14.20 Compenso dell'anemia: sistemi di riconoscimento della tolleranza individuale

A. De Gasperi

Anesthesiology 2009; 110:574–81

Risk Associated with Preoperative Anemia in Noncardiac Surgery

A Single-center Cohort Study

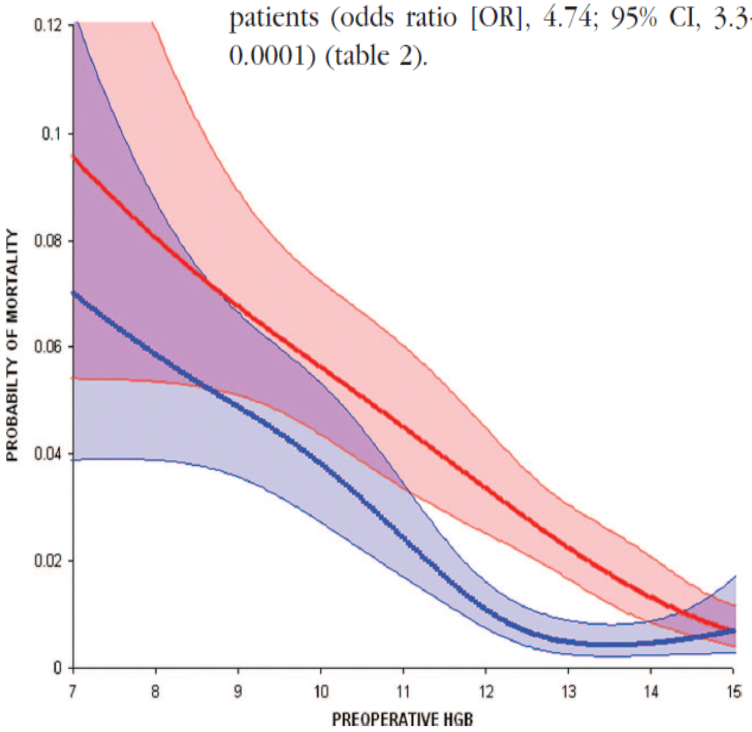
W. Scott Beattie, M.D., Ph.D., F.R.C.P.C., Keyvan Karkouti, M.D., M.Sc., F.R.C.P.C.,† Duminda N. Wijeysundera, M.D., F.R.C.P.C.,‡ Gordon Tait, Ph.D.§*

Results: Preoperative anemia was common and equal between genders (39.5% for men and 39.9% for women) and was associated with a nearly five-fold increase in the odds of postoperative mortality. After adjustment for major confounders using logistic regression, anemia was still associated with increased mortality (odds ratio, 2.36; 95% confidence interval 1.57–3.41). This relationship was unchanged after elimination of patients with severe anemia and patients who received transfusions. In a propensity-matched cohort of patients, anemia was associated with increased mortality (odds ratio, 2.29; 95% confidence interval, 1.45–3.63).

Conclusions: Anemia is a common condition in surgical patients and is independently associated with increased mortality. Although anemia increases mortality independent of transfusion, it is associated with increased requirement for transfusion, which is also associated with increased mortality. Treatment of preoperative anemia should be the focus of investigations for the reduction of perioperative risk.

preoperative hemoglobin concentration and postoperative mortality in men and women, respectively. Of note, the slopes for men and women are virtually identical, and the threshold for increased mortality falls within the 95% confidence interval (CI) for the World Health Organization definition of anemia.

The unadjusted relationships with preoperative anemia and important perioperative variables and measured outcomes are seen table 1. The unadjusted odds of a perioperative death is higher in anemic than nonanemic patients (odds ratio [OR], 4.74; 95% CI, 3.3–6.7; $P <$ 0.0001) (table 2).



anemo 2011

Anesthesiology 2009; 110:574–81

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The overriding clinical implications of this study's findings are that if the observed association between preoperative anemia and mortality in noncardiac surgery is causal, correcting the anemia will likely improve outcomes. We, however, advocate a cautious approach to correcting preoperative anemia. There are currently at

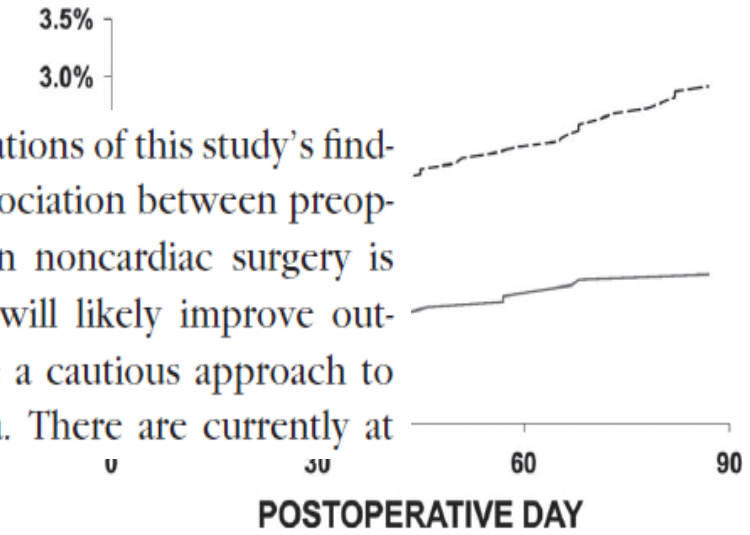


Fig. 2. The risk adjusted effect of anemia on postoperative mortality. This figure represents the time to event comparing anemic to nonanemic patients in the propensity-matched cohorts. *x axis* = postoperative day; *y axis* = percent mortality; *broken line* = patients with preoperative anemia; *solid line* = nonanemic patients.

Anesthesiology 2011; 114: 283-92

Association between Intraoperative Blood Transfusion and Mortality and Morbidity in Patients Undergoing Noncardiac Surgery

Laurent G. Glance, M.D.,* Andrew W. Dick, Ph.D.,† Dana B. Mukamel, Ph.D.,‡

Methods: This was a retrospecti
blood transfusion and 30-day m
in 10,100 patients undergoing g
surgery. We estimated separate
models for 30-day mortality and

transfusion was associated
odds ratio [OR], 1.29; 95%
g an intraoperative transfu-
lmonary, septic, wound, or
compared with patients not
fusion. Compared with pa-

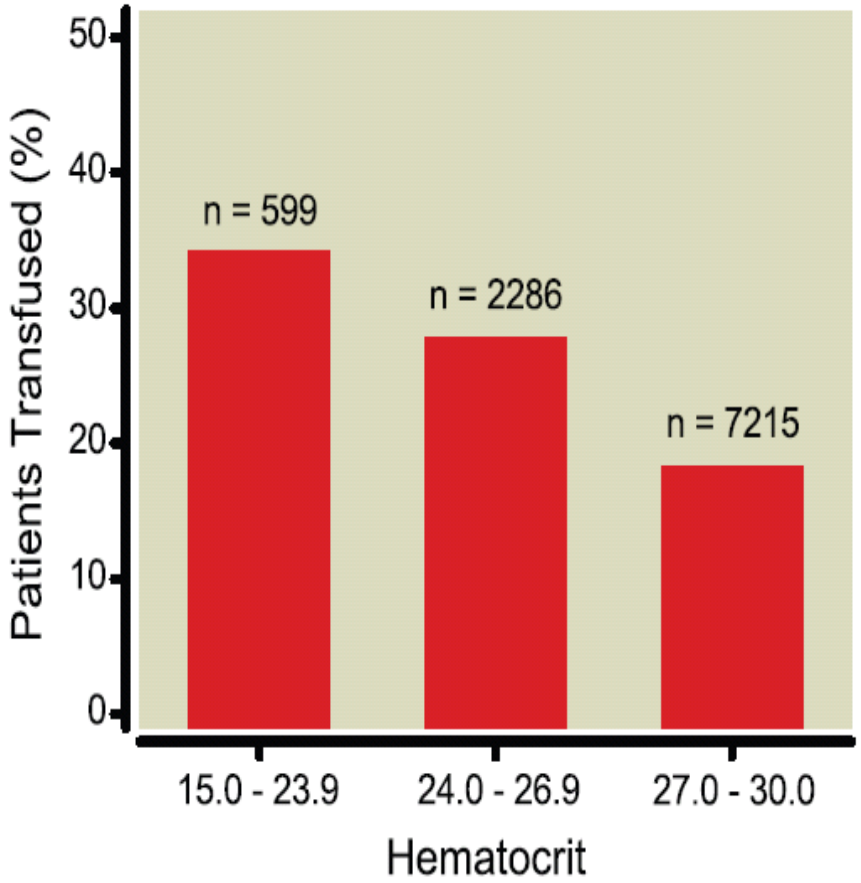


Fig. 1. Proportion of patients receiving one or two units of erythrocytes intraoperatively *versus* baseline hematocrit.

Table 1. Patient Demograp

Patient Risk Factors	*P* Value
Baseline hematocrit	< 0.001
Age, yr	< 0.001
Male	< 0.001

Anesthesiology 2011; 114: 283-92

In this study, we found that blood transfusion in the setting of noncardiac surgery is associated with increased risk of 30-day mortality and pulmonary, septic, wound, and thromboembolic complications. The increased risk of mortality and morbidity associated with blood transfusion was present after adjusting for patient demographics, functional status, comorbidities, and surgical complexity. Blood transfusion did not appear to be protective in patients with cardiovascular disease.

