DRUCK IST NICHT FLUSS -
…. aus Sicht der Makrohämodynamik

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Declaration of Interest

- No conflict of interest to declare

"A cat killer? Is that the face of a cat killer? Cat chaser maybe. But hey—who isn't?"

Druck ist nicht gleich Fluss

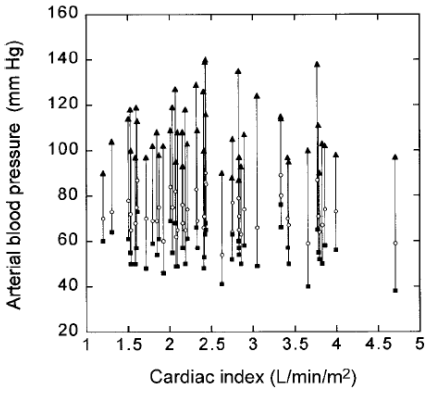
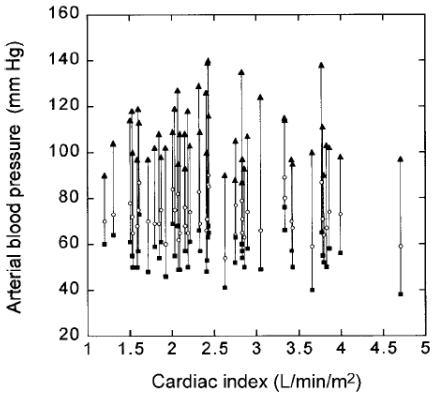


Fig 2. Systolic (▲), mean (○), and diastolic (■) pressures plotted against cardiac index. Least-squares regression indicated no significant correlation between cardiac index and systolic (r^2 = 0.005), mean (r^2 = 0.014), or diastolic (r^2 = 0.043) pressure.

Stephen Hales, 1733

Linton RF. J Cardiothorac Vasc Anesth 2002; 16:4-7

Druck oder Fluss?



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Fluss = Volumen pro Zeit

Druck = Wandspannung auf die Gefäße

Perfusiondruck

Definition of Hypotension:

- **keine einheitliche Definition (absolut vs. relativ; Dauer)**
- **Mitteldruck vs. Perfusionsdruck**
- **Druck an der Maschine; postoperativ auf der Intensiv**

Inzidenz:

- **abhängig von der Definition**
- **abhängig vom Eingriff**

Outcome:

- **?**

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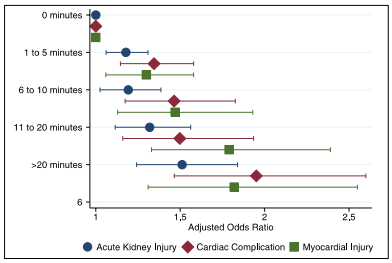
Hypotension während der Anästhesie

Time MAP <55 mmHg (min)	Adjusted Odds Ratio (95% CI)			
	Acute Kidney Injury	Myocardial Injury	Cardiac Complication	30-day Mortality
0	Referent			
1–5	1.18 (1.06–1.31)	1.30 (1.06–1.58)	1.35 (1.15–1.58)	1.16 (0.91–1.46)
6–10	1.19 (1.03–1.39)	1.47 (1.13–1.93)	1.46 (1.17–1.83)	1.16 (0.84–1.60)
11–20	1.32 (1.11–1.56)	1.79 (1.33–2.39)	1.50 (1.16–1.94)	1.26 (0.89–1.80)
>20	1.51 (1.24–1.84)	1.82 (1.31–2.55)	1.95 (1.46–2.60)	1.79 (1.21–2.65)



Walsh: 33.000 patients
MAP < 55 mmHg

- Acute kidney injury
- Perioperative myocardial injury
- Increased mortality

Walsh M Anesthesiology 2013;119:507-515

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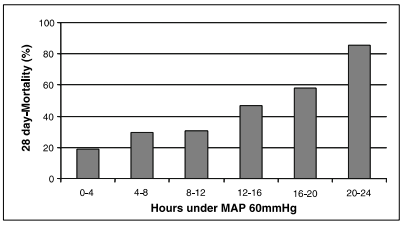
Perfusionsdruck auf der Intensiv



