

Őeytanın Gör Dediđi

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Acıbadem Üniversitesi

Anesteziyoloji ve Reanimasyon AD

Konu başlıkları

- Monitörizasyondaki temel eksiklikler
- Dolaşımın değerlendirilmesi
 - OAB
 - Akım
 - Hct/Hb
 - FiO₂
 - PCO₂
- Serebral otonöregülasyon
- Near Infrared Spectroscopy (NIRS)

Monitorizasyon

- Anestezi uygulaması sırasındaki temel hedeflerden biri, yeterli doku oksijenasyonunun sağlanmasıdır.
- Bu amaçla hastaların çoğunda **STANDART** ,
 - EKG, KB, SpO₂
- Az bir kısmında **İLERİ**
 - SVB, MvO₂, PAB
- Çok az bir kısmında da **GELİŞMİŞ** monitorizasyon uygulanmaktadır.
 - NIRS, KD, StO₂, PVI, PPV,SVV

Doku oksijenasyonunun değerlendirilmesinde

- Dokuya sunulan oksijenin (DO_2) ve
- Vücudun tükettiği oksijenin (VO_2) belirlenmesi gerekir

Standart monitorizasyonda

- Kalp hızı ve ritmi (EKG)
- Kan basıncı (invazif-noninvazif)
- Periferik doku oksijen saturasyonu (SpO₂)

Doku oksijenasyonunun değerlendirilmesi

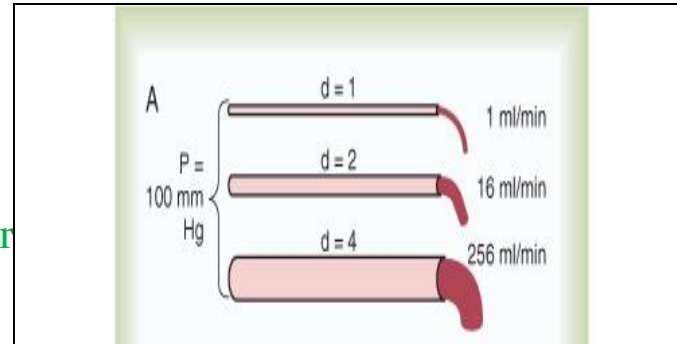
- Dokuya sunulan oksijen (DO₂)
- $KD \quad X \quad CaO_2$
- $KH \quad X \quad \underline{SV} \quad \underline{Hb \quad x \quad 1,34 \quad x \quad SpO_2}$
- Kontraktilite Preload Afterload
- KH ve SaO₂;
 - Birçok değişkenden sadece ikisi, dolayısıyla bütünün tümü_(DO₂) hakkında sağlıklı yorum yapmamız mümkün değil.

Doku oksijenasyonunun değerlendirilmesi

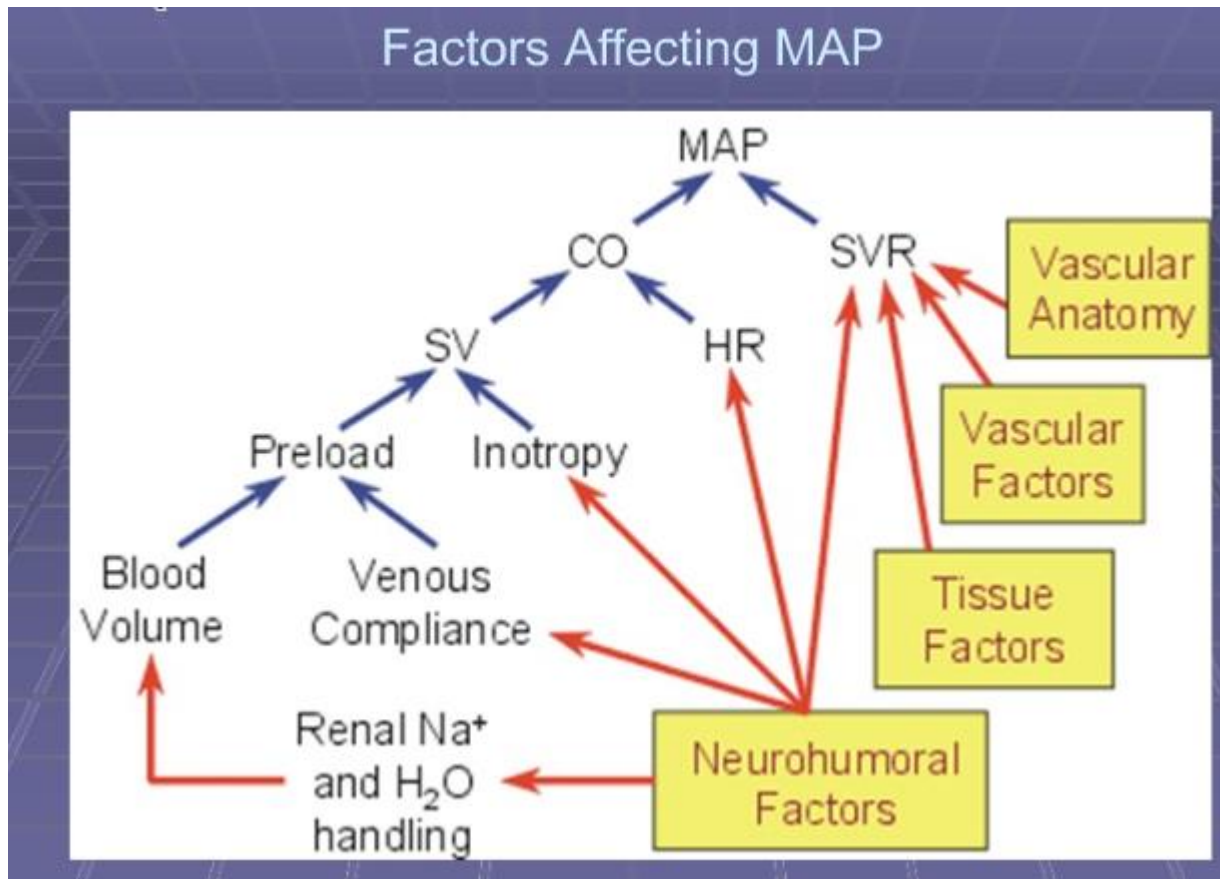
- Kan basıncı= $KD \times SVR$
- Akım= Hız x damar çapı (Cross-Sectional Area (cm²)
 - Aort cross-sectinal area 2,5 cm²
 - Kapiller cross-sectinal area 2500 cm² (1000 katı)
 - SVR' i asıl belirleyen kapiller çap.

– Poiseuille's law $Q = (\pi \Delta P r^4) / 8 \eta l$

– Akımın asıl belirleyicisi **DİRENÇ** dir
– çünkü, akım $\Leftrightarrow d^4$



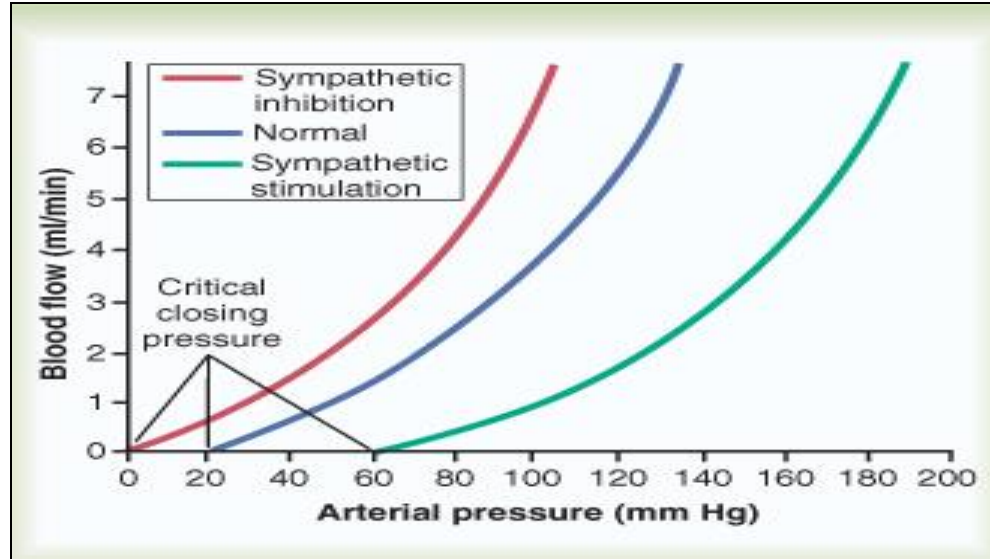
Kan basıncının oluşumuna bakarsak



- Standart monitorizasyonda bizler ölçtüğümüz **basınç değerleri** ile,
 - Basınç-volüm ilişkisinden faydalanarak
 - Atım volümü/ kalp debisi hakkında
- Her zaman doğru olmayan bir yargıda bulunmaktayız.
- Çünkü;
 - Basınç-volüm ilişkisi değişkenlik gösteren bir yapıdadır.

Basınç-volüm

- Yaş ve nöro-horm.değişiklikler
(ateroskleroz, cerrh.str., ağrı, ilaçlar, kalp yet.)



SONUÇ

- Basınç ile volüm arasındaki ilişki birçok nedene bağlı olarak azalabilir kaybolabilir
- Bu nedenle rutin uygulamalar sırasındaki basınç takipleri her zaman yeterli olmayabilir, özellikle
 - İleri yaş
 - Ateroskleroz
 - Hipovolemi
 - Hiperventilasyon ($PCO_2 \Leftrightarrow SVR$)
 - Hipoksi (kapanma volümü)
 - Anemi
 - Kalp yetmezliği
 - Majör cerrahi (artan stres yanıt)
 - Uzun süren anestezi-ameliyat süresi varlığında ileri monitorizasyon ihtiyacının gerekliliği aşıkardır.

Kalp Cerrahisinde Monitorizasyon

- Geleneksel uygulama standart /ileri monitorizasyon
- Ancak;
 - EKD sırasında iskemiye en duyarlı organlardan biri olan BEYNİN monitorize **edilmemesi** ciddi bir eksiklik
- Bu eksikliđi gidermek için de,
 - EKD sırasında hemodinamik ve kan gazı parametreleri **normal üstü/ normal** deđerlerde tutulmaya alıřılmakta
 - Bu ise beraberinde ciddi sorunları getirmektedir.

OAB,KD,Hb,PO2

Normal üstü olması durumunda

- **OAB:**
 - Artan kollateral dolaşıma bağlı olarak krosklemp altındaki kalpde fibrilasyon →oksijen tüketiminde artma
 - Krosklemp altındaki aort da yaralanma
- **Debi:**
 - Shear stress → hemoliz
 - Mikro bouble emboliler
 - VK ajan kullanımını veya volüm ilavesi
 - End organ iskemisi
 - Pozitif sıvı dengesi
- **PO₂:**
 - Serbest oksijen radikalleri miktarında artma
 - Hiperoksemiye bağlı olumsuz etkiler (Eritrosit Rheolojisinde bozulma end organ hasarı)
- **Hb:**
 - Artmış Transfüzyon oranları
 - Transfüzyonun erken ve geç dönem etkileri

OAB,KD,Hb,PO2

Normal deęerlerde olması durumunda

- Kime gore normal ?

Table 2. Arterial Pressure Management

| Potential advantages of higher MAPs | Potential advantages of lower MAPs |
|---|---|
| Enhanced tissue perfusion in high risk patients (hypertensive, diabetic, and elderly) | Less trauma to blood elements |
| Improved collateral flow to tissues at risk of ischemia | Reduction of blood in the surgical field |
| Allows for higher pump flow rates on CPB | Less cardiotomy suction |
| | Permits the use of smaller venous and arterial cannulae |
| | Enhanced myocardial protection (reduced collateral coronary blood flow) |
| | Reduced embolic load to the CNS (reduced pump flow) |

MAP = mean arterial blood pressure; CPB = cardiopulmonary bypass; CNS = central venous system.