Table 3. Impact of Intraoperative Transfusion on 30-Day Mortality and 30-Day Complications

Outcome	Transfusion Group, Outcome Rate (%)	No Transfusion Group, Outcome Rate (%)	Unadj OR Txf *vs.* No Txf (95% CI)	Adj OR Txf *vs.* No Txf (95% CI)	Adj OR Txf *vs.* No Txf (PS Method) (95% CI)
Mortality	6.44	4.26	1.55 (1.24, 1.90)	1.29 (1.03, 1.62)	1.21 (0.96, 1.52)
Cardiac complications	2.08	1.40	1.50 (1.06, 2.12)	1.40 (0.97, 2.03)	1.31 (0.88, 1.95)
Pulmonary complications	12.6	6.03	2.24 (1.92, 2.63)	1.76 (1.48, 2.09)	1.75 (1.47, 2.08)
Renal complications	2.69	1.85	1.46 (1.08, 1.99)	1.32 (0.93, 1.88)	1.29 (0.91, 1.84)
CNS complications	0.69	0.58	1.20 (0.67, 2.15)	0.84 (0.43, 1.64)	0.68 (0.34, 1.38)
Sepsis complications	16.4	9.81	1.81 (1.58, 2.07)	1.43 (1.21, 1.68)	1.46 (1.24, 1.72)
Wound complications	9.17	4.65	2.07 (1.73, 2.48)	1.87 (1.47, 2.37)	1.89 (1.49, 2.41)
Thromboembolic complicatioins	4.07	1.89	2.20 (1.69, 2.88)	1.77 (1.32, 2.38)	1.81 (1.34, 2.45)

Transfusion Medicine 1 Lancet 2007; 370: 415–26

Red blood cell transfusion in clinical practice

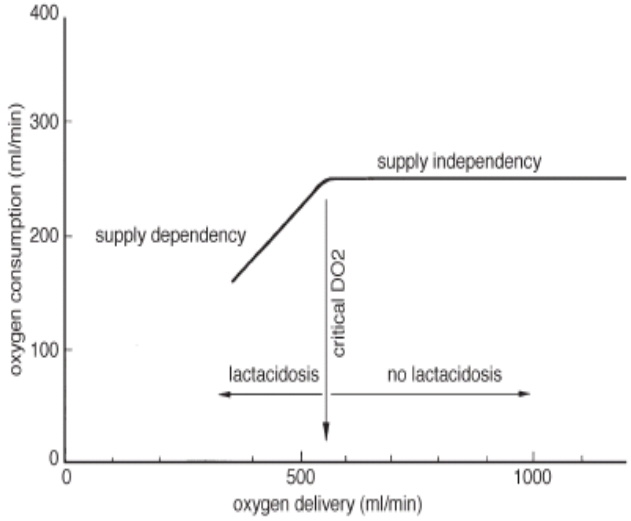
Harvey G Klein, Donat R Spahn, Jeffrey L Carson

Principles of oxygen transport

Acute loss of about 20% of blood volume elicits compensatory increases in heart rate and cardiac output, as well as a rise in vasoactive hormones, redistribution of blood flow, and influx of extravascular fluid to the intravascular compartment.[36–39] Acute blood loss is managed initially by restoring volume to avoid haemorrhagic shock. Infusions and fluid shifts result in an abrupt decrease in haemoglobin. As haemoglobin falls, compensatory mechanisms reach their limits in the different organ systems. These mechanisms are also less effective in people who are ill or elderly.

A decrease in the haemoglobin concentration does not necessarily result in reduced DO_2 because cardiac output usually increases. A second global compensatory mechanism involves increasing oxygen extraction, which lowers venous oxygen saturation and partial pressure.

DO2, VO2 and O2ER



Global oxygen consumption (VO_2) which describes the amount of oxygen consumed by the whole body per minute ranges under physiological conditions in a normal adult from 200 to 300 ml/min whereas DO_2 ranges from 800 to 1200 ml/min. The relationship VO_2/DO_2 defines the oxygen extraction ratio (O_2ER) which is thus in the range of 20 to 30%. A normal VO_2/DO_2-relationship is illustrated in Figure 1. It

In analogy to DO_2, the quantity of oxygen consumed by the whole body (VO_2) is calculated as follows:

$$VO_2 = CO \times (CaO_2 - CvO_2)$$

where CvO_2 is the oxygen content of venous blood after venous admixture of all organs. The normal VO_2 for a conscious resting subject breathing room air is $\sim 250\,mL\,O_2\,min^{-1}$, thus only 25% of the delivered oxygen are actually consumed. The unextracted oxygen forms the reserve which may be used under stress conditions (exercise, anaemia). It

Red blood cell transfusion in clinical practice

Lancet 2007; 370: 415–26

Harvey G Klein, Donat R Spahn, Jeffrey L Carson

critical haemoglobin across species appears to be remarkably constant, at approximately 20–25% of normal resting haemoglobin[10].

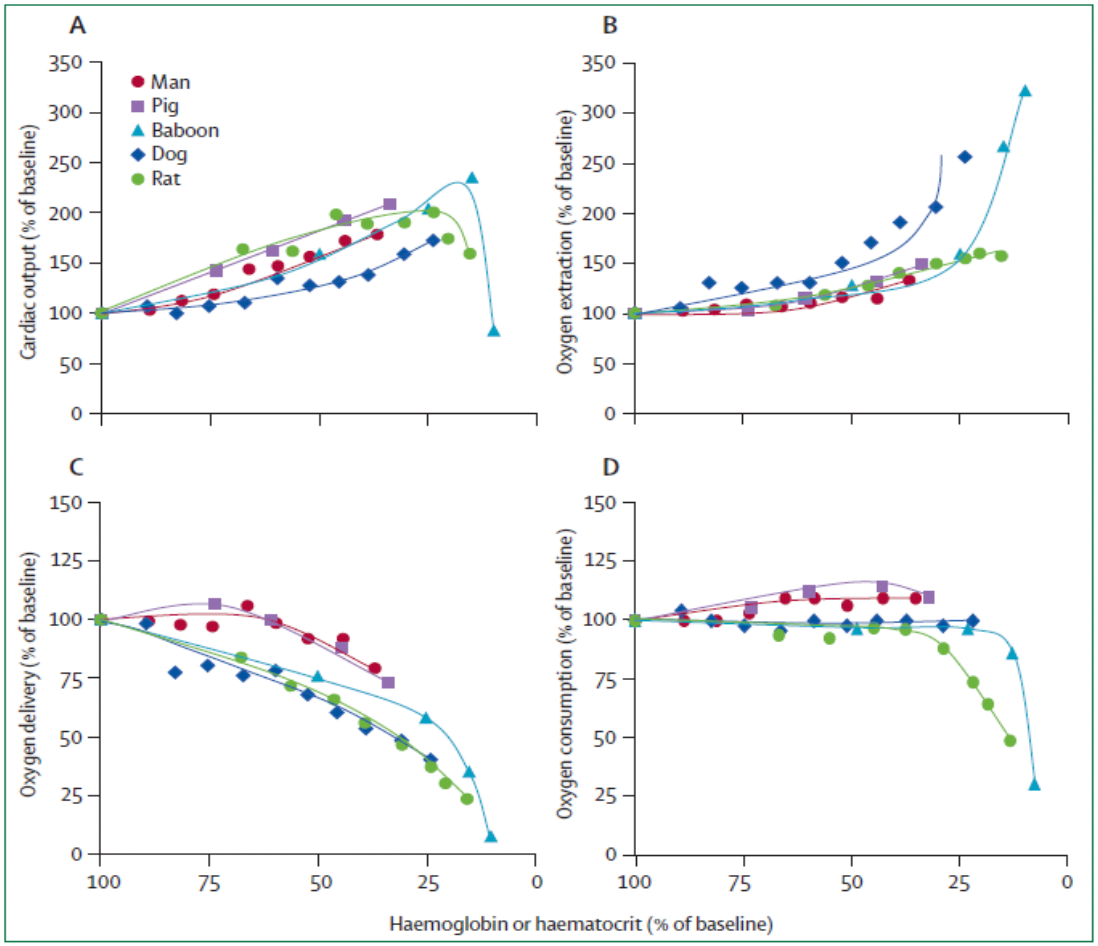


Figure 2: **Relative changes in cardiac output (A) oxygen extraction (B), oxygen delivery (C), and oxygen consumption (D) as haemoglobin concentration decreases in humans, pigs, baboons, dogs, and rats**
The combined increases in cardiac output and oxygen extraction allow maintainance of oxygen consumption until low haemoglobin levels. At extremely low haemoglobin levels, cardiac output and oxygen consumption can fall, indicating the exhaustion of the compensatory mechanisms. Data are from the original articles,[41–45] the curves were approximated.

ANEMO '11

Time course and etiology of death in patients with severe anemia

Aaron A.R. Tobian, Paul M. Ness, Helaine Noveck, and Jeffrey L. Carson

Mortality increases as hemoglobin (Hb) levels fall. A series of case reports on Jehovah's Witness patients demonstrated that survival is not significantly altered among individuals with a Hb above 5 g/dL.[1] Two studies involving cohorts of Jehovah's Witness patients undergoing surgery demonstrated that low preoperative Hb concentration or substantial operative blood loss increases mortality.[2,3] The odds of death increase 2.5 times for each gram decrement in postoperative Hb level with mortality becoming extremely high among individuals with a Hb below 5 g/dL.[4] Both animal and human studies have demonstrated that Hb concentrations below 3 g/dL are maximally life-threatening.[5,6]

TRANSFUSION 2009;49:1395-1399.

anemo 2011

Management of Adult Jehovah's Witness Patients with Acute Bleeding

The American Journal of Medicine (2009) 122, 1071-1076

Kenrick Berend. MD. PhD.[a] Marcel Levi. MD. PhD[b]

ues. In otherwise healthy individuals, the terminal hemoglobin is not precisely known, but appears to be below 5 g/dL, and might even be as low as 3 g/dL.[6,7] Survival has been reported in a few cases of Jehovah's Witnesses undergoing operative procedures with hemoglobins ranging from 2.2 to 3.0 g/dL,[8] and even postoperative hemoglobin as low as 1.4 g/dL has been successfully[9] Therefore, a key to successful management of severe bleeding episodes in these patients is to win time for recovery of the hemoglobin

9. Ng KO, Chow LH, Wang CC, et al. Successful management of massive blood loss to extremely low hemoglobin in an elderly woman receiving spinal surgery. *Acta Anaesthesiol Sin.* 2000;38:89-92.