Retrospective cohort study
274 patients with sepsis from PDMS

Perfusion Pressure > 45 mmHg
MAP > 60 mmHg

	Survivors		Nonsurvivors		
	Mean ± SD	CI 95%	Mean ± SD	CI 95%	*P* value
MAP <60 mmHg/h (min)	9 ± 13	7–11	22 ± 20	17–26	<0.001*
MAP <60 mmHg/day (min)	195 ± 272	157–233	455 ± 433	356–554	<0.001*

Dünser M. Intensive Care Med 2009; 35:1225-33

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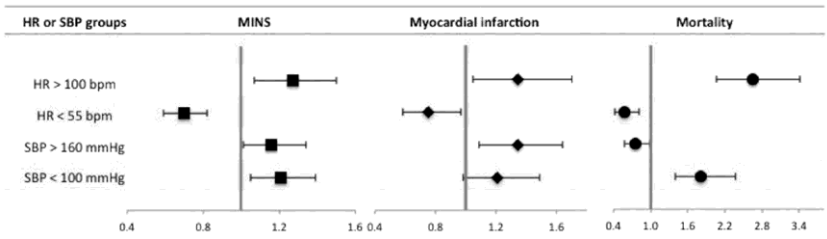
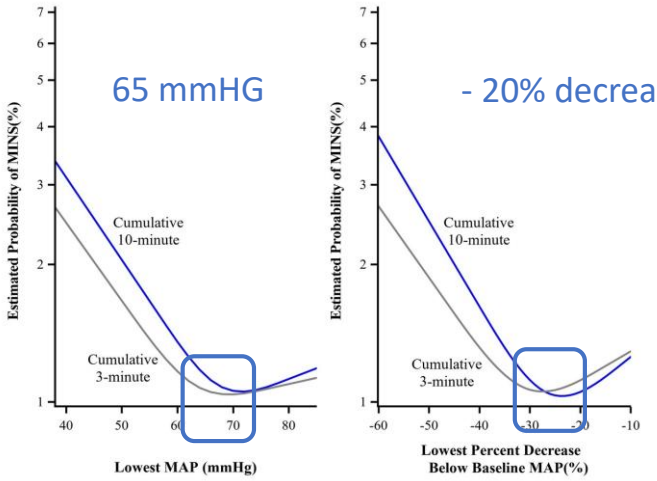
A Prospective International Multicentre Cohort Study of Intraoperative Heart Rate and Systolic Blood Pressure and Myocardial Injury After Noncardiac Surgery: Results of the VISION Study

Tom E. F. Abbott, MRCP,* Rupert M. Pearse, MD,* R. Andrew Archbold, MD,† Tahania Ahmad, MPH,* Edyta Niebrzegowska, MSc,† Andrew Wragg, FRCP,† Reitze N. Rodseth, PhD,‡ Philip J. Devereaux, PhD,§ and Gareth L. Ackland, PhD*



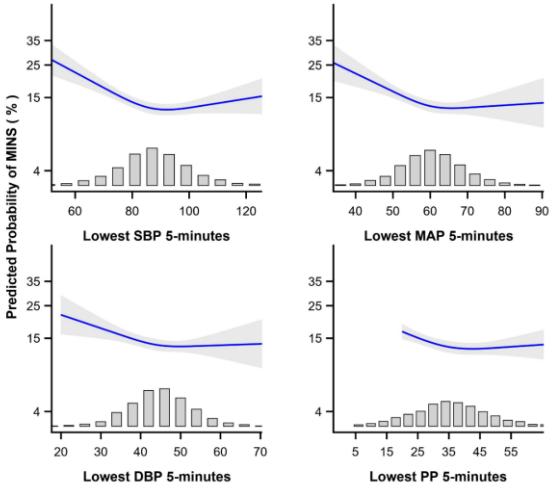
Abbott TEF Anesth Analg 2018;126:1936-1945

Intraoperative blood pressure & MINS



Salmasi, *et al*., 2016, Anesthesiology

Which BP component ?