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

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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 $>35^{\circ}\text{C}$ 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 \pm 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)

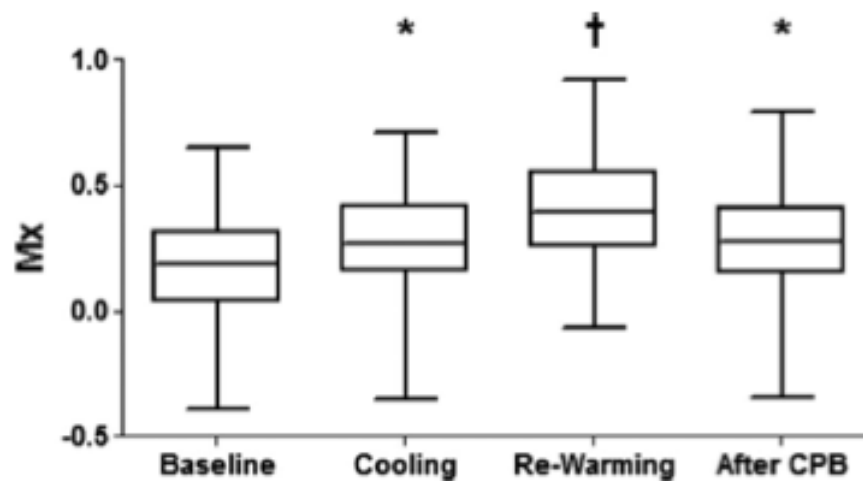


Figure 1. Mean velocity index (Mx) values obtained after anesthesia induction but before cardiopulmonary bypass (CPB) initiation (baseline) and during the cooling and re-warming phases of CPB. Mx is derived as the nonlinear correlation between cerebral blood flow (CBF) velocity of the right and left 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 (<i>n</i> = 60) | Impairment (<i>n</i> = 67) | <i>P</i> |
|------------------------------|--|---------------------------------------|-----------------|
| 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; $P = .008$).

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

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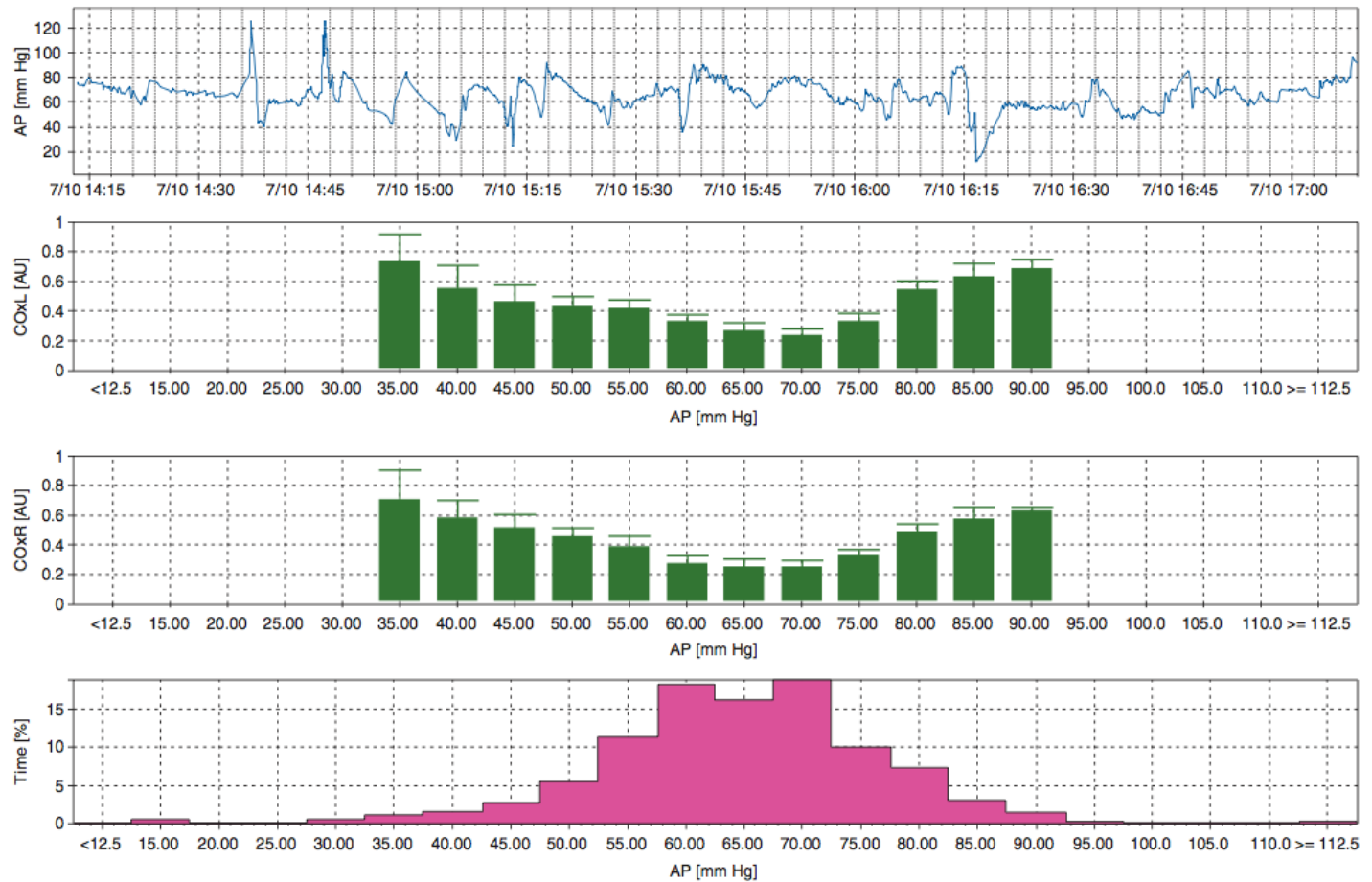


Fig 1 The representative graph of autoregulation monitoring during CPB. The COx represents the correlation coefficient between low-frequency regional cerebral oxygen saturation and MAP. When arterial pressure is above or below the autoregulation threshold, COx approaches 1, but when autoregulation is 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 mmHg | 0.30 (0.05–0.86) | 0.24 (0–0.75) | 0.321 |
| MAP > 105 mmHg | 0.08 (0–0.28) | 0.07 (0–0.31) | 0.634 |

Sonuç

- Daha önce yapılan çalışmalarda serebral otonöregülasyon alt sınırının altındaki bir OAB'nin sonuç parametrelerini olumsuz etkilediği gösterilmişti
- Bu çalışmada da otonöregülasyon üst sınırının üstündeki bir OAB'nin benzer etkiler oluşturduğu görülmüştür.
- **Bu nedenle serebral otonöregülasyonun NIRS ile izlenerek uygun OAB'nin sağlanması çok önemlidir.**

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Perioperative optimal blood pressure as determined by ultrasound tagged near infrared spectroscopy and its association with postoperative acute kidney injury in cardiac surgery patients

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Abstract

OBJECTIVES: Perioperative blood pressure management by targeting individualized optimal blood pressure, determined by cerebral blood flow autoregulation monitoring, may ensure sufficient renal perfusion. The purpose of this study was to evaluate changes in the optimal blood pressure for individual patients, determined during cardiopulmonary bypass (CPB) and during early postoperative period in intensive care unit (ICU). A secondary aim was to examine if excursions below optimal blood pressure in the ICU are associated with risk of cardiac surgery-associated acute kidney injury (CSA-AKI).

METHODS: One hundred and ten patients undergoing cardiac surgery had cerebral blood flow monitored with a novel technology using ultrasound tagged near infrared spectroscopy (UT-NIRS) during CPB and in the first 3 h after surgery in the ICU. The correlation flow index (CFx) was calculated as a moving, linear correlation coefficient between cerebral flow index measured using UT-NIRS and mean arterial pressure (MAP). Optimal blood pressure was defined as the MAP with the lowest CFx. Changes in optimal blood pressure in the perioperative period were observed and the association of blood pressure excursions (magnitude and duration) below the optimal blood pressure [area under the curve (AUC) < OptMAP mmHg_xh] with incidence of CSA-AKI (defined using Kidney Disease: Improving Global Outcomes criteria) was examined.

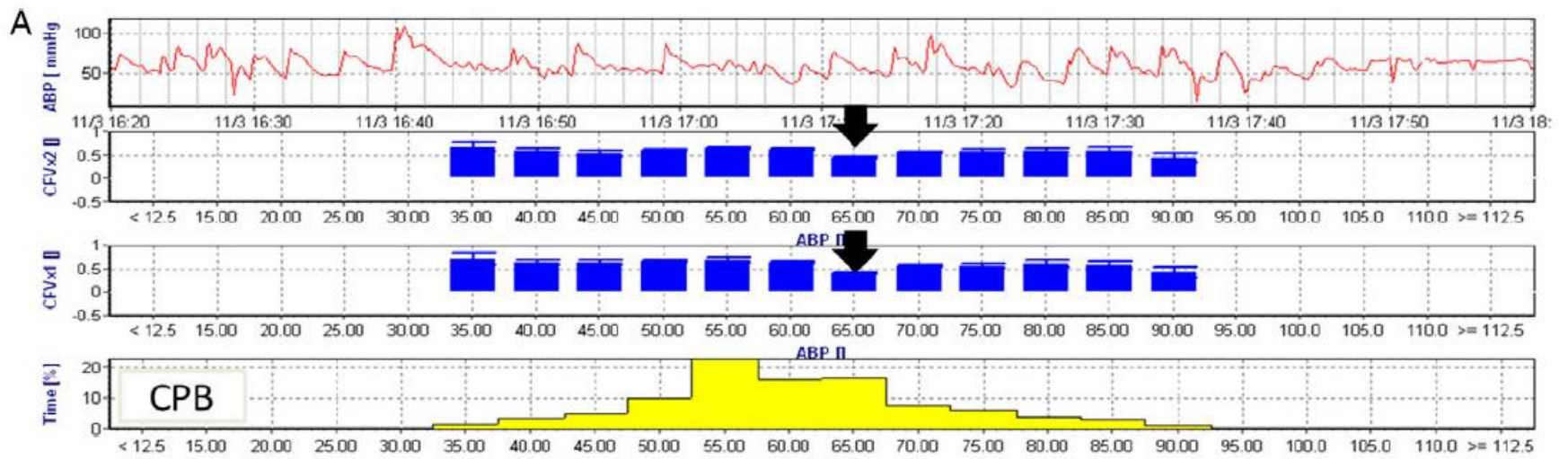
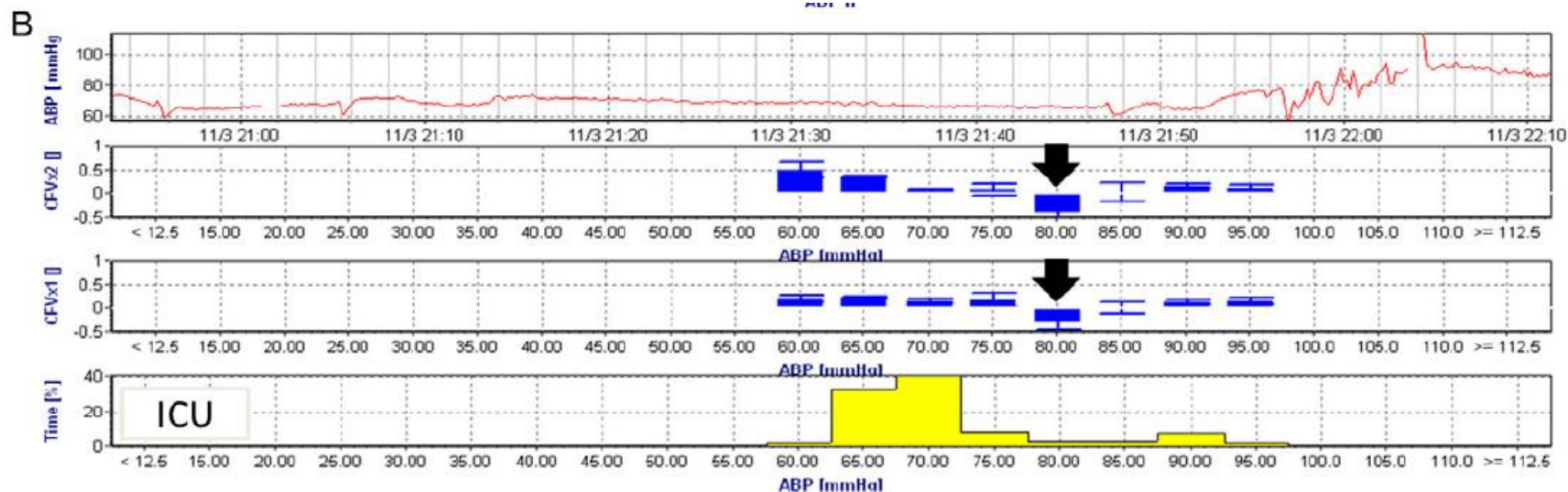