Anesth Analg 1992;75:818–21

Profound Hemodilution: What Is the Critical Level of Hemodilution at Which Oxygen Delivery-Dependent Oxygen Consumption Starts in an Anesthetized Human?

E. C. S. M. van Woerkens, MD, A. Trouwborst, MD, PhD, and J. J. B. van Lanschot, MD, PhD

In humans, the first report of such measures was documented in an 84-year-old Jehovah's Witness who refused transfusion and died postoperatively at a haemoglobin concentration of 1·6 g/dl; DO_2crit in this patient under anaesthesia was 4·9 ml O_2/kg·min for a VO_2 of 2·4 ml O_2/kg·min, and occurred at haemoglobin of 4·0 g/dl. The oxyhaemoglobin dissociation

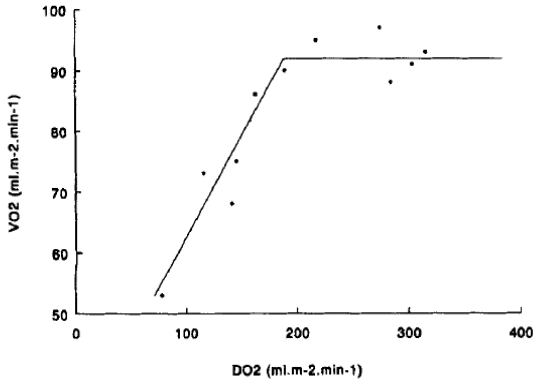


Figure 1. Relationship between oxygen delivery (Do_2) and oxygen consumption (Vo_2) during increasing hemodilution.

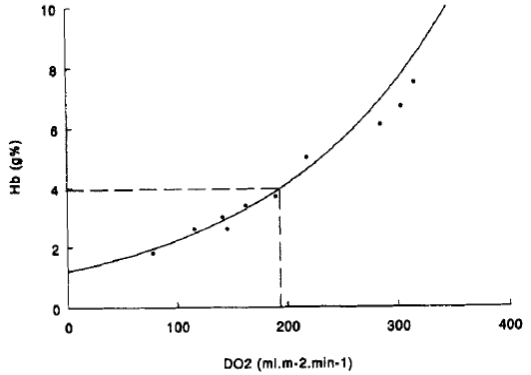


Figure 2. Relationship between oxygen delivery (Do_2) and hemoglobin (Hb) concentration with critical point of Do_2.

Anesth Analg 1992;75:818–21

Profound Hemodilution: What Is the Critical Level of Hemodilution at Which Oxygen Delivery-Dependent Oxygen Consumption Starts in an Anesthetized Human?

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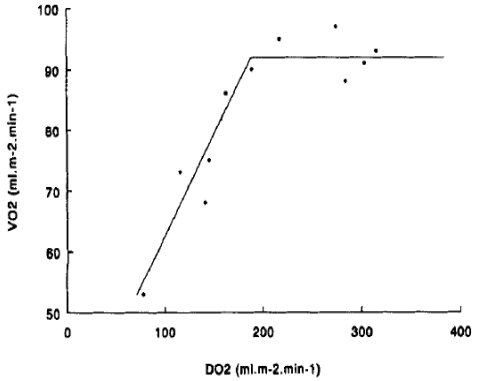

Figure 1. Relationship between oxygen delivery (Do_2) and oxygen

Table 2. Systemic Oxygenation Before and After Induction of Anesthesia

	Pao_2 (mm Hg)	$Paco_2$ (mm Hg)	Pvo_2 (mm Hg)	$Pvco_2$ (mm Hg)	pH art	pH ven	Do_2 ($L \cdot m^{-2} \cdot min^{-1}$)	Vo^2 ($L \cdot m^{-2} \cdot min^{-1}$)	ER (%)	P_{50} act (mm Hg)	CO (L/min)	SVR ($dyne \cdot s \cdot cm^{-5}$)	Hct (%)	Hb (g/dL)
Preinduction	75	37	31	40	7.46	7.44	339	126	37	26.2	4.4	1818	31	10.1
Postinduction	250	36	37	39	7.45	7.43	275	97	35	27.9	3.5	1440	30	9.6
H1	220	35	42	39	7.45	7.42	316	93	30	28.1	5.0	1040	24	7.5
H2	225	36	41	38	7.44	7.41	304	91	30	27.9	5.2	969	21	6.7
H3	215	36	42	38	7.43	7.40	285	88	31	28.3	5.4	844	20	6.1
1500-mL blood loss	92	34	33	36	7.40	7.38	218	95	44	28.6	5.3	1072	16	5.0
3500-mL blood loss	170	36	30	39	7.37	7.34	163	86	53	29.3	5.5	887	10	3.4
ES	226	37	32	39	7.37	7.33	146	75	51	28.7	5.8	648	9	2.6
1 h postop	230	44	33	46	7.37	7.34	190	90	48	28.7	5.7	1179	12	2.7
2 h postop	262	34	33	40	7.46	7.38	142	68	48	27.5	5.0	1120	12	3.0
4 h postop	293	30	26	38	7.48	7.40	116	73	63	27.5	4.5	800	9	2.6
8 h postop	345	30	31	38	7.39	7.26	78	53	68	34.6	3.8	653	8	1.8

Art, arterial; ven, venous; Do_2, oxygen delivery; Vo_2, oxygen consumption; ER, oxygen extraction ratio; P_{50} act, Po_2 at oxyhemoglobin saturation of 50% measured in mixed venous blood; P_{50} c, P_{50} of mixed venous blood corrected at pH = 7.40 and Pco_2 = 40 mm Hg; H1, H2, H3, data for each step of hypervolemic hemodilution; ES, end of surgery; postop, postoperatively.

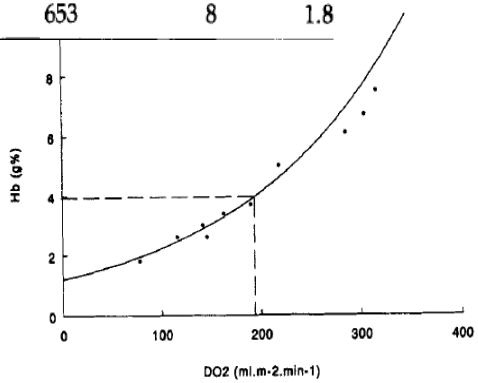

Figure 2. Relationship between oxygen delivery (Do_2) and hemoglobin (Hb) concentration with critical point of Do_2.

Transfusion Medicine 1 Lancet 2007; 370: 415–26

Red blood cell transfusion in clinical practice

Harvey G Klein, Donat R Spahn, Jeffrey L Carson

Moderate isovolemic haemodilution is well-tolerated in elderly patients (aged 65–88 years) with no known cardiac disease.[51] Elderly patients can tolerate a reduction in haemoglobin to 9 g/dL, and maintain VO_2 by increasing cardiac output and oxygen extraction as effectively as younger people. Autologous blood was re-transfused at a median haemoglobin of 7·7 g/dL and at a haemoglobin <7 g/dL in nine of 20 patients. No signs of circulatory instability or myocardial ischemia were

Spahn DR, Zollinger A, Schlumpf RB, et al. Hemodilution tolerance in elderly patients without known cardiac disease. *Anesth Analg* 1996; 82: 681–86.

Hemodilution Tolerance in Elderly Patients Without Known Cardiac Disease

Donat R. Spahn, MD*, Andreas Zollinger, MD*, Rolf B.

During hemodilution 769 ± 34 mL blood was removed and replaced with 780 ± 39 mL of 6% hydroxyethyl starch. Hemoglobin decreased during hemodilution from 11.6 ± 0.4 g/dL to 8.8 ± 0.3 g/dL (Table 1). With constant filling pressures (CVP, PCWP) (Table 1), CI increased during hemodilution (Fig. 1A). The

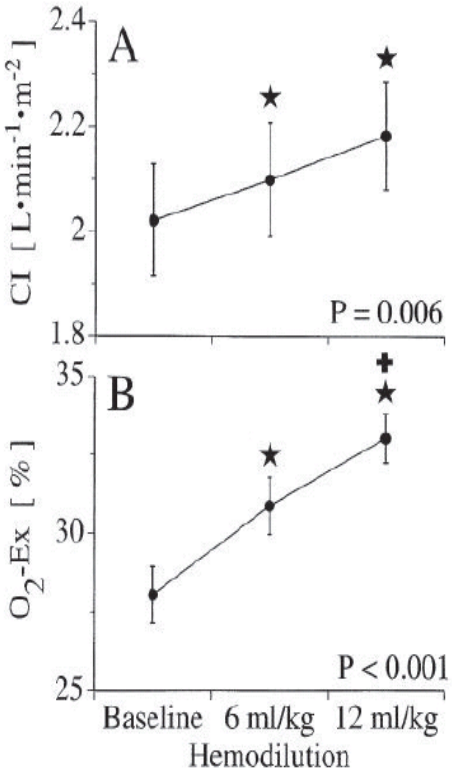

Figure 1. Alterations of cardiac index (CI) (A) and O_2 extraction (O_2-Ex) (B) during hemodilution. *P* values indicate significance level of change during hemodilution (repeated-measures analysis of variance); ★ significantly different ($P < 0.05$) versus baseline; +significantly different ($P < 0.05$) versus 6 mL/kg hemodilution.