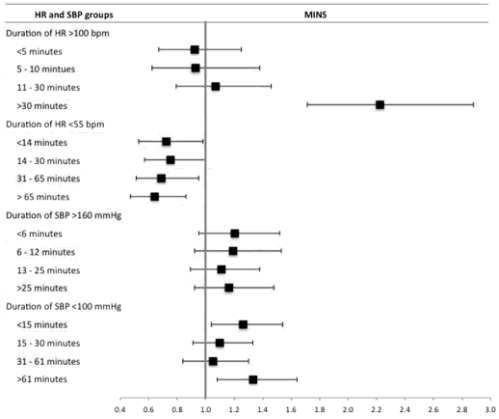


Systolic 90 mmHg

MAP 65 mmHg

Ahuja, *et al*., 2019, Anesthesiology

Determinant Time of Tachycardia



Abbott TEF Anesth Analg 2018;126:1936-1945

Original Article

Prolonged Tachycardia with Higher Heart Rate Is Associated with Higher ICU and In-hospital Mortality

Masao Hayashi[a]*, Arata Taniguchi[a], Ryuji Kaku[a], Shusaku Fujimoto[b], Satoshi Isoyama[a], Sei Manabe[c], Tsubasa Yoshida[d], Satoshi Suzuki[a], Kazuyoshi Shimizu[a], Hiroshi Morimatsu[a], and Ryusuke Momota[c]

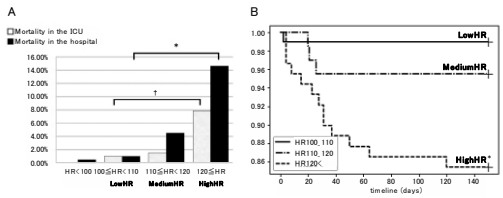


Fig. 2 A. The mortality in the ICU and in the hospital. The patients were stratified by HR alone as LowHR (100 ≦ HR ＜110), MediumHR (110 ≦ HR ＜120), and HighHR (HR ≧ 120). Both the ICU and in-hospital mortality rates were significantly higher in the HighHR group compared to the LowHR group. † $p < 0.05$, ✽ $p < 0.01$ by Fisher's exact test; B. The Kaplan-Meier curves of the LowHR, MediumHR, and HighHR groups. These results confirmed those illustrated in panel A † $p < 0.05$, ✽ $p < 0.01$ by log rank test.

Table 2 Odds Ratio of group MediumHR and HighHR to LowHR

Groups	OR of mortality in ICU			OR of mortality in hospital		
	OR	95%CI	*p* value	OR	95%CI	*p* value
LowHR	1	–	–	1	–	–
MediumHR	1.5	0.1–37.2	0.79	4.5	0.6–91.7	0.16
HighHR	7.3	1.2–138.0	＜0.05†	13.7	2.5–256.6	＜0.01✽

（†：＜0.05, ✽：＜0.01）

Hayashi M Acta Med Oca 2019;73:147-153

Conclusion der perioperativen Hypotension

Schweregrad und die Dauer einer perioperativen Hypotension sind unabhängige Risikofaktoren für:

- Acute kidney injury
- Myocardial damage
- Postoperative mortality

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Definition of low cardiac output

There is no consensus on the absolute definition
ESC: Cardiac index <2.4L/min/m2 with evidence of organ dysfunction
There is a continuum from a low-cardiac-output state to cardiogenic shock

"evidence of tissue hypoperfusion induced by cardiac dysfunction after correction of preload." ESC homepage

- Systolic blood pressure of <90 mmHg (or a drop of >30 mmHg)
- Urine output of <0.5 ml/kg/hour
- Heart rate >60 beats per minute
- With or without evidence of congestion

The European Society of Cardiology (ESC) homepage October 2023

Woher kommen die Werte des Low Cardiac Output?