Figure 1: A representative cerebral autoregulation monitoring recording from a single patient obtained during CPB (A) and after ICU admission (B). The top graph in each recording is the time series of ABP while the next two graphs represent the CFx from the left and right brain hemispheres. The latter represent the average CFx values placed in 5 mmHg blood pressure bins. When cerebral blood flow is in the autoregulated range, CFx is close to zero but when blood pressure is below or above the autoregulation range CFx approaches 1. The bottom graph (B) represents the percentage of time during the recording where blood pressure was in each 5



above the autoregulation range CFx approaches 1. The bottom graph (B) represents the percentage of time during the recording where blood pressure was in each 5 mmHg bins. Optimal mean arterial pressure is defined as the pressure with the lowest CFx (black arrow) or that blood pressure with the best autoregulation. Note, that during CPB (A), the optimal blood pressure is 65 mmHg. During the ICU period (B) the optimal blood pressure was 80 mmHg. The time scales between the two periods are different but note that blood pressure variability was more pronounced during CPB than in the ICU. Blood pressure increased around 21:25 h in the ICU likely coinciding with emergence from sedation and weaning from mechanical ventilation. CPB: cardiopulmonary bypass; ICU: intensive care unit; ABP: arterial pressure; CFx: correlation flow index.

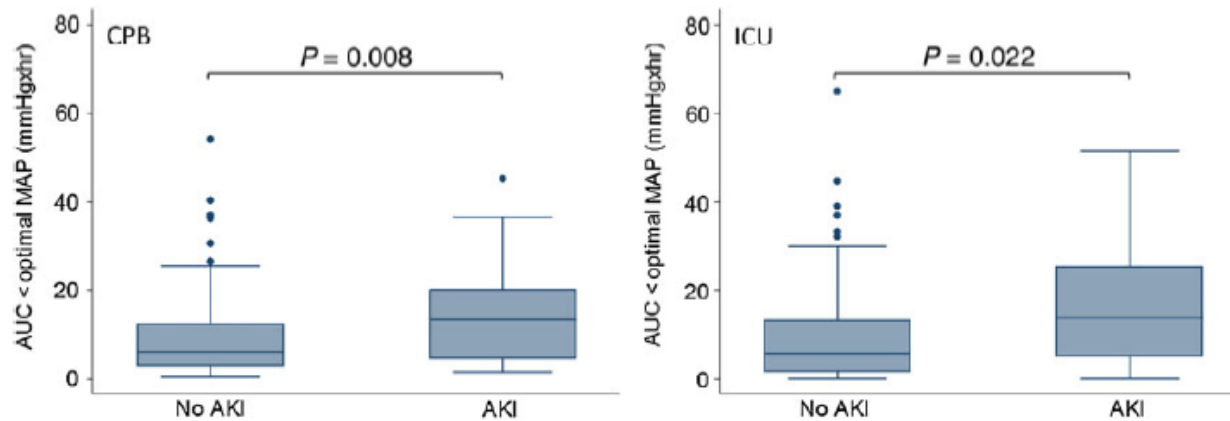


Figure 3: Box and whisker plot showing blood pressure excursion below the optimal blood pressure during CPB and in the ICU for patients with and without CSA-AKI. The horizontal line in the shaded box represents the median value, and the shaded box represents the interquartile range. The error bars below and above the shaded area represents $\pm 1.5 \times$ the interquartile range; points beyond the error bar are outliers. CPB: cardiopulmonary bypass; ICU: intensive care unit; CSA-AKI: cardiac surgery-associated acute kidney injury.

Table 5: Logistic regression model for cardiac surgery-associated acute kidney injury

| | Odds ratio | Standard error | 95% CI | P-value |
|----------------------------------|------------|----------------|-----------|---------|
| Total bypass time | 1.00 | 0.004 | 0.99-1.01 | 0.20 |
| Total cross-clamp time | 1.01 | 0.012 | 0.99-1.03 | 0.45 |
| Surgery other than isolated CABG | 0.95 | 0.456 | 0.36-2.43 | 0.91 |
| Preoperative eGFR | 0.99 | 0.012 | 0.96-1.01 | 0.30 |
| AUC < optimal blood pressure | 1.03 | 0.011 | 1.01-1.05 | 0.017 |

Total bypass time, total cross-clamp time, surgery other than isolated CABG, preoperative eGFR and the sum of blood pressure excursions below the optimal blood pressure during CPB and in the ICU were included in the model.

AUC: area under the curve; CABG: coronary artery bypass graft; CI: confidence interval; eGFR: estimated glomerular filtration rate; ICU: intensive care unit.

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 \times 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 |
| Paco ₂ (mm Hg) | 40 ± 3 | 41 ± 3 | 0.2670 |
| Pao 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.


^aValues 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 | <i>p</i> |
|--|----------------------|--------------------------------|-----------------|
| Magnitude of mean arterial pressure \leq lower limit of autoregulation (mm Hg \times 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 |

Increasing mean arterial pressure during cardiac surgery does not reduce the rate of postoperative acute kidney injury

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X Moreau,¹ C Baufreton² and L Beydon¹

Perfusion
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DOI: 10.1177/0267659114527331
prf.sagepub.com


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 1.73m² 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 |
|--|--------------------|---------------------|---------|
| <i>Preoperative renal function</i> | | | |
| Creatinine ($\mu\text{mol/L}$) | 95 \pm 28 | 93 \pm 25 | 0.53 |
| Indexed creatinine clearance (ml/min/1.73m^2) | 61 \pm 21 | 62 \pm 23 | 0.91 |
| BUN (mmol/l) | 8.6 \pm 3.5 | 8.5 \pm 3.3 | 0.85 |
| <i>Postoperative renal function</i> | | | |
| Renal resistivity index | 0.69 \pm 0.06 | 0.70 \pm 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 \pm 0.87 | 0.84 \pm 0.52 | 0.20 |
| Serum creatinine peak ($\mu\text{mol/L}$) | 108 \pm 52 | 105 \pm 50 | 0.73 |
| Time of occurrence for the serum creatinine peak (h) | 13.6 \pm 19.1 | 14.2 \pm 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 \pm 2.8 | 3.2 \pm 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.

Sonuç

- Fizyolojik sınırlar içindeki OAB dahi
 - Kişiselleştirilmiş fizyolojik farklılıklar nedeni ile
 - Güvenli olmayabilir
 - Şeytan buradadır ??????



DEBi : FLOW

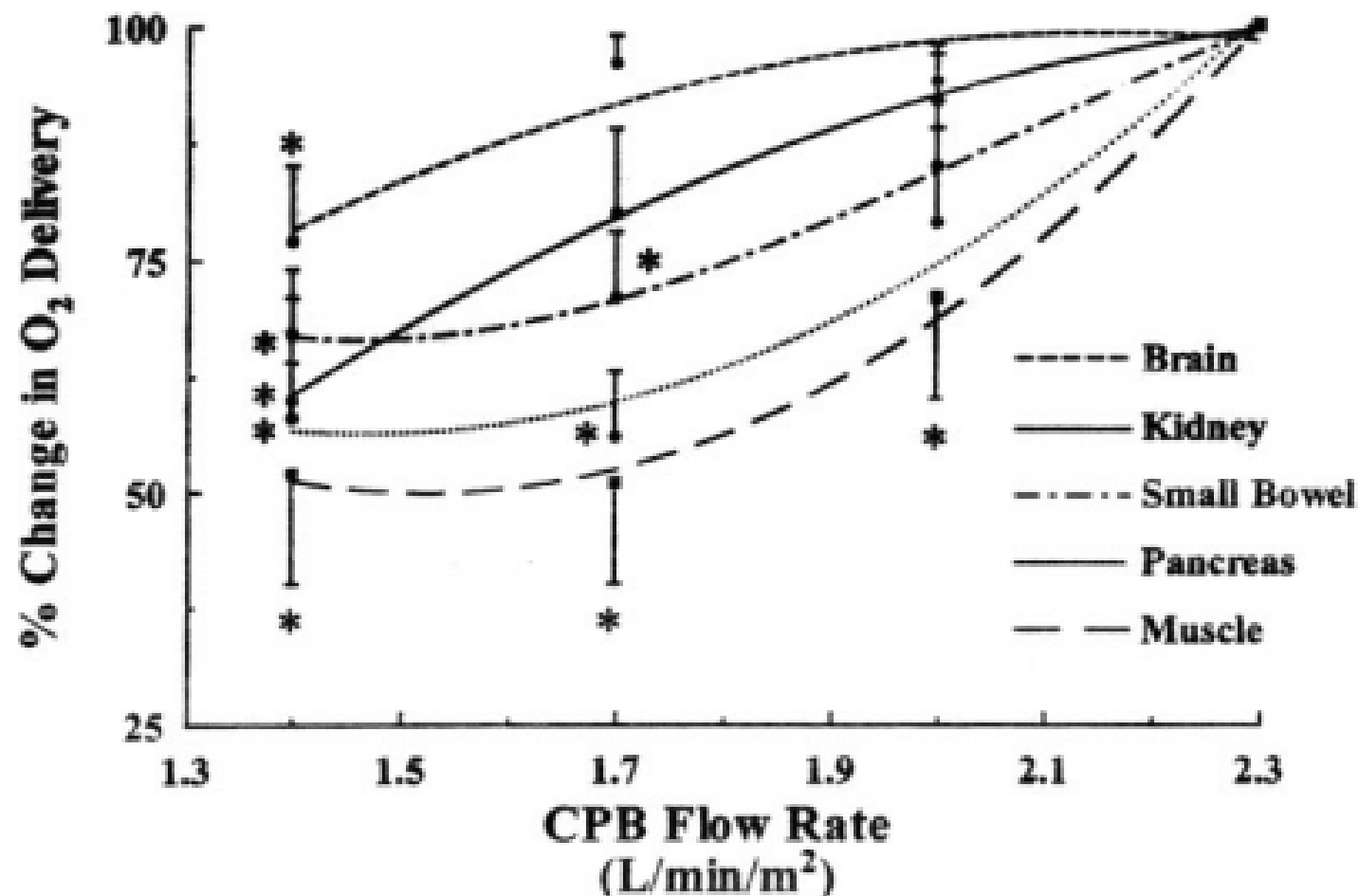
Arteriyel akım hızı :Debi ?

