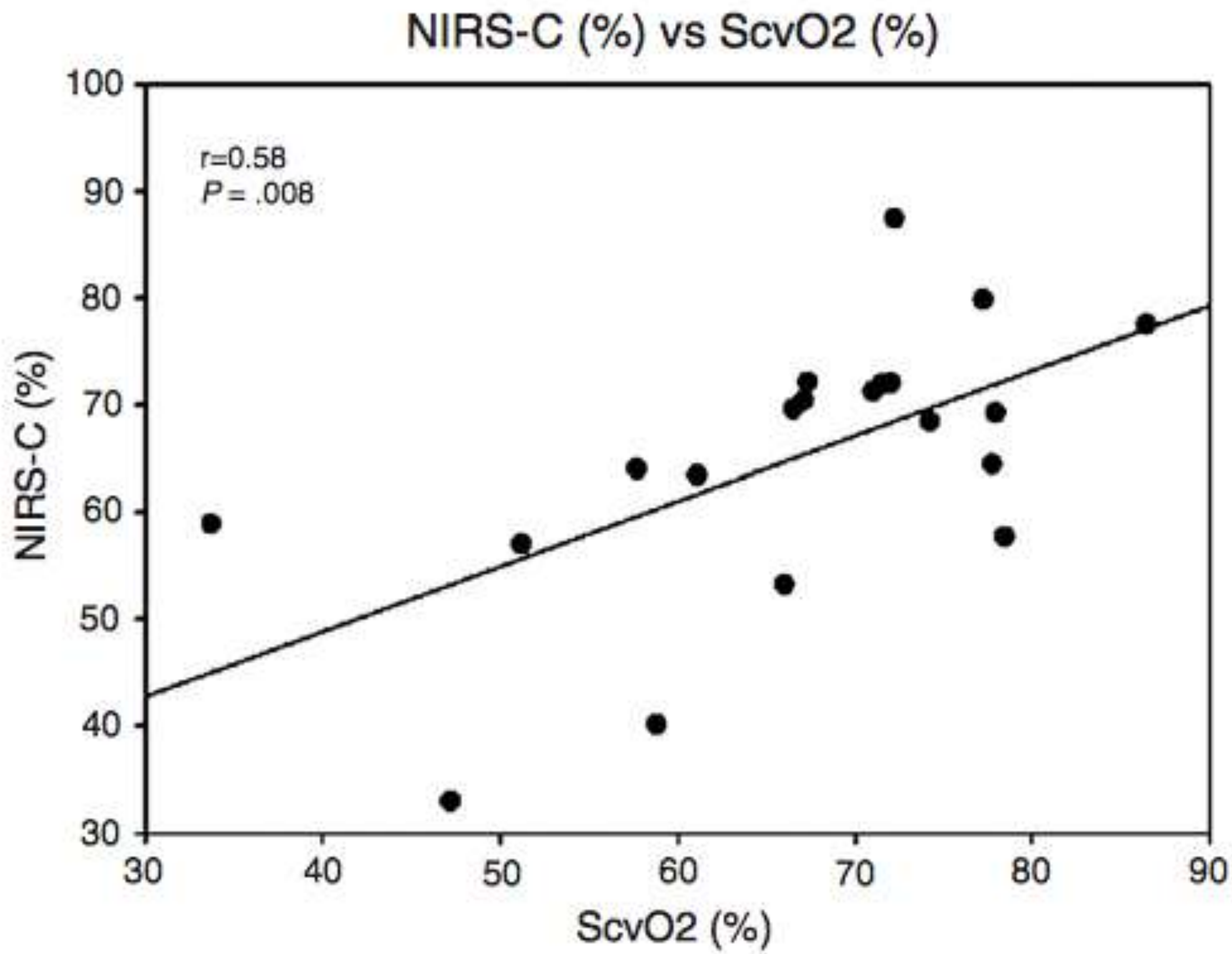

Fig. 2 Weighted scatter plot of NIRS-C vs $ScvO_2$-catheter saturation.