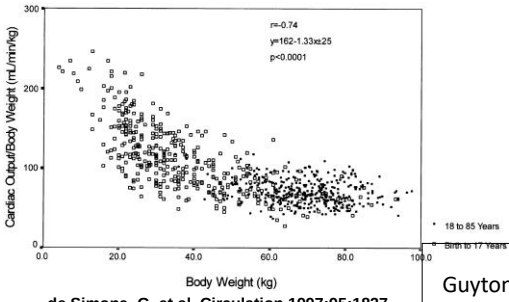
MEDICAL PROGRESS
Medical Therapy of Acute Myocardial Infarction by Application of Hemodynamic Subsets
James S. Forrester, M.D., George Diamond, M.D., Kanu Chatterjee, M.B., M.R.C.P., and H. J. C. Swan, M.D., Ph.D.
N Engl J Med 1976; 295:1404-1413

Patienten mit akutem Myokardinfarkt unter einem CI < 2.2 L/min/m2 signifikant höhere Mortalität

American Heart Association®
life is why™

Forrester JS. NEJM 1976;295:1356-1362

Fluss – was ist „normal“ - Woher kommen die Daten?



de Simone, G. et al. Circulation 1997;95:1837-1843

CI = 2.7 mL/min/m2 (median)

1.9 – 3.5 range!

Guyton 1996:

Median CI = 3 im Alter 40

Median CI = 2,4 im Alter 80

Guyton AC: Cardiac output, venous return, and their regulation, in Guyton AC: Textbook of Medical Physiology, Saunders 1996, p 241.

Heart Failure and Mortality in Non-Cardiac Surgery

Epidemiology and Prevention

Mortality and Readmission of Patients With Heart Failure, Atrial Fibrillation, or Coronary Artery Disease Undergoing Noncardiac Surgery

An Analysis of 38 047 Patients

Sean van Diepen, MD; Jeffrey A. Bakal, PhD; Finlay A. McAlister, MD, MSc; Justin A. Ezekowitz, MBBCh, MSc

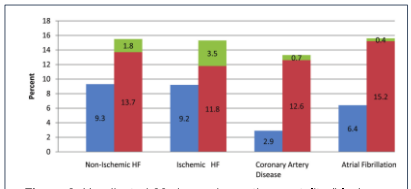


Figure 3. Unadjusted 30-day perioperative mortality (blue), rehospitalization (red), and cardiac rehospitalization (green). HF indicates heart failure.

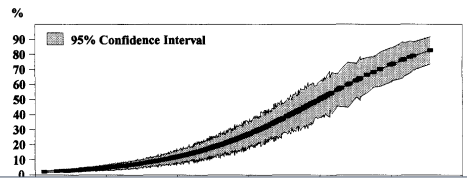
Van Diepen. Circulation 2011; 124:289-296

Table 2. Unadjusted 30-Day Perioperative Mortality Stratified by Cohort, Procedure Type, and Admission Urgency

	HF Cohorts			
Surgical Type and Admission Urgency	Nonischemic HF, n (%)	Ischemic HF, n (%)	CAD Cohort, n (%)	AF Cohort, n (%)
Abdominal	61 (12,5)	77 (11,2)	45 (5,4)	42 (9,2)
Emergent/urgent	59 (16,8)	65 (13,3)	39 (9,2)	36 (11,3)
Elective/outpatient	2 (1,5)	12 (6)	6 (1,5)	6 (4,3)
Orthopedic	127 (13,2)	215 (15,4)	63 (7,6)	50 (8,3)
Emergent/urgent	117 (14,5)	201 (18,0)	58 (12,2)	48 (10,2)
Elective/outpatient	10 (6,5)	14 (5,1)	5 (1,4)	2 (1,5)
Pelvic	4 (7,4)	2 (3,7)	2 (1,8)	5 (9,6)
Emergent/urgent	4 (16)	2 (10)	1 (4,8)	3 (9,5)
Elective/outpatient	0 (0)	0 (0)	1 (1,1)	2 (9,7)
Thoracic	0 (0)	0 (0)	1 (11,1)	0 (0)
Emergent/urgent	0 (0)	0 (0)	1 (14,9)	0 (0)
Elective/outpatient	0 (0)	0 (0)	0 (0)	0 (0)
Vascular	10 (4,9)	47 (10,9)	20 (5,8)	2 (2,5)
Emergent/urgent	9 (11,7)	31 (17,1)	11 (7,7)	2 (4,7)
Elective/outpatient	1 (0,8)	16 (6,4)	9 (4,6)	0 (0)
Minor	511 (8,5)	782 (8,1)	266 (2,3)	177 (5,7)
Emergent/urgent	351 (15,4)	477 (13,9)	180 (9,6)	121 (14,7)
Elective/outpatient	160 (4,3)	305 (4,9)	86 (0,9)	56 (2,4)
All major				
Emergent/urgent	189 (15)	299 (16,5)	110 (10,3)	88 (10,2)
Elective/outpatient	13 (2,9)	42 (5,5)	21 (2)	11 (3,2)
All				
Emergent/urgent	540 (15,3)	776 (14,8)	290 (9,9)	209 (12,4)
Elective/outpatient	173 (4,2)	347 (5,0)	107 (1,0)	67 (2,6)