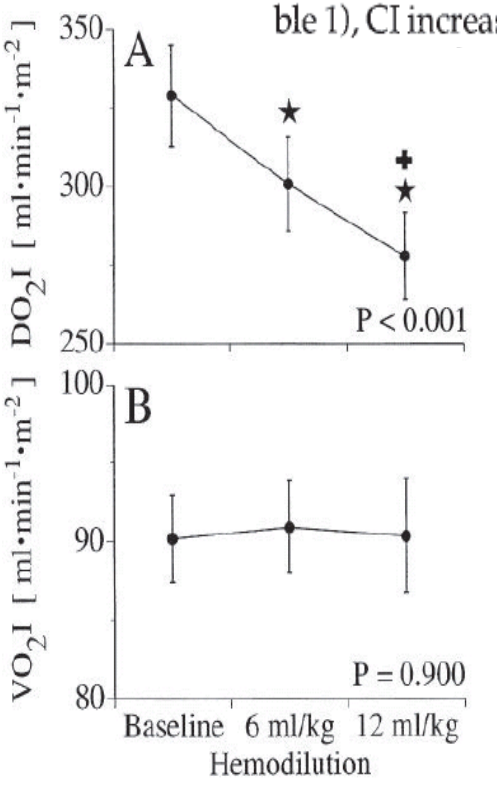

Figure 2. Alterations of O_2 delivery (DO_2I) (A) and O_2 consumption (VO_2I) (B) during hemodilution. *P* values indicate significance level of change during hemodilution (repeated-measures analysis of variance); ★ significantly different ($P < 0.05$) versus baseline; +significantly different ($P < 0.05$) versus 6 mL/kg hemodilution.

Effect of anaemia and cardiovascular disease on surgical mortality and morbidity

Lancet 1996; **348:** 1055–60

Jeffrey L Carson, Amy Duff, Roy M Poses, Jesse A Berlin, Richard K Spence, Richard Trout, Helaine Noveck, Brian L Strom

Discussion

This is the largest study to date on the natural history of anaemic patients undergoing surgery without transfusions.[8] It shows that the overall risk of mortality increases as the haemoglobin concentration decreases, and that even mild anaemia may be associated with some increase in death risk.

morbidity. These results strongly suggest that patients with underlying cardiovascular disease are less tolerant of anaemia than are patients without cardiovascular disease.

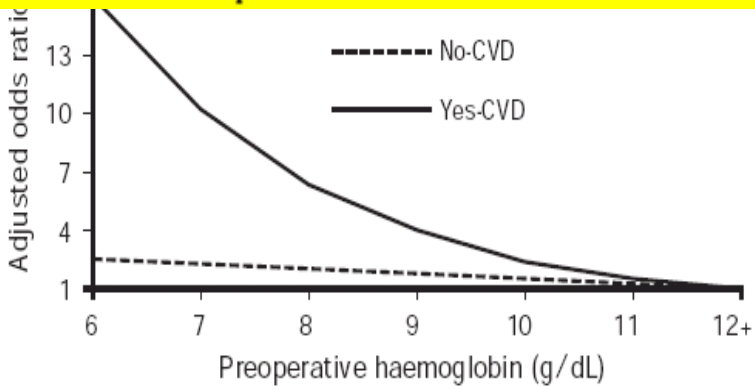

Figure: **Adjusted odds ratio for mortality by cardiovascular disease and preoperative haemoglobin**

Preoperative haemoglobin (g/dL)	Total number (n=1958)	Dead (n=63)	% dead (exact 95% CI)	Unadjusted relative risk (95% CI)	Morbidity/ mortality (n=123)	% morbidity/ mortality (95% CI)	Unadjusted relative risk (95% CI)
<6·0	36	12	33·3 (18·6–51·0)	26·1 (13·6–50·1)	23	63·9 (46·2–79·2)	16·1 (11·3–23·0)
6·0–6·9	27	5	18·5 (6·3–38·1)	14·5 (5·8–36·2)	7	25·9 (11·1–46·3)	6·5 (3·3–13·0)
7·0–7·9	49	6	12·2 (4·6–24·7)	9·6 (4·0–23·1)	15	30·6 (18·3–45·4)	7·7 (4·7–12·6)
8·0–8·9	39	5	12·8 (4·3–27·4)	10·1 (3·9–25·7)	10	25·6 (13·0–42·1)	6·5 (3·6–11·7)
9·0–9·9	75	6	8·0 (3·0–16·6)	6·3 (2·6–16·3)	8	10·7 (4·7–19·9)	2·7 (1·3–5·4)
10·0–10·9	109	5	4·6 (1·5–10·4)	3·6 (1·4–9·5)	14	12·8 (7·2–20·6)	3·2 (1·9–5·6)
11·0–11·9	212	5	2·4 (0·8–5·4)	1·9 (0·7–4·9)	14	6·6 (3·7–10·8)	1·7 (0·9–2·2)
≥12·0	1411	18	1·3 (0·8–2·0)	1·0 (reference)	56	4·0 (3·0–5·1)	1·0 (reference)

Table 3: **Unadjusted relation between preoperative haemoglobin mortality and mortality and morbidity**

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Hemodilution and anemia in patients with cardiac disease: what is the safe limit?

Robert Tircoveanu and Philippe Van der Linden

Current Opinion in Anaesthesiology 2008, 21:66–70

At the level of the heart, oxygen extraction is already nearly maximal under resting conditions. Therefore, maintenance of myocardial oxygen delivery during normovolemic hemodilution (ANH) depends essentially on the increase in coronary blood flow. Animal studies showed that, in the presence of experimental coronary artery stenosis, tolerance to ANH is significantly reduced, myocardial ischemia and cardiac failure occurring at a higher hemoglobin concentration (around 6 g/dl) than in the absence of stenosis (around 3 g/dl) [23,24]. Interest-

Intraoperative Management of Extreme Hemodilution in a Patient with a Severed Axillary Artery

JianQiang Dai, MS,* WeiFeng Tu, PhD,† Zheng Yang, MD,† and RiHui Lin, MS†

Table 1. Summary of Hemoglobin Values and Fluids Administered from the Patient's Arrival in the Emergency Department to After Completion of Surgery

Time	Event	Hemoglobin concentration (g/dL)	Fluid administered
0	Arrival in ED	0.9	2500 mL BSS
1			500 mL HES
2			2250 mL BSS
3			1750 mL HES
4			
5			
6			
7			
8			1500 mL BSS
9			1166 mL HES
10			250 mL $NaHCO_3$
11			1000 mL plasma
12			750 mL BSS
13			584 mL HES
			600 mL PRBCs
14	Immediately postoperatively (surgery lasted for 12 h)	4.7	
	Total fluids administered from arrival in the ED to immediately after surgery		7000 mL BSS
			4000 mL HES
			250 mL $NaHCO_3$
			1000 mL plasma
			600 mL PRBCs

acked red blood cells.
osttransfusion hemoglobin concentration was 8.7 g/dL.

35.5°C during the operation. Intraoperative blood loss was minimal and urine output was 3900 mL. In total, 12,250 mL of fluid was infused from the patient's arrival in the emergency department to the completion of surgery; 2500 mL of salt solution and 500 mL of HES were infused preoperatively, and 4500 mL of salt solutions, 3500 mL of HES, 250 mL of $NaHCO_3$, and 1000 mL of plasma were infused intraoperatively. A summary of Hgb values and

orrhagic shock as the result of an uncontrolled severed axillary artery, was not transfused with erythrocytes for 12 hours because crossmatched compatible blood was not available at that institution. The patient's ABO Rh type was

(Anesth Analg 2010;111:1204–6)

Emergency Transfusion for Acute Severe Anemia: A Calculated Risk

November 2010 • Volume 111 • Number 5

Richard B. Weiskopf, MD

recent reexamination of those data, it was estimated that the median hemoglobin concentration associated with anemia-induced mortality is approximately 2.5 g/dL (R. B. Weiskopf, unpublished data, 2010). Cardiovascular disease increases that value[3] (also R. B. Weiskopf, unpublished

Emergency Transfusion for Acute Severe Anemia: A Calculated Risk

November 2010 • Volume 111 • Number 5

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hemoglobin concentration. Classic thought is that the amount of oxygen dissolved in plasma (the solubility of oxygen in plasma is 0.0031 mL/dL/mm Hg O_2) is too little to be of physiologic consequence. Whereas that may be so during ordinary circumstances with an F_{IO_2} of 0.21, dissolved oxygen can be of substantial benefit during severe anemia, when the F_{IO_2} and P_{AO_2} are high. Hyperoxia reduces mortality of pigs subjected to acute severe anemia and maintained at their critical hemoglobin concentra-

Emergency Transfusion for Acute Severe Anemia: A Calculated Risk

Richard B. Weiskopf, MD

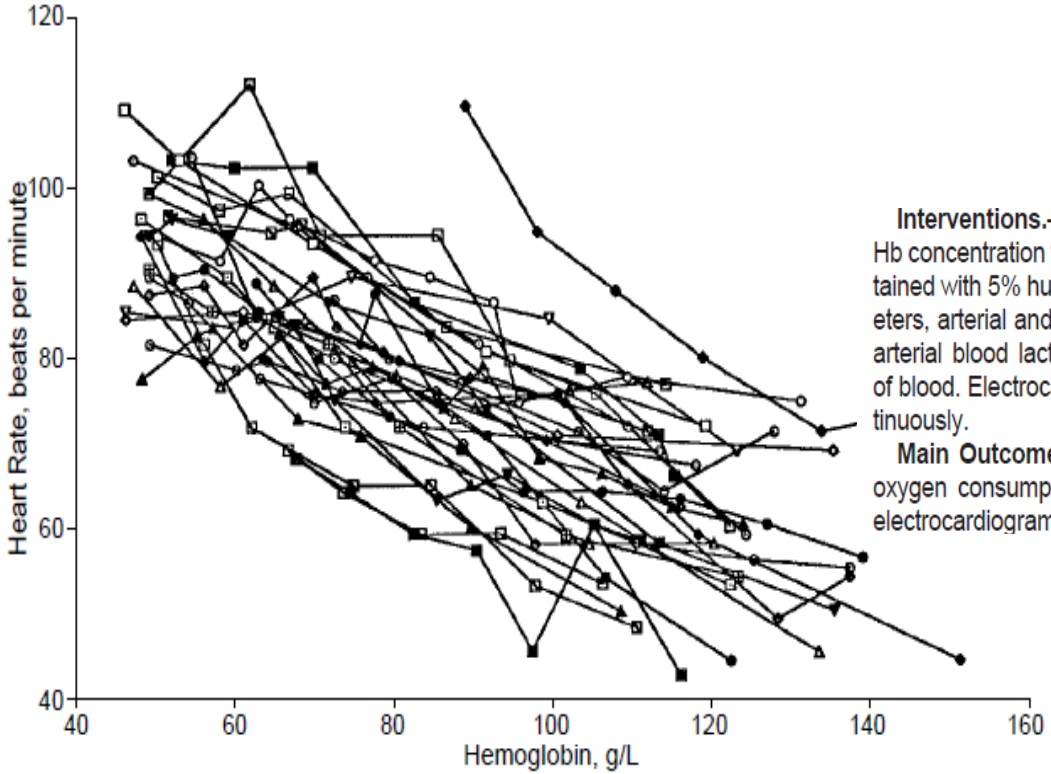
ologic effect of a Pao_2 in excess of 400 mm Hg has been estimated to be equivalent to approximately 3 g/dL hemoglobin[12] (also J. Feiner, et al., unpublished data, 2010). Thus, the physiologic effect of breathing oxygen when added to the patient's native hemoglobin of 0.7 g/dL produced a heart rate equivalent to nearly 4 g/dL hemoglobin, a value associated with approximately 80% survival (R. B. Weiskopf, unpublished data, 2010). Provision of a high FIO_2 can be a useful "bridge" until red cells are available for transfusion.