- Vücut ağırlığına (kg)
 - Erişkinde **30-70** ml/kg
- Vücut yüzey alanına göre ayarlanmakta (m²)
 - **1.6 -3.2** L/m²
- Görüldüğü gibi her iki yöntemde de aralık oldukça geniş
- **Lean Body Mass (LBM)**

Table 4. Clinical Studies Examining the Effect of Pump Flow Rate on Cerebral Blood Flow and Metabolism

| Study | No of Patients | Flow rate | Temperature | Acid-base management | MAP | Results (mm Hg) |
|-----------------------------------|----------------|---|-------------|----------------------|-------|--|
| Cook et al., 1997 ³² | 30 | 1.2–2.3 L · min ⁻¹ · m ⁻² | 27°C | α stat | 50–70 | No differences in mean CBF or CMR at high or low flows |
| Govier et al., 1984 ⁹ | 67 | 1.0–2.2 L · min ⁻¹ · m ⁻² | 27°C | α stat | 45–70 | No change in regional CBF or CMR at differing flow rates |
| Rogers et al., 1992 ³³ | 24 | 1.75–2.25 L · min ⁻¹ · m ⁻² | 27°C | α stat and pH stat | 68–75 | No difference in CBF or CMR at differing flow rates |
| Soma et al., 1989 ³⁴ | 21 | 40–70mL · kg ⁻¹ · min ⁻¹ | 27°C | pH stat | 59–70 | CBF increased proportionally to flow rate |

MAP = mean arterial blood pressures; CBF = cerebral blood flow; CMR = cerebral metabolic rate.



Distribution and Hierarchy of Regional Blood Flow During Hypothermic Cardiopulmonary Bypass

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Background. Cardiopulmonary bypass (CPB) may decrease oxygen delivery relative to the nonbypass state. We predicted that a hierarchy of regional blood flow could be characterized under hypothermic (27°C) CPB.

Methods. Ten pigs underwent bypass at 27°C. Fluorescent microspheres were administered before and during CPB at four randomized flows: 1.9, 1.6, 1.3, and 1.0 L · min⁻¹ · m⁻². At completion, tissue samples were obtained from brain, renal cortex and medulla, pancreas, small bowel, and limb muscle for regional blood flow determination.

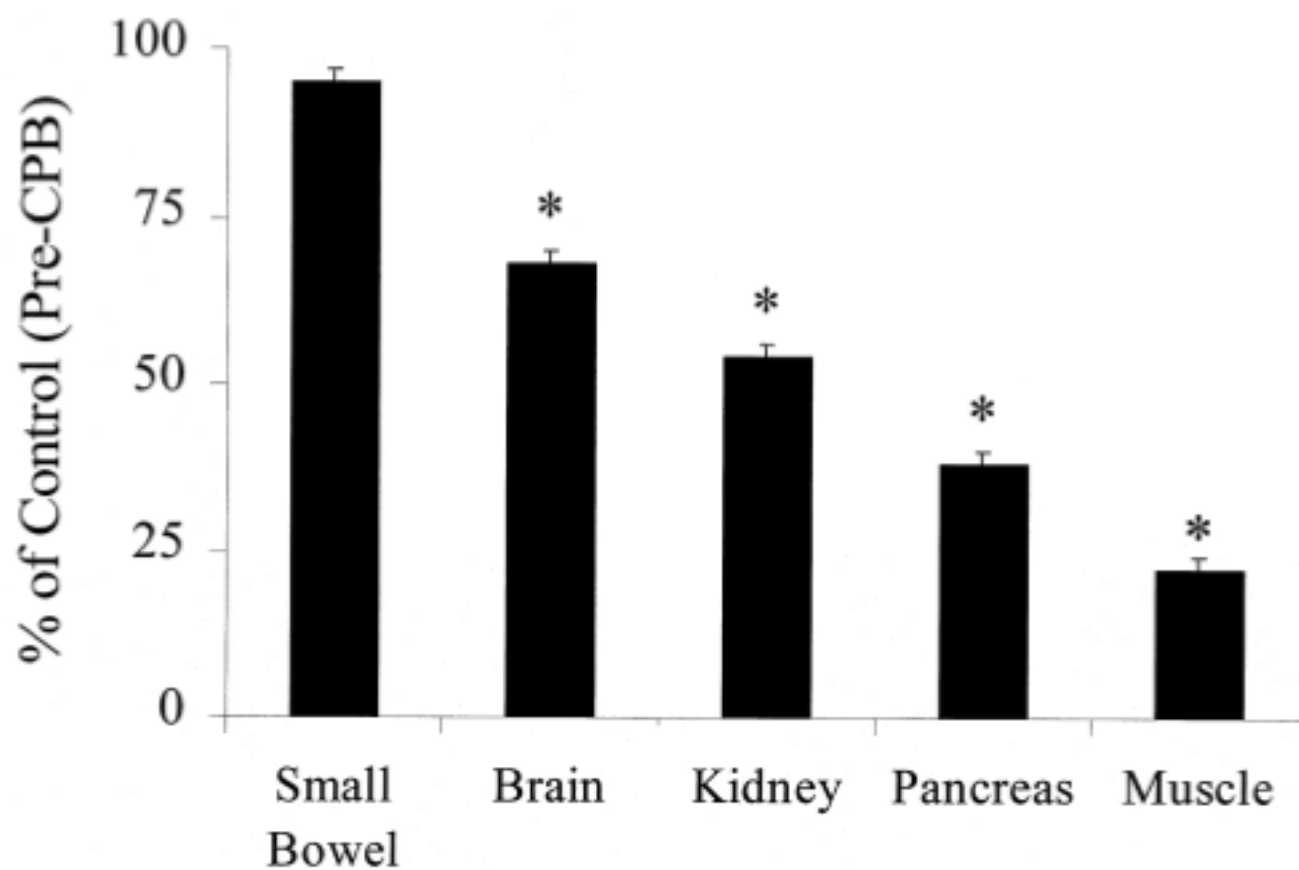
Results. Cerebral blood flow remained unchanged between CPB flows of 1.9 and 1.3 L · min⁻¹ · m⁻². Renal

perfusion was stable between flows of 1.9 and 1.6 L · min⁻¹ · m⁻², whereas perfusion of small bowel decreased linearly with pump flow. Pancreatic perfusion was unchanged over the range of flows studied; muscle blood flow was profoundly reduced at the highest CPB flow and further decreased if pump flow was reduced below 1.6 L · min⁻¹ · m⁻².

Conclusions. This study characterizes the organ-specific hierarchy of blood flow and oxygen distribution during hypothermic CPB. These dynamics are relevant to clinical decisions for perfusion management.

(Ann Thorac Surg 2001;72:542-7)

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*Fig 1. Change in regional blood flow in five organ beds during 27°C bypass at a flow of $1.9 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ relative to that under non-bypass conditions. Mean values \pm SD are also shown. * $p < 0.05$ versus cardiopulmonary bypass (CPB) at $1.9 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ by paired t test.*

Is body surface area still the best way to determine pump flow rate during cardiopulmonary bypass?

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For over four decades, pump flow rate during cardiopulmonary bypass (CPB) has been estimated using body surface area (BSA). As patients presenting for heart surgery are increasingly obese, this approach may no longer be appropriate and other estimates of systemic metabolism should be used, such as body mass index and lean body mass. Mixed venous oxygen saturation (SvO₂) is a robust and independent estimate of the global efficacy of CPB. The aim of this study was to determine which factors, including body surface area, body mass index and lean body mass, best predict SvO₂ during CPB.

Forty-eight patients undergoing elective cardiac surgery requiring CPB were studied. Patients' height, weight and skinfold thickness at four sites (biceps, triceps, subscapularis and suprailiac) were measured. Body surface area, lean body mass and body mass index were then calculated. Pump flow rate was maintained at 2.4 L/min/m² during CPB as per standard unit protocol. Arterial and mixed venous blood samples were taken during the

cooling, stable hypothermia and rewarming phases of CPB. Nasopharyngeal temperatures and flow rates were recorded contemporaneously. The blood samples were analysed for oxygen saturation, haemoglobin concentration and partial pressures of oxygen and carbon dioxide. The values of the three time points were meaned. All potential predictor variables were then univariately correlated with mixed venous oxygen saturation (SvO₂). Those correlating significantly ($p < 0.1$) were entered into a multivariate linear regression model. Nasopharyngeal temperature ($\beta = 0.615$, $p < 0.001$) and lean body mass ($\beta = 0.256$, $p < 0.028$) were the only significant predictors of SvO₂ ($r^2 = 0.433$, $p < 0.001$). Pump flow rates maintained at 2.4 L/min/m² throughout CPB results in relative over-perfusion during hypothermia. Lean body mass may be a more sensitive estimate of systemic metabolism and, therefore, may provide a more accurate means of determining pump flow rate than body surface area in patients undergoing heart surgery. *Perfusion* (2006) 21, 139–147.

For the above reasons, we hypothesized that LBM would be a better predictor of the efficacy of CPB than either BSA or BMI and, therefore, the aim of this study was to determine what factors, including BSA, BMI and LBM, best predict SvO₂ during CPB.

Appendix 6

Lean body mass¹⁴

$$\text{Density} = c - m \times \log(\text{biceps} + \text{triceps} \\ + \text{suprailiac} + \text{subscapularis})$$

where c and m are regression coefficients obtained from a table, which relates to various age groups and sex.

$$\text{Total fat (\%)} = (4.95/\text{density} - 4.5) \times 100$$

$$\text{Lean body mass (\%)} = 100 - \% \text{ fat}$$

Table 1 Univariate correlations between predictor variables and mixed venous oxygen saturation

| Variable | Mean | | Cooling | | Stable hypothermia | | Re-warming | |
|---------------------------|--------|--------|---------|--------|--------------------|--------|------------|--------|
| | r | p | r | p | r | p | r | p |
| Age | 0.138 | 0.350 | -0.011 | 0.943 | 0.174 | 0.243 | 0.285 | 0.058 |
| Weight | -0.126 | 0.393 | -0.225 | 0.124 | 0.013 | 0.933 | 0.002 | 0.989 |
| Height | 0.088 | 0.552 | -0.025 | 0.865 | 0.188 | 0.205 | 0.148 | 0.332 |
| SaO ₂ | -0.042 | 0.779 | -0.490 | 0.738 | -0.009 | 0.953 | -0.069 | 0.650 |
| <u>Temperature</u> | -0.595 | <0.001 | -0.610 | <0.001 | -0.764 | <0.001 | 0.214 | 0.157 |
| Pump flow rate | 0.060 | 0.686 | -0.088 | 0.554 | 0.123 | 0.409 | 0.255 | 0.091 |
| PaO ₂ | 0.005 | 0.971 | -0.049 | 0.740 | 0.347 | 0.017 | -0.159 | 0.297 |
| PvO ₂ | 0.682 | <0.001 | 0.751 | <0.001 | -0.626 | <0.001 | 0.752 | <0.001 |
| PvCO ₂ | -0.175 | 0.235 | -0.326 | 0.024 | 0.021 | 0.891 | -0.092 | 0.547 |
| Haemoglobin concentration | 0.150 | 0.309 | 0.224 | 0.126 | 0.079 | 0.597 | 0.302 | 0.044 |
| BSA | -0.062 | 0.677 | -0.176 | 0.231 | 0.079 | 0.597 | 0.056 | 0.715 |
| <u>BMI</u> | -0.296 | 0.041 | 0.390 | 0.006 | -0.123 | 0.412 | -0.092 | 0.548 |
| <u>LBM</u> | 0.336 | 0.020 | 0.291 | 0.045 | 0.257 | 0.081 | 0.171 | 0.262 |

Mean, mean of the variables from time point 1 (cooling), 2 (stable hypothermia) and 3 (rewarming); r, correlation coefficient value; PaO₂, partial pressure of oxygen; BSA, body surface area; BMI, body mass index; LBM, lean body mass.