Low Cardiac Output Syndrome and Mortality in Cardiac Surgery

The Journal of Thoracic and Cardiovascular Surgery
Volume 112, Number 1



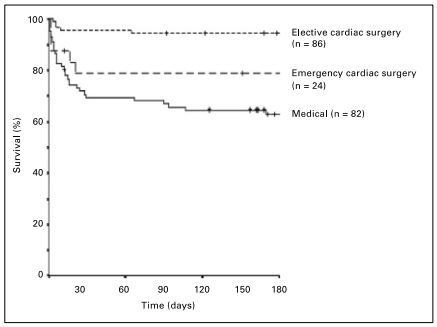
Mortality Herzchirurgischen Patienten mit LCOS 16,9% vs. 0,9% !!!

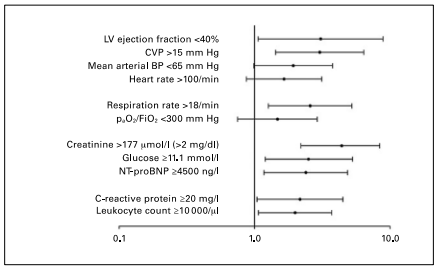
Rao V et al. J Thorac Cardiovasc Surg 1996; 12: 38-51
Rudiger A et al. Swiss Med Wkly. 2009; 139: 110-116

Acute Heart Failure and Outcome in Critically Ill

Presentation and outcome of critically ill medical and cardiac-surgery patients with acute heart failure

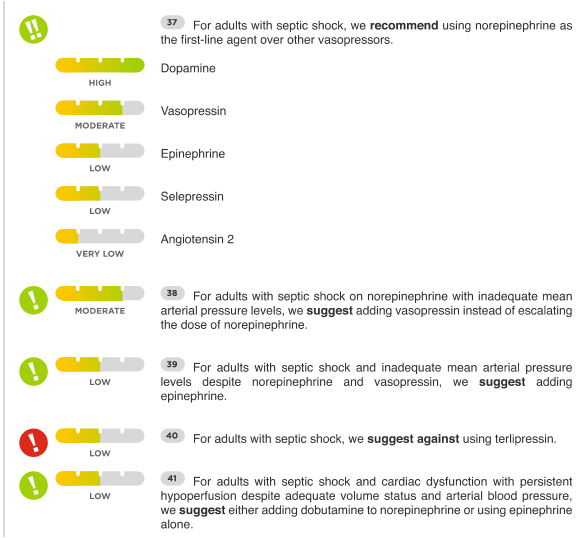
Rudiger, A; Businger, F; Streit, M; Schmid, ER; Maggiorini, M; Follath, F





Rudiger A. Swiss Medical Weekly 2009;139:110-116

Surviving Sepsis Campaign



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„Werte“ therapieren

Kein Hinweis für Organdysfunktion

keineTherapie

-Wach und orientiert

-Warme Extremitäten

-Adäquate Urinproduktion

-Akzeptable gemischt/zentralvenöse Sättigung

-Keine metabolische Azidose oder erhöhte Laktat-Werte

ration, and no metabolic acidosis or elevated serum lactate). Usually the preoperative or pre cardiopulmonary bypass cardiac index is similarly low. Postoperatively, attempts to achieve values >2.2 L/min/m^2 result in extra days in the ICU, after which the PA catheter is removed and the patient leaves the ICU with the same low cardiac index.