rocytes into the capillary network.[11] In conscious humans, Weiskopf *et al.*[12] recently demonstrated that an increase in cardiac output and oxygen extraction ratio allows the maintenance of adequate tissue oxygenation up to a hemoglobin concentration of 5.0 g/dl.

Human Cardiovascular and Metabolic Response to Acute, Severe Isovolemic Anemia

JAMA. 1998;279:217-221

Richard B. Weiskopf, MD; Maurene K. Viele, MD; John Feiner, MD; Scott Kelley, MD; Jeremy Lieberman, MD; Mariam Noorani; Jacqueline M. Leung, MD; Dennis M. Fisher, MD; William R. Murray, MD; Pearl Toy, MD; Mark A. Moore, MD



Interventions.—Aliquots of blood (450-900 mL) were removed to reduce blood Hb concentration from 131 (2) g/L to 50 (1) g/L [mean (SE)]. Isovolemia was maintained with 5% human albumin and/or autologous plasma. Cardiovascular parameters, arterial and mixed venous oxygen content, oxyhemoglobin saturation, and arterial blood lactate were measured before and after removal of each aliquot of blood. Electrocardiogram and, in a subset, Holter monitor were monitored continuously.

Main Outcome Measures.—"Critical" oxygen delivery (To_2) as assessed by oxygen consumption ($\dot{V}o_2$), plasma lactate concentration, and ST changes on electrocardiogram.

Human Cardiovascular and Metabolic Response to Acute, Severe Isovolemic Anemia

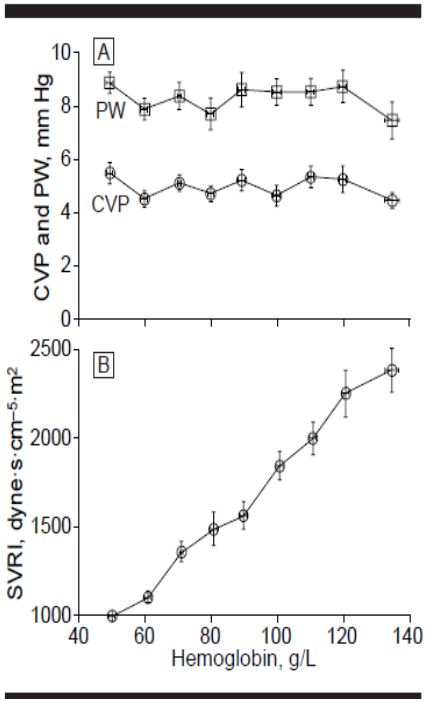
JAMA. 1998;279:217-221

Richard B. Weiskopf, MD; Maurene K. Viele, MD; John Feiner, MD; Scott Kelley, MD; Jeremy Lieberman, MD; Mariam Noorani; Jacqueline M. Leung, MD; Dennis M. Fisher, MD; William R. Murray, MD; Pearl Toy, MD; Mark A. Moore, MD

Table 2.—Response to Acute Isovolemic Anemia*

Variable	Hemoglobin Range 125-134 g/L (n=23)	45-54 g/L (n=28)
SVRI, dyne·s·cm^{-5}·m^2	2372 (541)	1001 (176)
HR, beats per minute	58 (11)	92 (12)
SVI, mL/m^2	52 (9)	62 (8)
CI, L/m^2	3.05 (0.69)	5.71 (0.87)
To_2, mL O_2·kg^{-1}·min^{-1}	13.5 (2.7)	10.7 (2.0)
S_vO_2, %	77.1 (3.3)	69.6 (5.6)
$\dot{V}O_2$, mL O_2·kg^{-1}·min^{-1}	3.01 (0.42)	3.42 (0.54)
Plasma lactate, mmol/L	0.77 (0.40)	0.62 (0.19)
Arterial blood pH	7.395 (0.016)	7.445 (0.025)
Base-excess, mEq/L	1.3 (1.5)	4.2 (2.2)
$\dot{V}O_2/To_2$	0.23 (0.03)	0.32 (0.04)

*Data are mean (SD). Group sizes are less than 32 because not all subjects had a hemoglobin concentration within the range described. The statistical results provided in the text refer to all data for all subjects: all variables shown in this table, except plasma lactate concentration, changed significantly with decreasing hemoglobin concentration. SVRI indicates systemic vascular resistance index; HR, heart rate; SVI, stroke volume index; CI, cardiac index; To_2, oxygen transport; S_vO_2, mixed venous oxyhemoglobin saturation; and $\dot{V}O_2$, oxygen consumption.



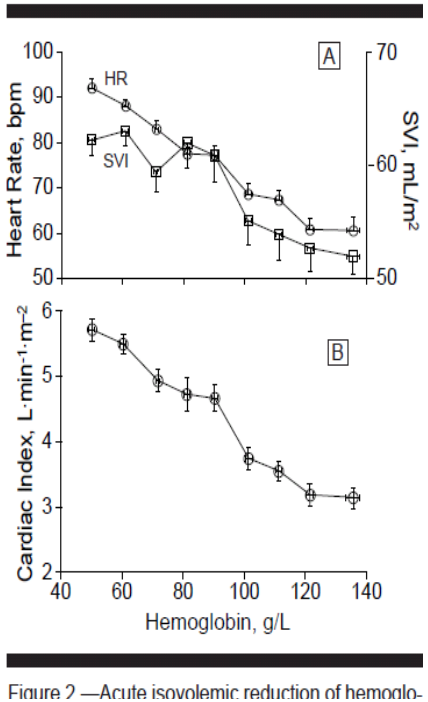


Figure 2.—Acute isovolemic reduction of hemoglo-

Human Cardiovascular and Metabolic Response to Acute, Severe Isovolemic Anemia

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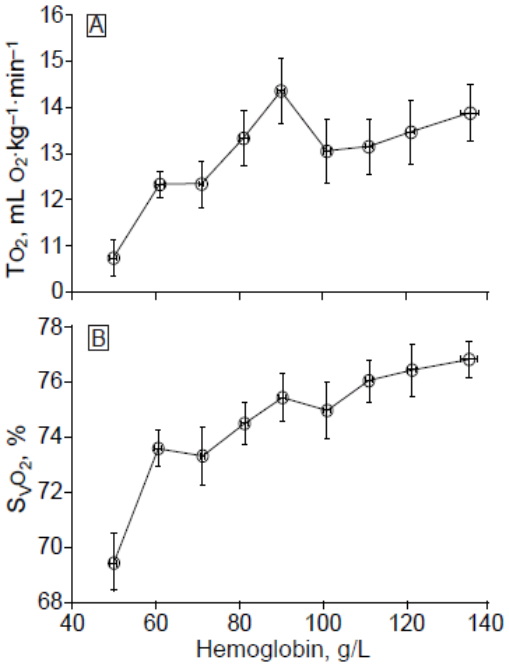


Figure 4.—Acute isovolemic reduction of hemoglobin concentration to 50 g/L decreased oxygen transport rate (To_2) (A; $P<.001$) and mixed venous oxyhemoglobin saturation (S_vO_2)(B; $P<.001$). Data are gathered into groups by hemoglobin increments of 10 g/L and represented as mean (SE) (N=32).

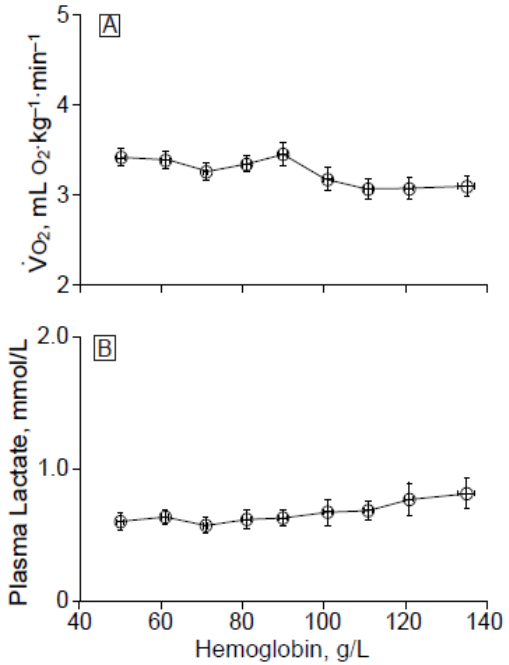


Figure 5.—Acute isovolemic reduction of hemoglobin concentration to 50 g/L increased oxygen consumption ($\dot{V}O_2$) (A; $P<.001$) but did not change plasma lactate concentration (B; $P=.09$). Data are gathered into groups by hemoglobin increments of 10 g/L and represented as mean (SE) (N=32).