Table 2 Regression models of the predictor variables and mixed venous oxygen saturation as the dependent variables meaned over the three time points

| | r^2 | <i>Variables entered</i> | <i>Variables excluded</i> | <i>Variables included</i> | β | p |
|---------|-------|--------------------------|---------------------------|---------------------------|---------|--------|
| Model 1 | 0.354 | BMI and temperature | BMI | Temperature | -0.595 | <0.001 |
| Model 2 | 0.416 | LBM and temperature | | Temperature | -0.601 | <0.001 |
| | | | | LBM | 0.249 | 0.034 |
| Model 3 | 0.433 | LBM and 1/temperature | | 1/Temperature | 0.615 | <0.001 |
| | | | | LBM | 0.256 | 0.028 |

r^2 , goodness of fit; β , standardized regression coefficient; p , significance level; BMI, body mass index (kg/m²); LBM, lean body mass (%); temperature, nasopharyngeal temperature (°C); 1/temperature, inverse of the nasopharyngeal temperature (°C).

Table 3 Regression models of predictor variables and mixed venous oxygen saturation as the dependent variable during cooling (time point 1).

| | r^2 | <i>Variables entered</i> | <i>Excluded variables</i> | <i>Included variables</i> | β | p |
|---------|-------|--------------------------|---------------------------|---------------------------|---------|---------|
| Model 1 | 0.372 | BMI and temperature | BMI | Temperature | -0.610 | < 0.001 |
| Model 2 | 0.372 | LBM and temperature | LBM | Temperature | -0.610 | < 0.001 |
| Model 3 | 0.393 | LBM and 1/temperature | LBM | 1/temperature | 0.627 | < 0.001 |

r^2 , goodness of fit; β , standardized regression coefficient; p , significance level; BMI, body mass index (kg/m^2); LBM, lean body mass (%); temperature, nasopharyngeal temperature ($^{\circ}\text{C}$); 1/temperature: inverse nasopharyngeal temperature ($^{\circ}\text{C}$).

Table 4 The regression models produced using the variables from time point two (stable hypothermia) and mixed venous oxygen saturation from time point 2 as the dependent variable

| | r^2 | <i>Variables entered</i> | <i>Excluded variables</i> | <i>Included variables</i> | β | p |
|---------|-------|-----------------------------|---------------------------|----------------------------|-----------------|------------------|
| Model 1 | 0.518 | LBM and temperature | | Temperature LBM | -0.766 0.243 | < 0.001 0.010 |
| Model 2 | 0.643 | LBM and inverse temperature | | Inverse temperature LBM | 0.680 0.268 | < 0.001 0.14 |

r^2 , goodness of fit; β , regression coefficient; p , significance; temperature, nasopharyngeal temperature ($^{\circ}\text{C}$); BMI, body mass index (kg/m^2); LBM, lean body mass (%).

Conclusion

- When the pump flow rate is maintained at 2.4 L/m²/ min, BSA and BMI do not influence SvO₂.
- In contrast, LBM is positively correlated and temperature is strongly negatively correlated with SvO₂.
- These findings suggest that using a fixed pump flow rate determined by BSA may no longer be appropriate.

Elevated flow rate during cardiopulmonary bypass is associated with fluid accumulation

Oddbjørn Haugen, MD,^a Marit Farstad, MD, PhD,^c Venny Kvalheim, MD,^b Olav Bøe, DDS, MSc,^d and Paul Husby, MD, PhD^c



Dr. Kvalheim, Haugen, Farstad, Bøe, and Husby (left to right)

Objective: High flow rates during cardiopulmonary bypass are assumed to increase fluid accumulation. This study aimed to determine whether two different flow rates during cardiopulmonary bypass alter the intraoperative fluid balance and extravasation rate.

Methods: Sixteen pigs underwent 60 minutes of normothermic bypass, followed by 90 minutes of hypothermic bypass. A high-flow group (HF group, $n = 8$) had a cardiopulmonary bypass flow rate of $110 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and a low-flow group (LF group, $n = 8$) had a rate of $80 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. Blood chemistry, hemodynamic parameters, plasma and interstitial colloid osmotic pressure, net fluid balance, plasma volume, fluid extravasation rate, and total tissue water content were measured or calculated. Results are presented as mean (standard deviation).

Results: The average net fluid balance during cardiopulmonary bypass was 1.02 (0.25) and 0.73 (0.23) $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ in the HF group and LF group, respectively ($P < .05$). The average fluid extravasation rate was 0.98 (0.22) and 0.77 (0.22) $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ in the HF group and the LF group ($P = .07$). Total water content was higher in the kidneys ($P < .05$) and tended to be higher in the lungs ($P = .05$), liver ($P = .07$), and brain ($P = .07$) of the HF group than in those of the LF group. The between-group differences in net fluid balance and fluid extravasation rate were present during the first 30 minutes of normothermic cardiopulmonary bypass. Thereafter, the values stabilized and remained similar in the two groups. Plasma volume and systemic vascular resistance differed between the groups.

Conclusion: Cardiopulmonary bypass flow rate of $110 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ was associated with higher positive net fluid balance and fluid extravasation rate than $80 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. The effect was mainly observed in the initial phase of cardiopulmonary bypass.

From the Department of Anesthesia and Intensive Care^a and the Section for Thoracic Surgery, Department of Heart Disease,^b Haukeland University Hospital; the Surgical Research Laboratory, Department of Surgical Sciences^c; and the Department of Oral Science, Dental Research,^d University of Bergen, Bergen, Norway.

This study was financially supported by The Western Norway Regional Health Authority, The Norwegian Council on Cardio-

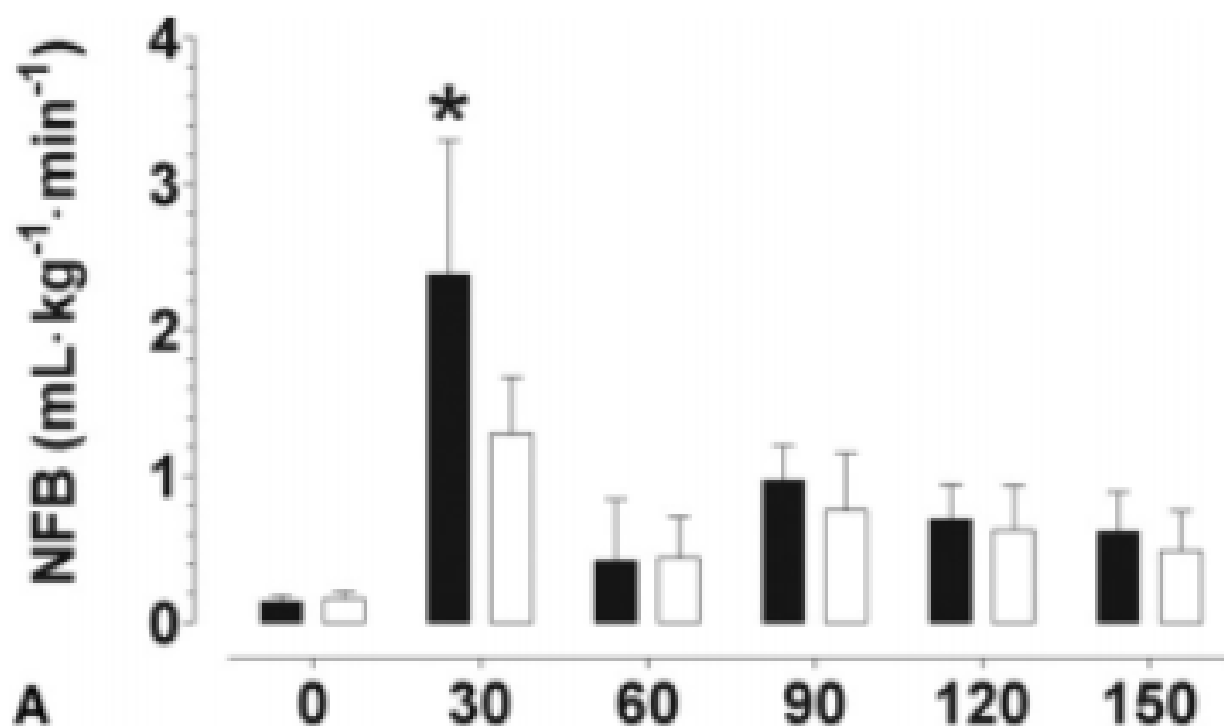


Figure 1. Net fluid balance (*NFB*) (A), plasma volume (B), and fluid extravasation rate (*FER*) (C) throughout 30-minute intervals during 60 minutes of normothermic cardiopulmonary bypass (*CPB*) followed by 90 minutes of hypothermic *CPB*. Solid black columns and squares, High-flow group. White columns and squares, Low-flow group. The values are as mean \pm SD. * $P < .05$ (between-group comparison).

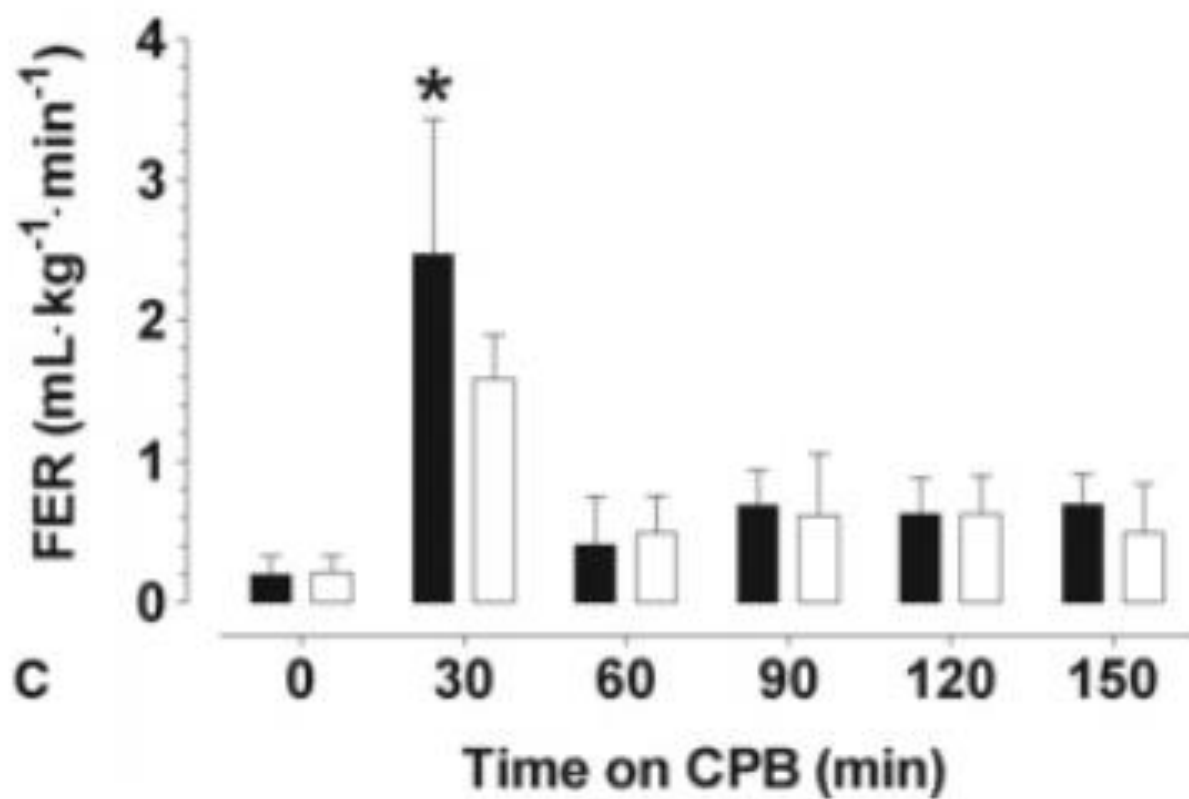


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Pump Flow Changes Do Not Impair Sublingual Microcirculation During Cardiopulmonary Bypass

Alessandro Forti, MD,* Alessandra Comin, PhD,* Nadia Lazzarotto, MD,* Giuseppe Battistella, MD,† Valeria Salandin, MD,* and Carlo Sorbara, MD*

Table 3. Microvascular Variables of Patients at the 2 Different CPB Flows Considered

| | CPB Flow 80% | CPB Flow 100% |
|---------------------------|--------------|---------------|
| DBS | 11.36 ± 2.56 | 11.36 ± 2.43 |
| TVD (mm/mm ²) | 20.45 ± 3.50 | 20.32 ± 3.58 |
| MFI | 2.91 ± 0.25 | 2.98 ± 0.25 |
| PVD (mm/mm ²) | 17.88 ± 3.51 | 18.04 ± 3.47 |
| PPV (%) | 87.58 ± 9.27 | 88.88 ± 8.29 |

NOTE. Data are presented as the mean ± standard deviation.