Before clinicians are drawn into monitoring cardiac output in larger numbers of patients, especially those who appear to be

Ramsay J. J Cardiothoracic and Vasc Anesthesia 2002; 16: 1-3

Outcome and Positive Inotropic Agents

Positive Inotropic Agents in Myocardial Ischemia–Reperfusion Injury

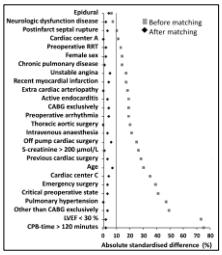
A Benefit/Risk Analysis

Jean-Luc Fellahi, M.D., Ph.D.,* Marc-Olivier Fischer, M.D.,† Georges Daccache, M.D.,† Jean-Louis Gerard, M.D.,* Jean-Luc Hanouz, M.D., Ph.D.‡

from deleterious influences on myocyte oxygen consumption which can lead to cardiac arrhythmias and myocyte death.[1] Besides, their benefits on medium- and long-term survival, especially in the setting of myocardial ischemia and reperfusion (acute coronary syndrome, revascularization by coronary angioplasty, perioperative acute myocardial infarction, and cardiac surgery with or without cardiopulmonary bypass), have never been validated.[2] Finally, there is a great

Fellahi JL. Anesthesiology 2013;118:1460-5

Catecholamines and Outcome



Health Outcomes with and without Use of Inotropic Therapy in Cardiac Surgery

Results of a Propensity Score–matched Analysis

Dorthe Viemose Nielsen, M.D., Malene Kærslund Hansen, M.B.B.S., Søren Paaske Johnsen, M.D., Ph.D., Mads Hansen, M.D., Karsten Hindsholm, M.D., H.D., Carl-Johan Jakobsen, M.D.

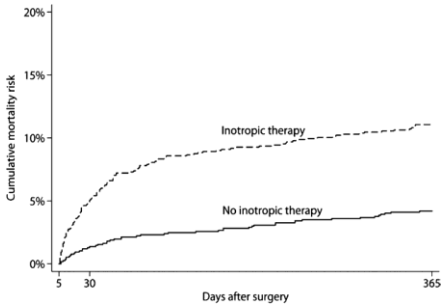


Fig. 3. Cumulative 1-yr mortality risk by treatment status. Log-rank *P* value <0.00001.

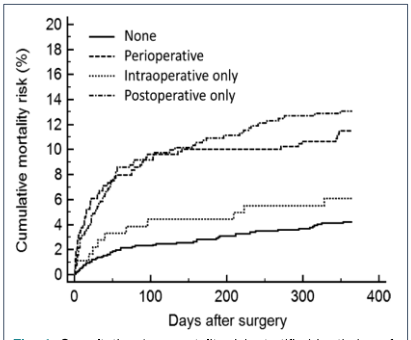


Fig. 4. Cumultative 1-yr mortality risk stratified by timing of inotropic therapy. Log-rank *P* value <0.0001.

Nielsen DV et al. Anesthesiology 2014; 120: 1098-1108.

Einfluss des Flusses auf das Outcome

employed. Recommended perfusion flows range from 30 to 60 mL · kg^{-1} · min^{-1} (1.2 to 2.4 L · m^{-2} · min^{-1}) and perfusion pressure ranges vary from 30 to 50 mm Hg to 60 to 100 mm Hg [1, 2]. Some outcome studies implicate hypotension during CPB as a cause of postoperative neurological (CNS) [3–6] and renal dysfunction [7–9], whereas others clearly fail to support these relationships [1, 10–15]. These reports, however, uniformly ignore the contribution of induced hypothermia to reduced tissue oxygen requirement and the contribution of hemodilution to the uncoupling of flow and pressure [16] in defining the role of hypotension or hypoperfusion in adverse outcome of CPB. Even though systematic review of these studies

when major organ dysfunction follows a cardiac operation. We therefore undertook a prospective investigation directed primarily to the roles of perfusion pressure and flow with hemodilution and moderate hypothermia in the genesis of new postoperative renal and clinically apparent CNS dysfunction in unselected adult patients undergoing operations requiring CPB. These data were then analyzed against the background of patient characteristics and other perioperative events that might also account for these adverse outcomes.