The major finding of this study is that acute reduction of blood Hb concentration to 50 g/L in conscious healthy resting humans does not result in detectable inadequate systemic To_2. The systemic markers we used to detect consequences of inadequate To_2 ($\dot{V}O_2$ and plasma lactate concentration) did not demonstrate inadequate To_2 with decreased Hb concentration. The lack of significantly increased plasma lactate in any of the 32 subjects indicates, with a 95% assurance, that acute reduction of Hb concentration to 50 g/L would not produce lactic acidemia in more than 9% of the population.[8] It would

Anesthesiology 2000; 93:1011-6

Cardiovascular and Metabolic Response to Acute Normovolemic Anemia

Effects of Anesthesia

Brigitte E. Ickx, M.D., Michel Rigolet, M.D.,† Philippe J. Van der Linden, M.D., Ph.D.‡*

rocytes into the capillary network.[11] In conscious humans, Weiskopf *et al.*[12] recently demonstrated that an increase in cardiac output and oxygen extraction ratio allows the maintenance of adequate tissue oxygenation up to a hemoglobin concentration of 5.0 g/dl.

The influence of anesthesia on these compensatory mechanisms remains poorly studied in humans. Because most anesthetic agents decrease myocardial contractility and venous return,[13,14] they may blunt the compensatory increase in cardiac output observed during acute normovolemic hemodilution. The use of opioids, such as

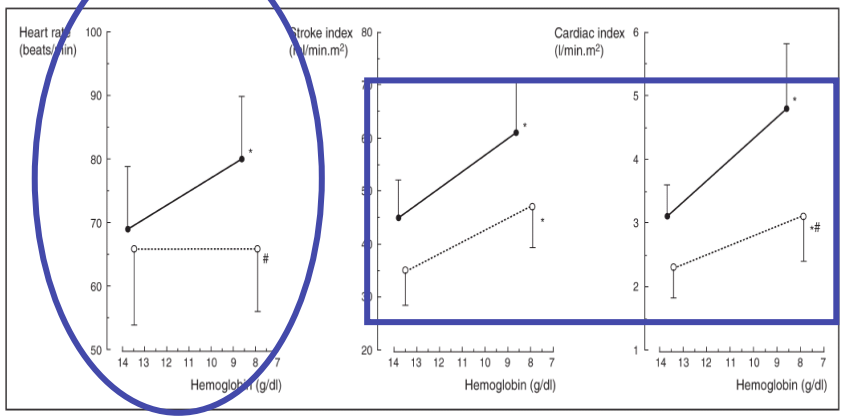
Anesthesiology 2000; 93:1011-6

Cardiovascular and Metabolic Response to Acute Normovolemic Anemia

Effects of Anesthesia

Brigitte E. Ickx, M.D., Michel Rigolet, M.D.,† Philippe J. Van der Linden, M.D., Ph.D.‡*

Figure 1 Cardiac index response to acute normovolemic hemodilution



Cardiac index response to acute normovolemic hemodilution (ANH) in awake (closed circles) and anesthetized (open circles) patients undergoing major abdominal surgery. *$P < 0.05$ versus before hemodilution. #Significantly different response to ANH between groups. Adapted with permission from [36].

Do_2 (ml · min^{-1} · m^{-2})	Awake		575 ± 90	577 ± 112
	Anesthetized	616 ± 146	424 ± 116§	349 ± 93§
Vo_2 (ml · min^{-1} · m^{-2})	Awake		121 ± 17	145 ± 29†
	Anesthetized	120 ± 27	88 ± 14§	94 ± 11§‖
O_2ER (%)	Awake		21.4 ± 3.3	25.4 ± 4.0*
	Anesthetized	19.8 ± 3.1	21.7 ± 5.1	28.1 ± 6.0†
Svo_2 (%)	Awake		76.9 ± 3.7	74.6 ± 4.4
	Anesthetized	78.5 ± 3.6	77.5 ± 5.7	72.2 ± 6.0†

In the awake group, ANH was associated with an increase in cardiac index, related to both an increase in heart rate and stroke index (table 2). Systemic vascular resistance decreased, and left ventricular stroke work index increased. Mean pulmonary artery pressure, right ventricular end-diastolic volume index, and right ventricular stroke work index also increased. Despite the decrease in arterial oxygen content, Do_2 remained stable, but $\dot{V}o_2$ increased, resulting in an increase in oxygen extraction (table 2).

In the anesthetized group, ANH was associated with a increase in cardiac index, related solely to an increase in stroke index (table 2). Mean arterial pressure and systemic vascular resistance decreased. Right ventricular end-diastolic volume index increased. The decrease in arterial oxygen content was associated with a slight decrease in Do_2, but $\dot{V}o_2$ was maintained as oxygen extraction increased. Between the two groups, there was a significant different response to ANH for body temperature, heart rate, cardiac index, and $\dot{V}o_2$.

Perioperative Use of β-Adrenergic Antagonists and Anemia

Anesthesiology 2010; 112:12-5

Known Knowns, Known Unknowns, Unknown Unknowns; and Unknown Knowns

Richard B. Weiskopf, M.D.,

data are available, although β-adrenergic antagonists seem to protect the myocardium of high-risk patients and may well be of lesser or no efficacy for patients at lesser risk, it would seem prudent to avoid those agents that substantially impair the cardiac response to acute severe anemia when that or substantial hemorrhage is anticipated.

Weiskopf and colleagues[39] made the interesting observation that the deterioration of neurocognitive function after isovolemic hemodilution from a hemoglobin of 12.7 ± 1.0 to 5.7 ± 0.3 was reversed by increasing PaO_2 from around 100 to 400 mm Hg. This value is equivalent to an increase in hemoglobin concentration of roughly 3 g/dL.[20] Similar results have been found in animal studies.[33]

Weiskopf R, Viele M, Feiner J, et al. Human cardiovascular and metabolic response to acute, severe, isovolemic anemia. JAMA 1998;279:217–21.

Weiskopf R, Kramer J, Viele M, et al. Acute severe isovolemic anemia impairs cognitive function and memory in humans. Anesthesiology 2000;92(6): 1646–52.

Weiskopf R, Feiner J, Hopf H, et al. Oxygen reverses deficits of cognitive function and memory and increased heart rate induced by acute severe isovolemic anemia. Anesthesiology 2002;96:871–7.

Anesthesiology
2000; 92:1646–52

Acute Severe Isovolemic Anemia Impairs Cognitive Function and Memory in Humans

Richard B. Weiskopf, M.D., Joel H. Kramer, Psy.D.,† Maurene Viele, M.D.,‡ Mireille Neumann, M.D.,§ John R. Feiner, M.D.,‖ Jessica J. Watson, M.A.,# Harriet W. Hopf, M.D.,** Pearl Toy, M.D.††*

tested the hypothesis that acute severe decreases of hemoglobin concentration alters human cognitive function.

Methods: Nine l
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Results: No test showed any change in reaction time or error rate at hemoglobin concentration of 7 g/dl compared with the data at the baseline hemoglobin concentration of 14 g/dl. Reaction time, but not error rate, for horizontal addition and digit–symbol substitution test (DSST) increased at hemoglobin 6 g/dl (mean horizontal addition, 19%; 95% confidence interval [CI],) and further at 5 g/dl
, 6–79%; mean DSST,
delayed memory was
at 6 g/dl. Return of
o baseline, except for
and returned to base-
sion of all autologous

prospective data to demonstrate subtle degraded human function with acute anemia of hemoglobin concentrations of 6 and 5 g/dl. This reversibility of these decrements with erythrocyte transfusion suggests that our model can be used to test the efficacy of erythrocytes, oxygen therapeutics, or other treatments for acute anemia. (Key words: Brain function; erythrocytes; hemodilution; transfusion.)

Anesthesiology
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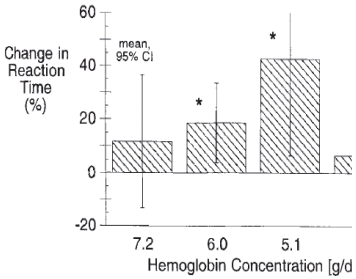


Fig. 1. Reaction time for horizontal addition increased a globin concentrations of 6 and 5 g/dl but not at 7 g/ pared with 14 g/dl in nine healthy subjects. Data are 95% confidence intervals. *P < 0.05 for comparisor difference between experimental and control days for tl ence between the value at the indicated hemoglobin conce and the value at the baseline hemoglobin concentration o

We have found that acute isovolemic anemia to a hemoglobin concentration of ≤ 6 g/dl results in mild, reversible decrements in reaction time, and that a hemoglobin concentration of 5 g/dl reversibly impairs immediate and delayed memory. While maintaining high levels of accuracy, subjects exhibited slower reaction at hemoglobin concentration of ≤ 6 g/dl and performed less well on measures of immediate and delayed recall at a hemoglobin concentration of 5 g/dl.

The changes in human cognition after acute isovolemic anemia are similar to those found in subjects experiencing hypobaric hypoxia. Performance of the

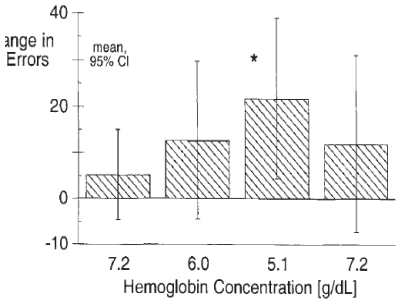


Fig. 4. Delayed memory is impaired at a hemoglobin concentration of 5 g/dl but not 6 or 7 g/dl, compared with a hemoglobin concentration of 14 g/dl in nine healthy subjects. Data are mean ± 95% confidence interval. *P < 0.05 for comparison of the difference between experimental and control days for the difference between the value at the indicated hemoglobin concentration and the value at the baseline hemoglobin concentration of 14 g/dl.

Hemoglobin Concentration [g/dL]

Fig. 2. Reaction time for digit–symbol substitution increased at hemoglobin concentrations of 6 and 5 g/dl but not at 7 g/dl, compared with 14 g/dl in nine healthy subjects. Data are mean ± 95% confidence interval. *P < 0.05 for comparison of the difference between experimental and control days for the difference between the value at the indicated hemoglobin concentration and the value at the baseline hemoglobin concentration of 14 g/dl.