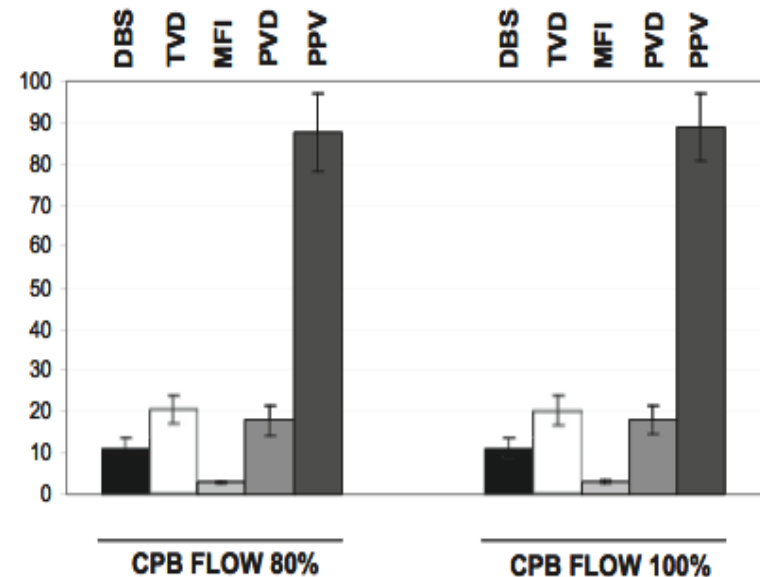


Fig 3. A graphic representation of the evaluated microdynamic parameters at the CPB flow rate of 80% and 100%. Each microcirculatory parameter is shown as an average value ± the relative standard deviation. None of the indices reveals a statistically significant difference as the pump flow rate changed.

Physiology of Cardiopulmonary Bypass

Pressure & Flow

Modern CPB machines pump blood into the arterial system using a *centrifugal pump*. The flow of a centrifugal pump increases as its angular velocity (RPMs) increases. Pressures are therefore a function of the set flow rate and the resistance to flow, and can thus be modified by changing either the flow rates or the vascular resistance (ex. phenylephrine, vasopressin, phentolamine). That said, **CPB machines cannot increase flows *ad infinitum*** – 1) centrifugal pumps are afterload-sensitive and plateau as SVR increases 2) CPB flows are limited by venous return, as past a certain point collapse of veins limits VR, lowering CPB reservoirs, and potentiating entrainment of air and 3) depending on the size of the arterial cannula, excessive flow may require excessive line pressure, leading to tissue (blood) trauma

Average flows for a normal adult (assuming a hemoglobin of 8 mg/dL and a temperature of 30C, i.e. moderate hypothermia) might be **2.4 L/min/m²**, although this will be adjusted based on age, hemoglobin, temperature, and depth of anesthesia.

Pressure vs. Flow

Fundamental to CPB is the delivery of oxygen to organs that need it. Because flow is a function of pressure gradient and vascular resistance, from a purely teleological standpoint, it would appear that maximizing blood flow (as opposed to maximizing pressure, which may or may not maximize flow, depending on vascular resistance) would be the most rational approach to CPB management. That said, despite over 50 years of experience and research, the ideal blood flow and/or perfusion pressure is not known. Nor is it known whether flow or pressure is more important

There are several problems with maximizing flows. First, higher flow rates are associated with significant trauma (to hematologic elements) and increase the inflammatory response to CPB. Second, increased flows carry an increased embolic burden. Third, an increase in total flows does not assure increased DO₂ to organs of interest, because the CPB machine cannot alter regional flows. Thus, if CPB flow rates are increased by perfusing relatively unimportant tissue beds, the increase in trauma/inflammation cannot be justified and might be harmful. With regards to the latter, Slater et al studied regional flow rates in pigs on CPB, and found that regional flows to the brain, kidney, and pancreas all decreased when flows were increased from 1.6 to 1.9 L/min/m² [Slater JM et al. Ann Thorac Surg 72: 542, 2001]. Indeed, at these high levels, brain and kidney flows were at 65% and 55% of pre-CPB flow rates, respectively. This data is similar to Rogers et al.'s human data which showed that increasing CPB from 1.75 to 2.25 L/min/m² has no effect on cerebral blood flow or cerebral metabolic rate consumption in humans [Rogers AT et al. J Thorac Cardiovasc Surg 103: 363, 1992], and consistent with Govier et al.'s data which showed that changes in flow rates from 1.0 to 2.0 L/min/m² had no effect on CBF during hypothermic CPB in humans [Govier AV et al. Ann Thorac Surg 38: 592, 1984]

Arguments can be made in favor of optimizing pressure at the expense of flows – first, **cerebral autoregulation maintains constant cerebral blood flow (CBF) from MAPs of 50-150 mm Hg** (and possibly lower during hypothermic CPB). Hypertensive patients are *thought* to have a right-shifted autoregulation curve, and diabetics may have altered autoregulation, thus increased pressures (or flows) can potentially be justified in these patients. Second, and related, **while the CPB machine can manipulate total flows, it cannot individually affect regional flows** – this ability is left to the organs themselves. Under CPB, end organs have only one mechanism by which they can modulate regional flows – alterations in regional vascular resistance (RVR). The higher the pressure, the broader the range of regional flows available to each individual organ. Note that this concept has not been proven (neither have concepts in support of a flow-based approach to CPB) and is offered only as a possible explanation for the failure of the flow-based approach to prove itself clinically superior

High flow rates are also disadvantageous in that they increase suture line strain, increase bronchial flow to the lungs, and increase collateral flow to the heart (which diminishes the duration of effective cardioplegia)

Considerations against CPB Flow Rates Increased hematologic trauma Increased stress response Strain on suture lines Increased pulmonary shunting Accelerated washout of cardioplegia May not affect regional flow advantageously [Slater JM et al. Ann Thorac Surg 72: 542, 2001] No data to support it

“Low Flow” Cardiopulmonary Bypass

The major putative advantages of “low flow” CPB are reductions in hematologic stress and reduced embolic load. Note, however, that there are no prospective, randomized, controlled trials that adequately address the lower limit of acceptability

Alterations in Afterload

Decreased SVR is the predominant cause of hypotension following initiation of CPB (secondary to reduced blood viscosity, dilution of endogenous catecholamines in priming solution, and differences in pO₂, pH, and electrolyte concentrations between the priming solution and native blood). **As CPB progresses, SVR gradually increases**, eventually to supranormal levels. This is presumably due to hypothermia (leading to vasoconstriction and catecholamine release), stress response (also leading to vasoconstriction and catecholamine release), and vessel closure (i.e. maldistribution of flow).

Microperfusion and Pulsatility

Clearly *altered by temperature changes, edema, loss of pulsatility, RBC injury, emboli, and the inflammatory response*. Some perfusionists will try to counteract these changes by using mannitol, vasodilators, hemodilution, microfiltration, and pulsatile perfusion techniques. Pulsatile flow is difficult to achieve in the aorta, as the arterial lines significantly dampen the oscillatory component of pulsatile pressure. In the absence of any convincing data to support its use, **pulsatile pumps, despite being more “physiologic,” cannot be justified**

Sonuç

- Kabul edilebilir sınırlar içindeki POMPA AKIMI da
 - Kişiselleştirilmiş fizyolojik farklılıklar nedeni ile
 - Güvenli olmayabilir
 - Şeytan buradadır ??????

- Ortalama arter basıncı ve kalp/EKD debisi ile ilgili belirsizliklere;
 - Yaş, DM, PAH ve kalsifik aort gibi diğer nedenler de eklendiğinde,
- Postoperatif dönemde % 30-60 oranında nörokognitif fonksiyon bozukluğunun (POCD) görülmesi kaçınılmaz olmaktadır.

POCD

- En önemli iki nedeni
 - Embolizasyon
 - Serebral hipoperfüzyondur.
Diegeler A. Ann Thorac Surg 2000;69:1162-6.
- Bilinen risk faktörleri;
 - İleri yaş
 - Sistemik inflamasyon
 - DM
 - Şiddetli PAH
 - Cerrahi tipi (prosedür)
- *Hong SW Eur. J Cardiothorac Surg 2008;33:560-65.*

Nörolojik hasar- yaş ilişkisi

- Yaş < 64 ise nörolojik hasar riski %1 den az
- Yaş 65-75 ise %5
- Yaş > 75 ise % 7-9

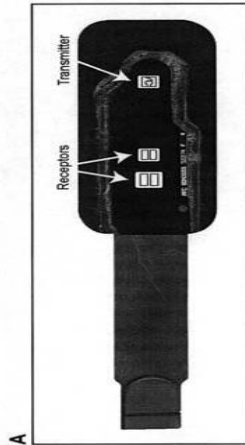
Hong SW Eur. J Cardiothorac Surg
2008;33:560-65

- Tüm bu sonuçlar;
- Riskli hastalar ve majör cerrahi uygulamalarında **GELİŞMİŞ** bir monitorizasyonu gerekli kılmaktadır
- Bu ilave monitorizasyon
 - Non-invazif
 - Kolay uygulanabilir olmalı ve
 - Bölgesel değişiklikleri (serebral) yansıtmalı

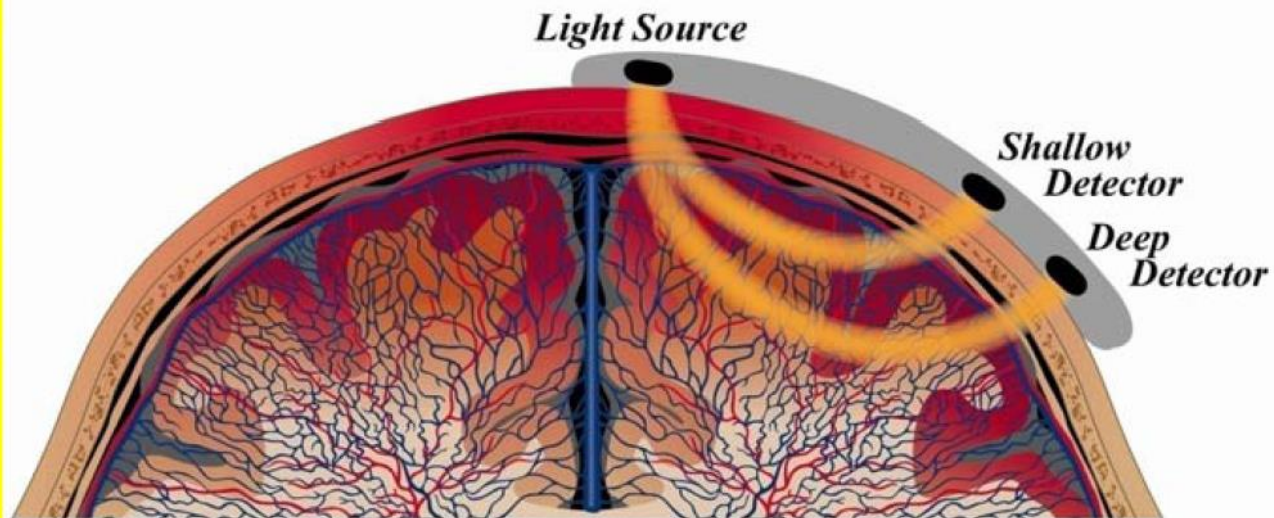
Gelişmiş monitorizasyon

–Near Infrared Spectroscopy
(NIRS)

INVOS 5100 C System



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.

rSO2' deki anlamlı deęişim

- INVOS;
 - Bazal deęerden %20 den fazla düşme
 - %50- ölçülen deęer(%) x saniye >3000 %sn
 - 50-35 x 300 =4500 %sn

Goldman S. Heart Surg Forum 2004;7:E376-81.