Material and Methods

Prospective observational study
504 patients
Ziel: Flow 40 mL/kg/min
Druck > 50 mmHg
Endpunkte: adverse renal and neurologic outcome
Ziel nicht erreicht Fluss: 21,6% und im Druck 97,1%

kidneys after an unperfused heart protected by cardioplegia and hypothermia has been surgically treated. To achieve this outcome institutional guidelines for optimal

Was passiert, wenn ich bei konstantem Fluss den Druck mit Vasopressoren anhebe?

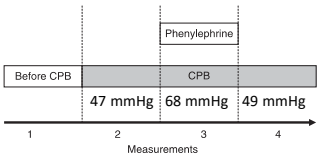
B.J.A. Advance Access published February 25, 2009

British Journal of Anaesthesia Page 1 of 7
doi:10.1093/bja/aep018

BJA

Effects of phenylephrine on the sublingual microcirculation during cardiopulmonary bypass

S. Maier[1], W. R. Hasibeder[1], C. Hengl[1], W. Pajk[1], B. Schwarz[1], J. Margreiter[1], H. Ulmer[2], J. Engl[3] and H. Knotzer[1]*



Ergebnisse

Table 2 Systemic and microvascular variables. Variables were recorded in each patient after initiation of general anaesthesia, during CPB (systemic blood flow=2.4 litre m^{-2}), after increasing perfusion pressure by 20 mm Hg due to continuous infusion of phenylephrine, and after termination of phenylephrine infusion. CPB, cardiopulmonary bypass; Hb, haemoglobin; Hct, haematocrit; pHa, arterial pH; HCO_3^-, arterial bicarbonate; arterial BE, base excess; Pa_{O_2}, arterial oxygen tension; Pa_{CO_2}, arterial carbon dioxide tension; Sv_{O_2}, mixed central venous oxygen saturation; DO_2I, systemic oxygen delivery index; VO_2I, systemic oxygen uptake index; PU, microvascular perfusion units; Smc_{O_2}, microcirculatory haemoglobin oxygen saturation; MFI_s, small vessel microvascular flow index; MFI_m, medium vessel microvascular flow index. Data are given as mean (SD). MFI_s and MFI_m are given as median and inter-quartile range in brackets. *Significant ($P<0.0125$) compared between *before CPB* and *CPB (before phenylephrine)*. †Significant ($P<0.0125$) compared between *before phenylephrine* and *phenylephrine*. ‡Significant ($P<0.0125$) compared between *after phenylephrine* and *phenylephrine*

	Before CPB	CPB			*P*-value
		Before phenylephrine	Phenylephrine	After phenylephrine	
Systemic variables					
Perfusion pressure (mm Hg)	72.5 (10.8)	47.0 (8.8)*	68.1 (7.0)†,‡	48.7 (6.0)	<0.001
Syst. flow index (litre m^{-2})	2.6 (0.4)	2.4 (0.0)*	2.4 (0.0)	2.4 (0.0)	<0.001
Temperature (°C)	35.8 (0.4)	33.2 (1.2)*	32.3 (1.4)	32.8 (1.3)	<0.001
Hb (g dl^{-1})	12.1 (1.3)	7.9 (1.1)*	8.2 (0.8)	8.2 (0.7)	<0.001
Hct (%)	35.5 (3.7)	23.3 (3.1)*	24.0 (2.5)	24.3 (2.1)	<0.001
pHa	7.41 (0.04)	7.35 (0.04)*	7.36 (0.04)	7.35 (0.05)	<0.001
HCO_3^- (mmol $litre^{-1}$)	24.6 (2.2)	22.7 (2.2)	23.2 (2.2)	23.0 (2.1)	<0.001
BE	0.0 (2.2)	−2.7 (2.5)	−2.2 (2.6)	−2.6 (2.7)	<0.001
Pa_{O_2} (kPa)	33.3 (16.0)	36.3 (6.0)	30.3 (3.5)	30.6 (5.7)	0.243
Pa_{CO_2} (kPa)	5.2 (0.8)	5.6 (0.5)	5.5 (0.4)	5.5 (0.5)	0.196
Sv_{O_2} (%)	84.2 (2.4)	78.9 (3.8)*	79.3 (4.9)	79.3 (5.1)	0.002
DO_2I (ml m^{-2} min^{-1})	457 (97)	279 (33)*	282 (26)	283 (22)	<0.001
VO_2I (ml m^{-2} min^{-1})	98 (52)	71 (12)	68 (12)	65 (15)	0.006
Microvascular variables					
PU (arbitrary units)	120 (105)	110 (54)	197 (100)†,‡	89 (66)	0.007
Smc_{O_2} (%)	73 (7)	72 (11)	84 (7)†,‡	72 (8)	<0.001
MFI_s (arbitrary units)	2.1 (1.2)	2.5 (2)	1.8 (1.2)†	2.2 (1.2)	0.039
MFI_m (arbitrary units)	3 (2)	3 (2)	2.8 (1)	2.9 (2)	0.281