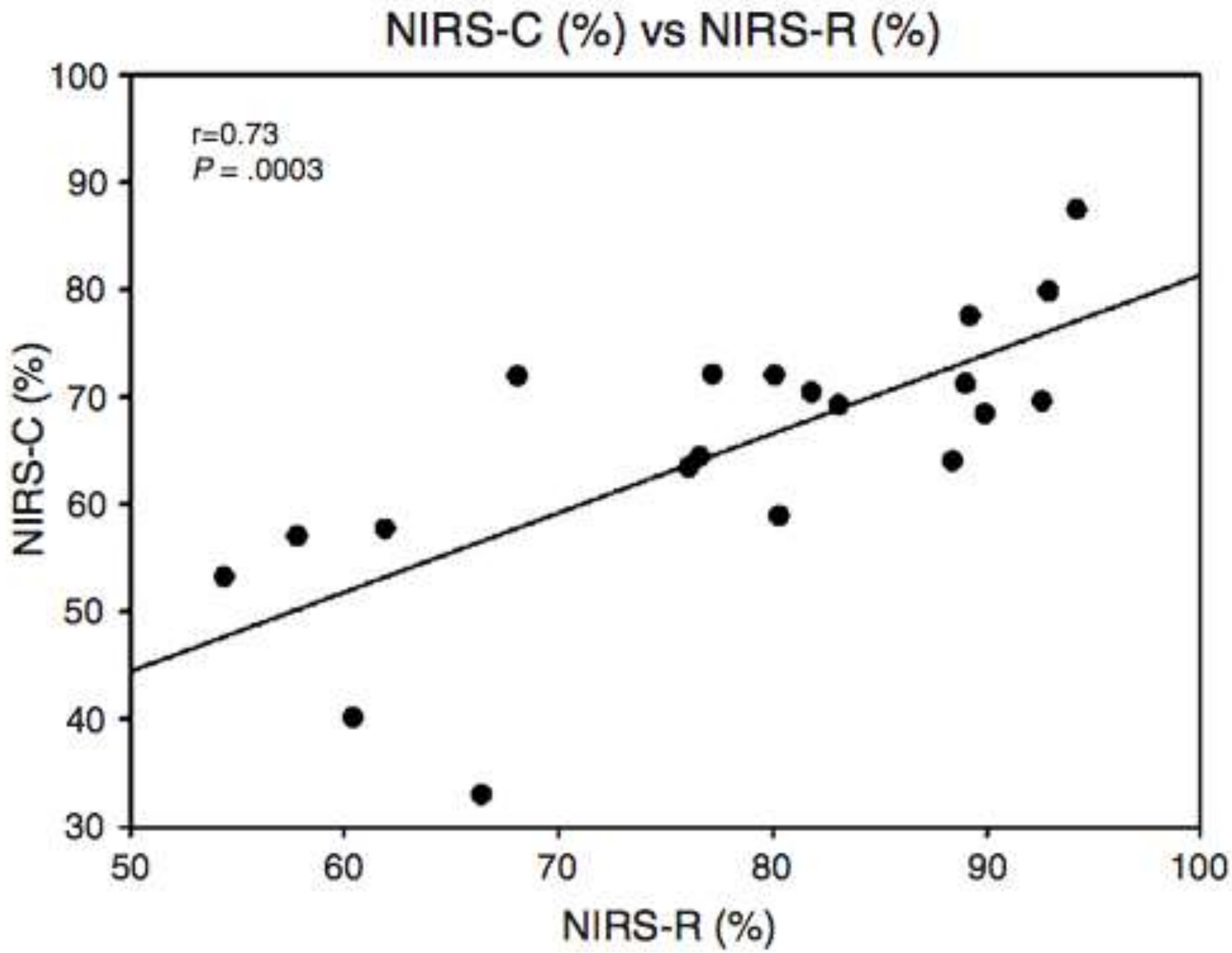

Fig. 3 Weighted scatter plot of NIRS-C vs NIRS-R saturation.

Correlation of abdominal site near-infrared spectroscopy with gastric tonometry in infants following surgery for congenital heart disease*

Jon Kaufman, MD; Melvin C. Almodovar, MD; Jeannie Zuk, PhD, RN; Robert H. Friesen, MD

Objective: Splanchnic oximetry, as measured by near-infrared spectroscopy (NIRS), correlates with gastric tonometry as a means of assessing regional (splanchnic) oxygenation and perfusion.

Design: Prospective, data-gathering study.

Setting: Pediatric cardiac intensive care unit in a tertiary care children's hospital.

Subjects: Neonates and infants with congenital heart disease who underwent catheter intervention or surgical repair requiring cardiopulmonary bypass.

Interventions: None.