- rSo₂ uygulanan 1034 vaka ile (Grup I)
- rSo₂ uygulanmayan 1245 (Grup II) açık kalp cerrahisi vakasını karşılaştırmış
- Grup I: hastalarında;
 - Serebral oksijen sunumu/Oksijen tüketimi
 - Fio₂
 - Baş ve kanul pozisyonu ayarı
 - PCO₂
 - MAP
 - Pompa akımı
 - Anestezi derinliği
 - SVR düzenlemesi (NTG, SNP,Arterenol)
 - ES transf.

Sonuç parametreleri

- Kalıcı inme % 0,97 vs %2,25 $p<0,044$
- Uzamış ventilasyon %6,8 vs % 10,6
 $p<0,001$
- Risk matcah (NYHA class) uzamış
hastanede kalış süresi (5. günden sonraki)
0,2 gün vs 2,3 gün $p< 0,046$

Efficacy of Near-Infrared Spectrometry for Monitoring the Cerebral Effects of Severe Dilutional Anemia

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ABSTRACT

Introduction: Clear guidelines for red cell transfusion during cardiac surgery have not yet been established. The current focus on blood conservation during cardiac surgery has increased the urgency to determine the minimum safe hematocrit for these patients. The aim of this study was to determine whether monitoring of cerebral regional oxygen saturation (rSO₂) via near-infrared spectrometry (NIRS) is effective for assessing the cerebral effects of severe dilutional anemia during elective coronary arterial bypass graft surgery (CABG).

Methods: The prospective observational study involved patients who underwent cerebral rSO₂ monitoring by NIRS during elective isolated first-time CABG: an anemic group (N=15) (minimum Hemoglobin (Hb) <7 g/dL at any period during cardiopulmonary bypass (CPB) and a control group (N=15) (Hb >8 g/dL during CPB). Mean arterial pressure (MAP), pump blood flow, blood lactate level, pCO₂, pO₂ at

INTRODUCTION

Hemodilution and anemia during cardiovascular surgery are associated with postoperative morbidity and mortality [Goodnough 1989]. There has been much debate regarding the minimum safe hematocrit during CPB. The limited experimental available data suggests that hematocrit as low as 10% may be safe during hypothermic CPB [Rand 1964]; however, clear guidelines for red cell transfusion during CPB have not yet been established. Surgeons and anesthesiologists frequently rely on personal experience and anecdotal accounts to develop their own transfusion practices during CPB [Goodnough 1989; Gordon 1975; Kuduvali 2005]. The current emphasis on blood conservation during cardiac surgery has increased the need to establish the minimum safe hematocrit for this patient group. The systemic response to hemodilution and anemia under non-CPB conditions is well described. Under physiologic conditions, levels of systemic and regional oxygen consumption are independent of

Table 2. Group results for hemoglobin level prior to anesthetic induction, minimum hemoglobin during extracorporeal circulation, and hemodynamic and arterial blood gas values at time of minimum Hb during extracorporeal circulation.

| | Anemic group (N=15) | Control group (N=15) | <i>P Value</i> |
|--|------------------------|-------------------------|----------------|
| Hemoglobin (g/dL) at T1 | 10.3 ± 0.9 | 14.2 ± 1.3 | .001 |
| Min. Hemoglobin during extracorporeal circulation (g/dL) | 6.2 ± 0.4 | 10.3 ± 1.3 | .001 |
| Mean arterial pressure (mmHg) | 65 ± 5 | 56 ± 7 | .001 |
| Lactate level (mmol/dL) | 1.1 ± 0.3 | 1.1 ± 0.5 | NS |
| Pump blood flow (L/min/m ²) | 2.5 ± 0.2 | 2.2 ± 0.2 | .001 |
| pO ₂ (mmHg) | 202 ± 43 | 145 ± 42 | .001 |
| pCO ₂ (mmHg) | 33 ± 3 | 39 ± 2 | .001 |

pO₂: partial oxygen pressure; pCO₂: partial carbon dioxide pressure; T1: prior to anesthetic induction; NS: Non-sufficient.

Table 3. Oxygen saturation findings and their change in time for the right and left cerebral hemispheres, respectively, in the two study groups.

| | Anemic group (N=15) | Control group (N=15) |
|--|------------------------|-------------------------|
| Right hemisphere: | | |
| rSO ₂ (%) at T1 | 52 ± 9 | 66 ± 6 |
| rSO ₂ (%) at time of lowest Hemoglobin during ECC | 49 ± 7 | 58 ± 6 |
| Change in rSO ₂ from T1 to lowest Hemoglobin during ECC (%) | 5.7 | 12 |
| Left hemisphere: | | |
| rSO ₂ (%) at T1 | 54 ± 6 | 69 ± 7 |
| rSO ₂ (%) at time of lowest Hemoglobin during ECC | 50 ± 7 | 58 ± 6 |
| Change in rSO ₂ from T1 to lowest Hemoglobin during ECC (%) | 7.4 | 11.5 |

ECC: extracorporeal circulation; rSO₂: regional oxygen saturation; T1: prior to anesthetic induction.

Table 4. Interventions required during cardiopulmonary bypass for each patient in the anemic group.

| Patient no. | Increased FiO ₂ | Increased pump blood flow | Noradrenaline boluses to achieve MAP >60 mmHg | Crystalloid administration | Erythrocyte transfusion |
|-------------|----------------------------|---------------------------|---|----------------------------|-------------------------|
| 1 | | + | | + | |
| 2 | | | + | | |
| 3 | + | + | | | |
| 4 | + | + | | | |
| 5 | + | + | | | |
| 6 | | + | | + | |
| 7 | + | | | | |
| 8 | | | | | |
| 9 | | + | | + | |
| 10 | + | + | | + | |
| 11 | | + | + | | + |
| 12 | + | + | | + | |
| 13 | | | | | |
| 14 | | + | | + | |
| 15 | + | + | | | |

FiO₂: inspiratory oxygen fraction; MAP: mean arterial pressure.

As a conclusion

- Our findings suggest that NIRS monitoring of cerebral rSO₂ is an effective method that can assist in decision-making related to blood transfusions aimed at addressing dilutional anemia during CPB



RESEARCH ARTICLE

Open Access

Monitoring of brain oxygen saturation (INVOS) in a protocol to direct blood transfusions during cardiac surgery: a prospective randomized clinical trial

George Vretzakis¹, Stavroula Georgopoulou¹, Konstantinos Stamoulis¹, Vassilios Tassoudis¹, Dimitrios Mikroulis², Athanasios Giannoukas³, Nikolaos Tsilimingas⁴ and Menelaos Karanikolas^{5*}

Table 3 Hematocrit values, intravenous fluids and fluid balance by group

| Hematocrit values (%) | Group A (INVOS, n = 75) | Group B (Control, n = 75) | P |
|--------------------------------|------------------------------------|--------------------------------------|----------|
| Preoperative | 39.54 ± 3.90 | 40.38 ± 4.53 | 0.246 |
| After arterial line placement | 38.45 ± 4.32 | 38.68 ± 4.40 | 0.765 |
| After anesthesia induction | 38.19 ± 4.61 | 37.84 ± 4.53 | 0.655 |
| After first cardioplegia | 20.20 ± 3.60 | 20.16 ± 3.83 | 0.947 |
| End of CPB | 23.07 ± 3.45 | 23.26 ± 3.03 | 0.721 |
| End of operation | 27.55 ± 4.18 | 27.50 ± 4.15 | 0.943 |
| 6 hours in the ICU | 28.15 ± 3.38 | 28.79 ± 3.32 | 0.263 |
| 12 hours in the ICU | 28.61 ± 3.77 | 29.29 ± 3.58 | 0.254 |
| Day of discharge | 30.67 ± 3.07 | 31.28 ± 2.58 | 0.193 |
| Fluid balance (ml) | | | |
| IV fluids to initiation of CPB | 368.5 ± 177.0 | 416.4 ± 184.6 | 0.101 |
| Urine to initiation of CPB | 110.8 ± 95.9 | 135.7 ± 127.6 | 0.164 |
| Fluid balance | | | |
| After 1st cardioplegia | 2240.2 ± 238.8 | 2326.0 ± 306.4 | 0.055 |
| Urine output during CPB | 666.2 ± 594.0 | 694.0 ± 423.0 | 0.743 |
| Total urine output | 1326.2 ± 842.2 | 1419.3 ± 690.7 | 0.452 |
| Use of filter, n (%) | 8 (10.6%) | 9 (12.0%) | 0.796 |
| Overall fluid balance | 685.4 ± 784.1 | 809.9 ± 651.1 | 0.290 |

Table 4 Transfusion data by group (analysis based on “intention to treat”)

| | Group A (n = 75) | Group B (n = 75) | P |
|-----------------------|------------------|------------------|--------------|
| In OR | | | |
| RBC units transfused | 18 | 40 | |
| Patients transfused | 14 (18.6%) | 25 (33.3%) | <i>0.040</i> |
| RBC per transfused pt | 1.29 ± 0.47 | 1.60 ± 0.58 | <i>0.090</i> |
| RBC/pt overall | 0.24 ± 0.54 | 0.53 ± 0.84 | <i>0.011</i> |

Conclusions

This prospective randomized clinical study suggests that the use of cerebral oximetry (INVOS) as part of an algorithm to guide RBC transfusions can result in significant reduction of RBC use in patients undergoing elective cardiac surgery, when the established protocol for fluid restriction and decision to transfuse is properly followed. However, the observed benefit is no longer significant when protocol violations are included in the analysis (“intention to treat” analysis). We suggest that, based on these results, INVOS could be a useful tool for monitoring patients during cardiac surgery, but data from well designed clinical trials with rigorous attention to study protocol, in an attempt to minimize protocol violations are needed to better assess the validity of our findings.

The role of regional cerebral oxygen saturation on adjustment of fraction of inspired oxygen during coronary artery bypass graft surgery

Koroner arter baypas greft cerrahisi sırasında inspire edilen oksijen fraksiyonunun ayarlanmasında rejyonel serebral oksijen satürasyonunun rolü

**Fevzi Toraman,¹ Şahin Şenay,² Zehra Serpil Ustalar Özgen,¹ Ebuzer Aydın,²
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Background: The aim of the study is to detect the regional cerebral oxygen saturation by the help of near infrared spectroscopy (NIRS) monitorization and to diagnose the hypoxemic or hyperoxemic episodes for the assessment of the effects of hypoxia episodes on cerebral perfusion during cardiopulmonary

Amaç: Bu çalışmada yakın kızılötesi spektroskopisi (NIRS) monitörizasyonu ile rejyonel serebral oksijen satürasyonunun saptanması ve kardiyopulmoner baypas (KPB) sırasında hipoksi epizotlarının serebral perfüzyon üzerindeki etkilerinin değerlendirilebilmesi için mümkün olduğunca en kısa sürede hipoksemik

Ekstrakorporeal dolařım (EKD) sırasında

- İnspire edilen oksijen fraksiyonu (F_{iO_2})
 - Genelde %70-100 aralıęında tutulmakta,
 - Ancak bu uygulama sırasında hiperoksemi ($P_{O_2} > 180$ mmHg) görölmektedir.
 - Hiperoksemi;
 - Eritrosit reolojisini bozarak
 - Mikrosirkülasyonun bozulmasına ve
 - End organ patolojilerinin gelişmesine neden olmaktadır

– Belboul A. J Extra Corpor Technol 1991;23:43-8.