Intravitalmikroskop

Ohne Phenylephrine | Mit Phenylephrine | Ohne Phenylephrine

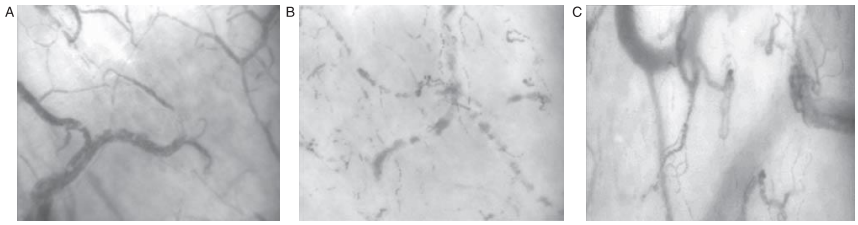
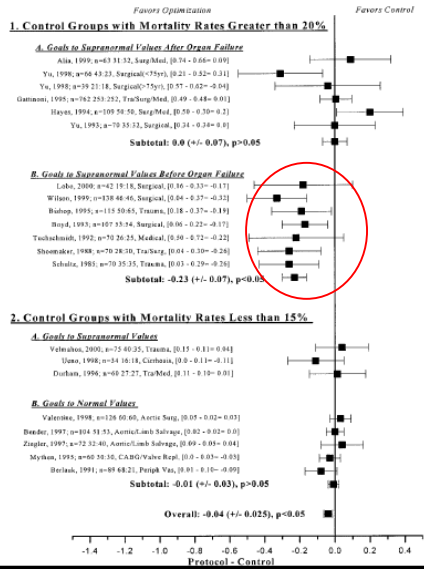


Fig 1 Visualization of the microcirculation in the sublingual mucosal area recorded with the SDF imaging technique. (A) After initiation of CPB without haemodynamic intervention; (B) during pressure increase with phenylephrine; and (C) after discontinuation of phenylephrine. Note the diminished functional capillary density in a patient on CPB with pressure increase with phenylephrine.

Klinische Folgerung: Wenn ich den Druck erhöhen möchte, dann über den Fluss bei gleichem Widerstand

Endpoints of resuscitation normal or supranormal? Ausflug in die Intensivmedizin



Supranormal:

CI > 4.5 L/min/m2

DO2I > 600 ml/min/m2

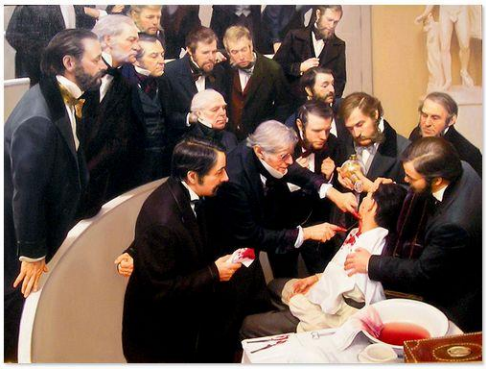
VO2I > 170 ml/min/m2

Kern JW, Shoemaker WC. Crit Care Med 2002; 30:1686-1692

Fluss: Kapitän

Druck: 1. Offizier

Discussion



'Ether Day' von W.Prosperi, 1846

"Gentlemen, this is no Humbug"

John C. Warren, 1846

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