Measurements and Main Results: Twenty neonates and infants were studied within 48 hrs of surgery. We measured somatic saturation (rSO_2) via NIRS sensors placed over the anterior abdomen (splanchnic bed) and dorsal lateral flank (renal bed). Somatic rSO_2 readings were paired with simultaneous points of intramucosal gastric pH (pH_i), measured by tonometry. The rSO_2 readings between the abdominal rSO_2 and pH_i ($r = .79$; $p < .0001$) as well as between abdominal rSO_2 and $S\bar{v}o_2$ ($r = .89$; $p < .0001$). There was also significant negative correlation between the abdominal rSO_2 and serum lactate ($r = .77$; $p < .0001$). Correlations between the dorsal lateral (renal) rSO_2 measurements and serum lactate and $S\bar{v}o_2$ were also significant but not as strong.

Conclusions: Abdominal site rSO_2, measured in infants with either single or biventricular physiology, exhibits a strong correlation with gastric pH_i as well as with serum lactate and $S\bar{v}o_2$. The results indicate that rSO_2 measurements over the anterior abdominal wall correlate more strongly than flank rSO_2 with regard to systemic indices of oxygenation and perfusion. This study suggests that the NIRS monitor is a valid modality to obtain an easy, immediate, and noninvasive measurement of splanchnic rSO_2 in infants following cardiac surgery for congenital heart disease. (Pediatr Crit Care Med 2008; 9:62–68)

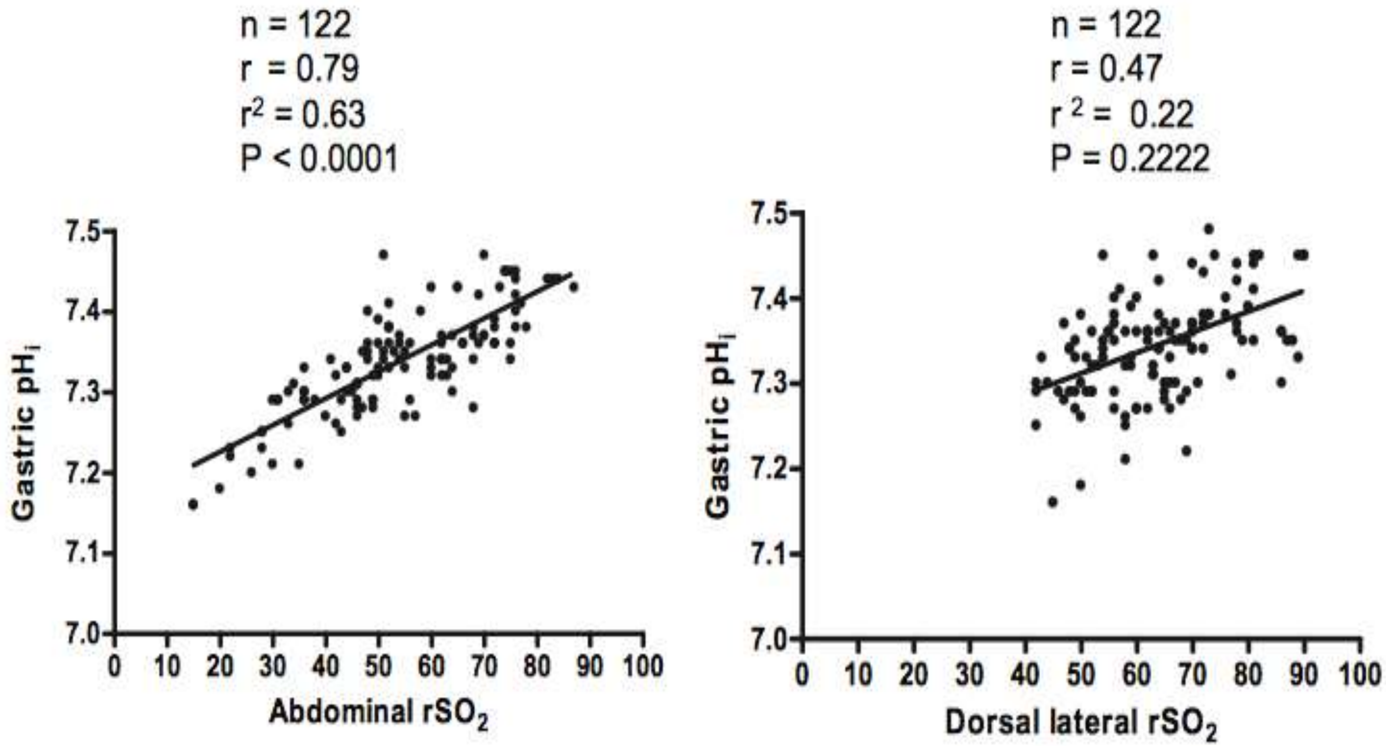

Figure 1. Linear regression plot of correlation between anterior abdominal somatic saturation (*rSO_2*) and intramucosal gastric pH (*pH_i*) and between dorsal lateral rSO_2 and gastric pH_i for all subjects.