Materyal-metot

- Grup I: Normotermi ve Hipotermi de F_{iO_2} :0,35, yeniden ısınma da F_{iO_2} :0,45 olacak şekilde ayarlanan 35 hastadan,
- Grup II: Normotermi ve Hipotermi de F_{iO_2} :0,40, yeniden ısınma da F_{iO_2} :0,50 olacak şekilde ayarlanan 35 hastadan,
- Tüm hastaların standart monitorizasyonuna ilaveten serebral oksijen saturasyonu (INVOS SOMANETICS 5000 C) takipleri yapıldı.

Amaç

- F_{iO_2} %35-45 aralığında iken oluşabilecek hipoksi atağının serebral oksijen monitorizasyonu (rSO_2) ile tespiti ve
- F_{iO_2} %40-50 aralıklarında iken oluşan hiperoksi oranını ve bunun rSO_2 ' e etkisini araştırmayı amaçladık.

Table 1. Demographic data

| | Group 1 (F _I O ₂ : 0.35-0.45) (n=35) | | | Group 2 (F _I O ₂ : 0.40-0.50) (n=35) | | | <i>p</i> |
|--|---|----|---------|---|----|---------|----------|
| | n | % | Mean±SD | n | % | Mean±SD | |
| Age (years) | | | 60±10 | | | 57±11 | NS |
| Gender | | | | | | | |
| Female | 12 | 35 | | 18 | 50 | | NS |
| Male | 23 | | | 17 | | | |
| Cardiopulmonary bypass time (minutes) | | | 66±22 | | | 62±23 | NS |
| Cross-clamp time (minutes) | | | 43±20 | | | 40±18 | NS |

FiO₂: Fraction of inspired oxygen; SD: Standard deviation; NS: Not significant.

Table 2. The values of cerebral cortical oxygen saturation

| | Group 1 | Group 2 | <i>p</i> | Group 1 | Group 2 | <i>p</i> | Group 1 | Group 2 | <i>p</i> |
|----------------|-------------------|-------------------|----------|-------------------|-------------------|----------|---------|---------|----------|
| | RScO ₂ | RScO ₂ | | LScO ₂ | LScO ₂ | | Hct | 4Hct | |
| | (%) | (%) | | (%) | (%) | | (%) | (%) | |
| T ₁ | 64±9 | 60±10 | NS | 65±9 | 62±10 | NS | 39±6 | 39±6 | NS |
| T ₂ | 56±8 | 55±9 | NS | 56±8 | 54±12 | NS | 27±6 | 27±6 | NS |
| T ₃ | 53±7 | 54±8 | NS | 55±8 | 54±9 | NS | 27±6 | 27±6 | NS |
| T ₄ | 53±8 | 53±9 | NS | 54±9 | 55±8 | NS | 27±7 | 28±6 | NS |
| T ₅ | 54±8 | 54±8 | NS | 56±9 | 56±9 | NS | 28±6 | 28±6 | NS |

RSc: Right cerebral oxygen saturation; Hct: Hematocrit; NS: Not significant.

Table 4 . The hemodynamic parameters

| | Group 1 | Group 2 | <i>p</i> | Group 1 | Group 2 | <i>p</i> | Group 1 | Group 2 | <i>p</i> |
|----------------|---------|---------|----------|----------|----------|----------|------------------------|------------------------|----------|
| | MAP | MAP | | Lactate | Lactate | | CI | CI | |
| | (mmHg) | (mmHg) | | (mmol/l) | (mmol/l) | | (L/dk/m ²) | (L/dk/m ²) | |
| T ₁ | 78±14 | 75±11 | NS | 0.9±0.3 | 0.9±0.3 | NS | – | – | |
| T ₂ | 65±17 | 71±13 | NS | 1±0.4 | 1.1±0.4 | NS | 2.5±0.2 | 2.3±0.2 | 0.001 |
| T ₃ | 66±17 | 68±19 | NS | 1±0.4 | 1.1±0.4 | NS | 2.4±0.3 | 2.2±0.2 | 0.034 |
| T ₄ | 51±12 | 59±15 | 0.015 | 1.2±0.5 | 1.2±0.4 | NS | 2.3±0.3 | 2.3±0.2 | NS |
| T ₅ | 53±12 | 55±15 | NS | 1.3±0.6 | 1.3±0.6 | NS | 2.3±0.3 | 2.4±0.3 | NS |

MAP: Mean arterial pressure; CI: Confidence interval; NS: Not significant.

Sonuçlar

| | G I (Fio2: 0,35-0,45) (n=35) | GII (Fio2: 0,40-0,50) (n=35) |
|--------------------------------------|---|---|
| Hipoksi (PO ₂ <80mmHg) | 5 (%14) | - |
| Hiperoksi (PO ₂ >180mmHg) | 8 (%23) | 15 (%43) |
| rSO ₂ deki azalma >%20 | 12 (%34) | 8 (23) |

Müdahale sonrası

| | G I (Fio2: 0,35-0,45) (n=35) | GII (Fio2: 0,40-0,50) (n=35) |
|---|---|---|
| Hipoksi (PO ₂ <80mmHg) | 0 | - |
| rSO ₂ deki azalma >%20 | 7 (%20) | 8 (23) |
| Hiperoksi (PO ₂ >180mmHg) | 8 (%23) | 15 (%43) |

Tartışma

- EKD sırasında hipoksinin tespitinde ve F_{iO_2} nin ayarlanmasında faydalı olduđu kanısına vardık

Utility of Cerebral Oxymetry for Assessing Cerebral Arteriolar Carbon Dioxide Reactivity during Cardiopulmonary Bypass

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ABSTRACT

Background: Our study evaluated changes in cerebral arterial oxygen saturation (rSO₂) during cardiopulmonary bypass (CPB) that were caused by changes in arterial carbon dioxide tension (PaCO₂).

Methods: A group of 126 patients undergoing routine, elective, first-time coronary artery bypass graft surgery (CABG) was entered into a prospective study using bilateral near-infrared spectroscopy (NIRS) before anesthetic induction (T1), after anesthetic induction (T2), and continuing at 5-minute intervals during moderate hypothermic (32°C) CPB. Pump flows were set at 2.5 L/min/m² and adjusted to maintain mean arterial pressure (MAP) within 10 mmHg of the MAP recorded at the initial fifth minute of CPB (T3). Thirty-two patients were excluded from data collection because MAP could not be stabilized within the target range of 60-90 mmHg. In the remaining 94 patients, after obtaining steady state flow, MAP, and oxygenation, a trial period

INTRODUCTION

Cerebral blood flow (CBF) and distribution are highly sensitive to changes in the arterial carbon dioxide tension (PaCO₂) [Laffey 2002a]. Elevated PaCO₂ (hypercapnia) leads to decreased cerebrovascular resistance and consequent increases in CBF and cerebral oxygen delivery (DO₂), whereas hypocapnia leads to increased cerebrovascular resistance and related decreases in CBF and DO₂ [Laffey 2002a]. This local control process, known as cerebrovascular carbon dioxide reactivity, provides a vital homeostatic function that helps regulate not only the peripheral arterial pH, but also the resistance of cerebral arterioles, which directly affects CBF and DO₂. In the normothermic life state, this control process usually maintains adequate arterial flow to the brain. During open-heart surgery, a patient's body temperature, continuous blood flow, fresh gas flow, and arterial blood-gas concentrations shift due to alterations related to cardiopulmonary bypass (CPB); consequently, maintaining adequate

EKD sırasındaki PCO_2 değeri;

- Taze gaz akım hızına (TGAH),
 - Vücut yüzey alanına ve
 - Vücut ısısına bağlı olarak değişir.
-
- Bu nedenle EKD sırasında PCO_2 değerinin belli aralıkta (35-45 mmHg) tutulması çok önemlidir.

Amaç

- Biz bu çalışmamızda sabit
 - OAB ve pompa akımı değerlerinde;
- Değişen PCO_2 değerlerinin,
 - Serebral arterioller üzerine olan etkisini,
 - Near infrared spektroskopisi (NIRS) yöntemi ile değerlendirmeyi amaçladık.

| | T1 Anestezi öncesi | T3 (KPB 5. dk) TGAH 2.5l/dk./m2 | T4 (KPB 10.dk) TGAH 2.5l/dk./m2 | T5 (KPB 20.dk) TGAH 0.75l/dk./m2 |
|-----------------------------------|--------------------------|---------------------------------------|--|---|
| PCO ₂ (mmHg) | 38 ± 4 | 35 ± 3 ^{βα} | 30 ± 2 [€] | 41 ± 4 |
| LScO ₂ (%) | 63 ± 7 | 59 ± 7 ^β | 56 ± 7 [€] | 60 ± 7 |
| Bazal değerlere göre % değişim | | -6 ± 9 ^β | -10 ± 9 [€] | -5 ± 9 |
| RScO ₂ (%) | 63 ± 7 | 59 ± 7 ^β | 56 ± 7 [€] | 60 ± 7 |
| Bazal değerlere göre % değişim | | -6 ± 9 ^β | -10 ± 9 [€] | -4 ± 9 |

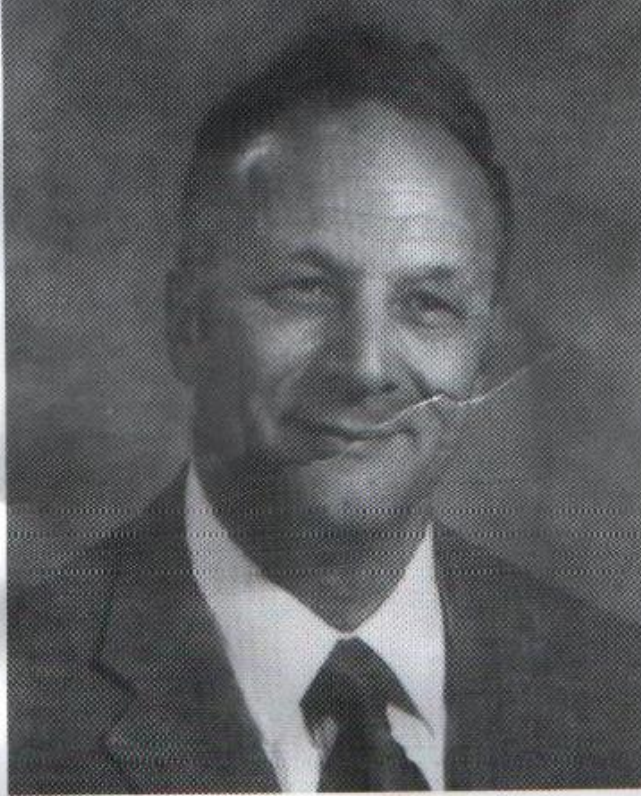
TGA: Taze gaz akımı, **β**: T3 vs T4, P<0,001, **α**: t3 vs T5, P<0,005, **€**:T4 vs T5, P<0,001

Tartışma

- KPB sırasındaki sabit akım ve basınç değerlerinde;
- PCO_2 değerindeki minör değişiklikler, serebral arterioler damar direncinde önemli değişiklikler oluşturabilmekte
- Bu da serebral regional oksijen saturasyonu değerinin değişmesine neden olmaktadır.

ŞEYTAN

- Hep,
 - Tahmin ettiğimiz,
 - Düşündüğümüz yerdedir
- Yeter ki biz onu görmek isteyelim
- Unutmayalım ki
 - Görmek istemek,
 - Görmenin
 - Aramak
 - Bulmanın yarısıdır.



Ahmet Ercüment KOPMAN
1920 - 2002