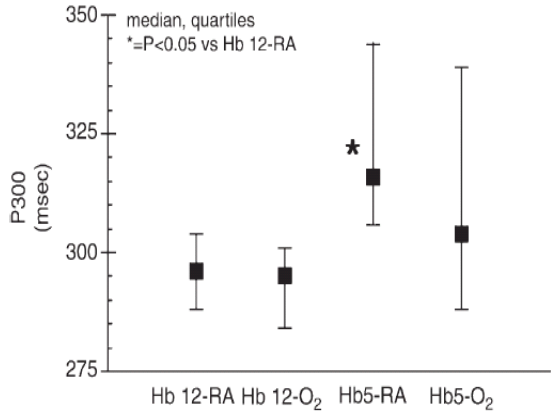


Fig. 1. Auditory P300 latencies in nine volunteers at hemoglobin concentration of 12.4 g/dL breathing air (Hb12-Air) or oxygen (Hb12-O2), and at hemoglobin concentration of 5.1 g/dL breathing air (Hb5-Air) or oxygen (Hb5-O2). Data are median and quartiles. *=$P<0.05$ versus Hb12-Air.

Clinical Neurophysiology 116 (2005) 1028–1032

volemic anemia impairs central processing as determined by P300 latency

*, Pearl Toy[b], Harriet W. Hopf[c], John Feiner[d], Heather E. Finlay[b],

nses to clinical tests of cognitive function. We tested the hypothesis that these slowed responses ealthy unmedicated humans result from impaired central processing.

he latency of the P300 peak in nine healthy volunteers at each volunteer's baseline hemoglobin ic hemodilution to Hb 5 g/dL. At both Hb concentrations, the P300 latency was measured twice: 0% oxygen, administered in random order.

Results: Anemia increased P300 latency significantly from baseline values ($P<0.05$). Breathing oxygen during induced anemia resulted in a P300 latency not different from that at baseline when breathing air ($P=0.5$) or oxygen ($P=0.8$).

Conclusions: Impaired central processing is, at least in part, responsible for the slowed responses and deficits of cognitive function that occur during acute isovolemic anemia at Hb 5–6 g/dL.

Significance: The P300 latency appears to be a potential measure of inadequate central oxygenation. In healthy young adults with acute anemia, erythrocytes should be transfused to produce Hb > 5–6 g/dL. As a temporizing measure, administration of oxygen can reverse the cognitive deficits and impaired central processing associated with acute anemia.

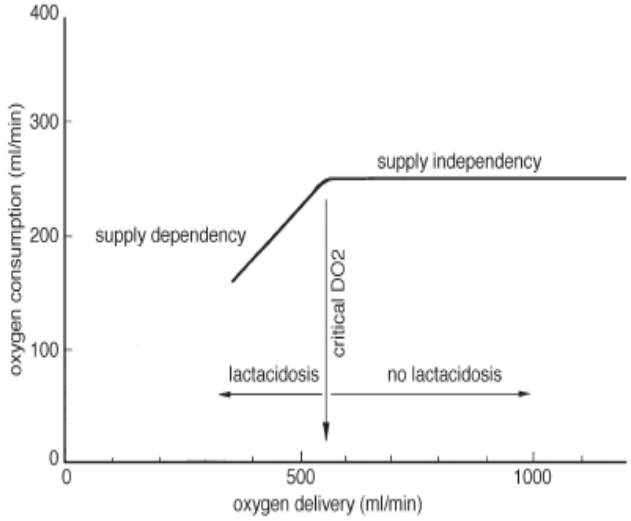
BMJ 2010;341:c4408

Managing anaemia in critically ill adults

Timothy S Walsh,[1] Duncan L A Wyncoll,[2] Simon J Stanworth[3]

high or rising lactate concentration and a low or falling central venous haemoglobin oxygen saturation (measured from a central venous catheter) are clinically useful triggers that signal the need to increase oxygen delivery. When

DO2, VO2 and O2ER



Global oxygen consumption (VO_2) which describes the amount of oxygen consumed by the whole body per minute ranges under physiological conditions in a normal adult from 200 to 300 ml/min whereas DO_2 ranges from 800 to 1200 ml/min. The relationship VO_2/DO_2 defines the oxygen extraction ratio (O_2ER) which is thus in the range of 20 to 30%. A normal VO_2/DO_2-relationship is illustrated in Figure 1. It

In analogy to DO_2, the quantity of oxygen consumed by the whole body (VO_2) is calculated as follows:

$$VO_2 = CO \times (CaO_2 - CvO_2)$$

where CvO_2 is the oxygen content of venous blood after venous admixture of all organs. The normal VO_2 for a conscious resting subject breathing room air is $\sim 250\,mL\,O_2\,min^{-1}$, thus only 25% of the delivered oxygen are actually consumed. The unextracted oxygen forms the reserve which may be used under stress conditions (exercise, anaemia). It

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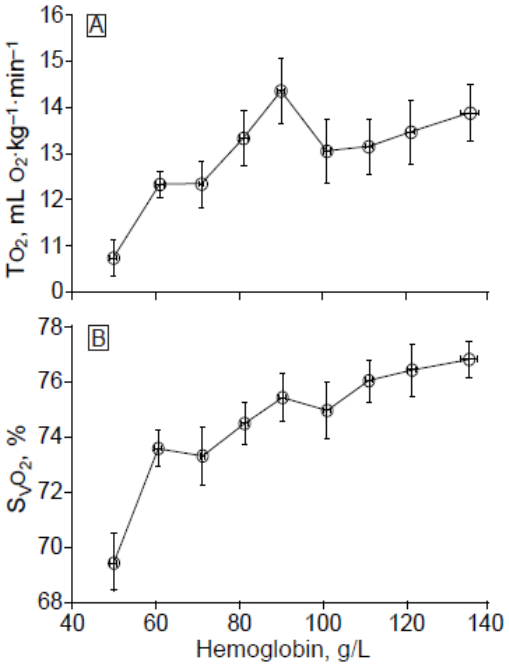


Figure 4.—Acute isovolemic reduction of hemoglobin concentration to 50 g/L decreased oxygen transport rate (To_2) (A; $P<.001$) and mixed venous oxyhemoglobin saturation (S_vO_2)(B; $P<.001$). Data are gathered into groups by hemoglobin increments of 10 g/L and represented as mean (SE) (N=32).

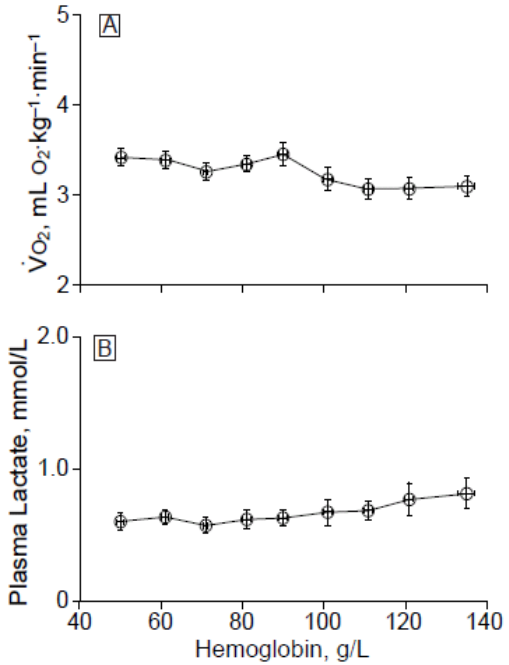


Figure 5.—Acute isovolemic reduction of hemoglobin concentration to 50 g/L increased oxygen consumption ($\dot{V}O_2$) (A; $P<.001$) but did not change plasma lactate concentration (B; $P=.09$). Data are gathered into groups by hemoglobin increments of 10 g/L and represented as mean (SE) (N=32).

The major finding of this study is that acute reduction of blood Hb concentration to 50 g/L in conscious healthy resting humans does not result in detectable inadequate systemic To_2. The systemic markers we used to detect consequences of inadequate To_2 ($\dot{V}O_2$ and plasma lactate concentration) did not demonstrate inadequate To_2 with decreased Hb concentration. The lack of significantly increased plasma lactate in any of the 32 subjects indicates, with a 95% assurance, that acute reduction of Hb concentration to 50 g/L would not produce lactic acidemia in more than 9% of the population.[8] It would

Critical Care 2010, 14:213

Venous oxygen saturation as a physiologic transfusion trigger

Benoit Vallet*, Emmanuel Robin and Gilles Lebuffe

Venous oxygen saturation is a clinical tool which integrates the whole body oxygen uptake-to-delivery (VO_2-DO_2) relationship. In the clinical setting, in the absence of pulmonary artery catheter (PAC)-derived mixed venous oxygen saturation (SvO_2), the central venous oxygen saturation ($ScvO_2$) is increasingly being used as a reasonably accurate surrogate [1]. Central venous catheters

Comparison of central-venous to mixed-venous oxygen saturation during changes in oxygen supply/demand.

K Reinhart, T Rudolph, D L Bredle, L Hannemann and S M Cain

Chest 1989;95;1216-1221

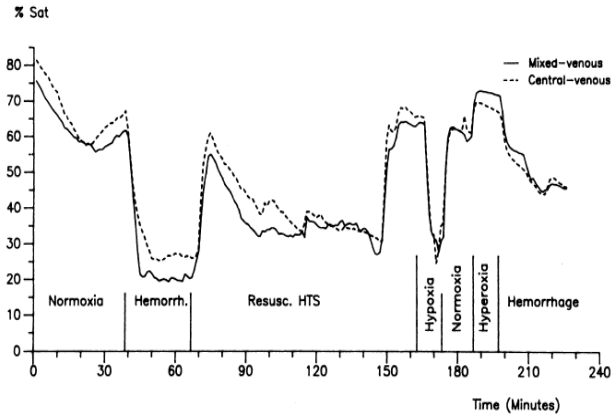


FIGURE 3. Time course of mixed and central venous O_2 saturation during different experimental perturbations of the animal. HTS = hypertonic saline solution (7.5%).