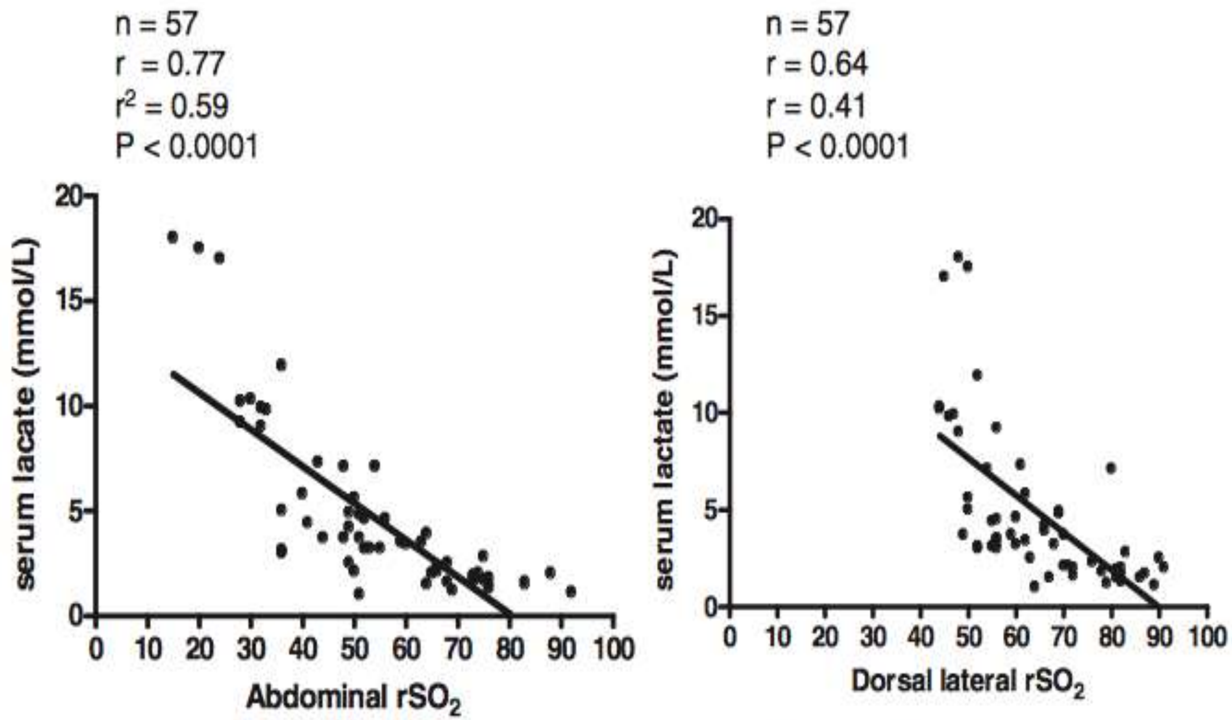

Figure 3. Linear regression plot of correlation between anterior abdominal somatic saturation (*rSO_2*) and dorsal lateral rSO_2 and serum lactate for all subjects.

CONCLUSION

This study suggests that the NIRS abdominal site oximeter can be used as a valid, continuous, noninvasive monitor of splanchnic tissue oxygenation in the postoperative neonate and infant with congenital heart disease. This is the first study to demonstrate correlation between abdominal rSO_2 and gastric tonometry: an accepted means of monitoring abdominal perfusion through a wide range of gastric pH_i and rSO_2 values. As in other recently published studies, we have shown strong correlation between somatic rSO_2 and $S\bar{v}O_2$ as well as between somatic rSO_2 and lactate. Abdominal site NIRS may assist in the perioperative management of cardiac patients by early identification of impaired splanchnic oxygenation due to regional hypoperfusion or a global low cardiac output state.

TEŞEKKÜRLER