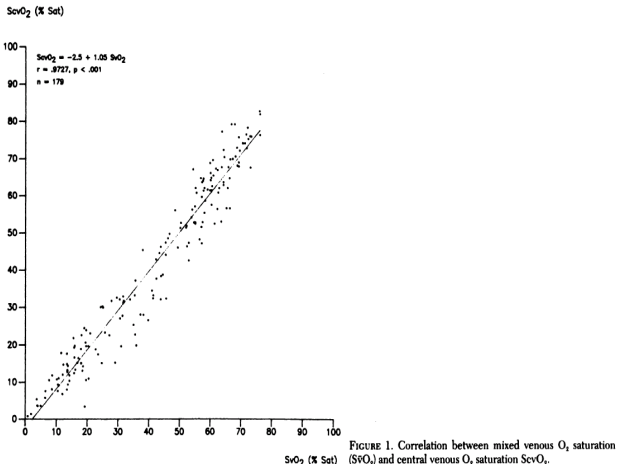


FIGURE 1. Correlation between mixed venous O_2 saturation ($S\bar{v}O_2$) and central venous O_2 saturation $ScvO_2$.

Table 1—*Mean Values and Correlations for Mixed and Central Venous Fiberoptic Catheters under Various Conditions

Condition	$S\bar{v}O_2$	$ScvO_2$	r	n	$\lvert S\bar{v}O_2\text{-}ScvO_2\rvert$
Total	53 ± 16	52 ± 15	0.96	29531	3.7 ± 2.9
Control	59 ± 14	57 ± 15	0.98	14167	2.8 ± 2.0
Hemorrhage	33 ± 14	37 ± 12	0.94	1490	6.0 ± 3.1

$ScvO_2$ was expected to be slightly lower than $S\bar{v}O_2$ during steady-state conditions, due to a relatively large contribution of highly saturated venous renal effluent to the inferior vena cava.[15] Our data in Table 1 are in general agreement with this expectation. In nonshock patients[12,14] and healthy volunteers[16] similar differences between mixed and central venous saturations have been reported. During hypoxia and hemorrhagic shock, a redistribution of blood flow away from renal and splanchnic beds to the heart and brain would tend to reverse this difference.[17,18] Such redistribution is consistent with the somewhat higher $ScvO_2$ saturations we observed in both hemorrhage and hypoxia (Table

Intensive Care Med (2004) 30:1572–1578
DOI 10.1007/s00134-004-2337-y

ORIGINAL

Konrad Reinhart
Hans-Jörg Kuhn
Christiane Hartog
Donald L. Bredle

Continuous central venous and pulmonary artery oxygen saturation monitoring in the critically ill

The present data confirm the findings of others that in critically ill patients with circulatory failure from various causes $ScvO_2$ is generally higher than SvO_2 measured in the pulmonary artery. In healthy individuals $ScvO_2$ is typically slightly lower than SvO_2 [27, 28]. This, however, is not true in heart failure, cardiac shock [16, 17], and severe sepsis [29, 30]. In circulatory shock and heart failure blood flow is redistributed away from the hepatosplanchnic region to the coronary and cerebral circulation, and in sepsis there is a marked increase in O_2 consumption in the hepatosplanchnic region [30, 31]. This results in greater O_2 desaturation from venous blood that drains into the hepatic vein and inferior vena cava, re-

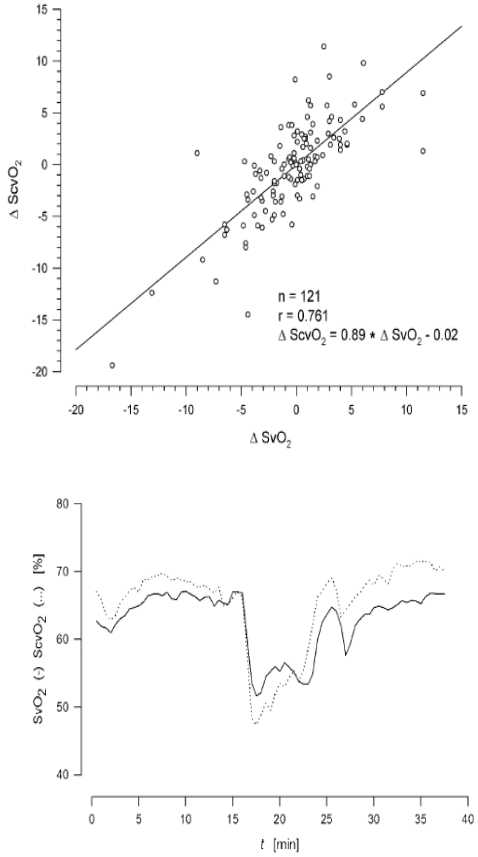


Fig. 4 Time course of continuously measured SvO_2 and $ScvO_2$ in a patient with acute respiratory distress syndrome who developed tension pneumothorax that was treated by insertion of a chest tube

Venous oxygen saturation as a physiologic transfusion trigger

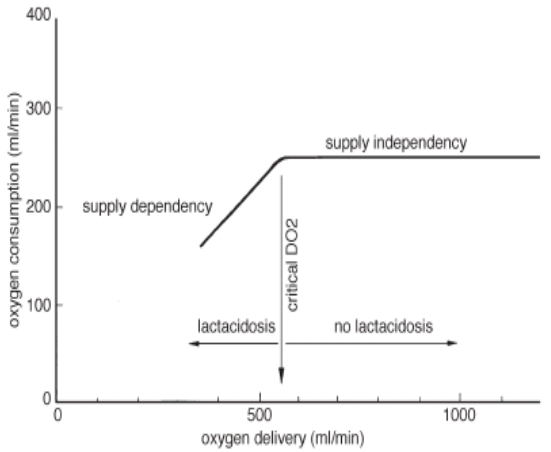
Critical Care 2010, 14:213

Benoit Vallet*, Emmanuel Robin and Gilles Lebuffe

When DO_2 decreases, VO_2 is maintained (at least initially) by an increase in oxygen extraction (O_2ER) since $O_2ER = VO_2/DO_2$. As $VO_2 \approx (SaO_2 - SvO_2) \times (Hb \times 1.34 \times CO)$ and $DO_2 \approx SaO_2 \times Hb \times 1.34 \times CO$, O_2ER and SvO_2 are thus linked by a simple equation: $O_2ER \approx (SaO_2 - SvO_2)/SaO_2$ or even simpler: $O_2ER \approx 1 - SvO_2$. Assuming $SaO_2 = 1$ [3], if SvO_2 is 40 %, then O_2ER is 60 %.

Because it integrates Hb, cardiac output, VO_2 and SaO_2, the venous oxygen saturation therefore helps to assess the VO_2-DO_2 relationship and tolerance to anemia during blood loss.

observations [7] we can conclude that $ScvO_2$ appears to be an interesting parameter to help with transfusion decisions in hemodynamically unstable patients with severe sepsis or in stable high-risk surgical patients equipped with a CVC. $ScvO_2$ can be proposed as a simple and universal physiologic transfusion trigger. This



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ANEMO '11

The clinical utility of an index of global oxygenation for guiding red blood cell transfusion in cardiac surgery

David Orlov, Rachel O'Farrell, Stuart A. McCluskey, Jo Carroll, Humara Poonawala, Siroos Hozhabri, and Keyvan Karkouti

$$O_2ER = \frac{V_{O_2}}{D_{O_2}} = \frac{CO(CaO_2 - CvO_2)}{CO(CaO_2)}$$

Where:

VO_2 = oxygen uptake
DO_2 = oxygen delivery

CO = cardiac output[1]
CaO_2 = arterial oxygen content[2] = (Hb x 1.36 x SaO_2) + (0.0031 x PaO_2)
CvO_2 = mixed venous oxygen content[3] = (Hb x 1.36 x SvO_2) + (0.0031 x PvO_2)

Hb = Hemoglobin concentration (g/L)
SaO_2 = Arterial oxygen saturation (%)
PaO_2 = Arterial oxygen tension (mm Hg)
SvO_2 = Mixed venous oxygen saturation (%)
PvO_2 = Mixed venous oxygen tension (mm Hg)

[1] Measured using thermodilution method
[2] Measured using arterial indwelling catheter
[3] Measured using pulmonary artery indwelling catheter

Fig. 1. O_2ER formula.

$$O_2ER = \frac{\left[(Hb \times 1.36 \times S_aO_2) + (0.0031 \times P_aO_2)\right] - \left[(Hb \times 1.36 \times S_vO_2) + (0.0031 \times P_vO_2)\right]}{(Hb \times 1.36 \times S_aO_2) + (0.0031 \times P_aO_2)}$$

Fig. 2. Net O_2ER calculation.

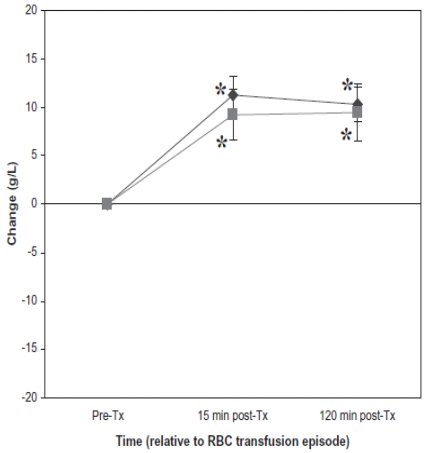
The clinical utility of an index of global oxygenation for guiding red blood cell transfusion in cardiac surgery

David Orlov, Rachel O'Farrell, Stuart A. McCluskey, Jo Carroll, Humara Poonawala, Siroos Hozhabri, and Keyvan Karkouti

TABLE 3. Hb concentrations and O_2ERs

	Transfusion episodes (n = 62)*		
	Baseline O_2ER >30% (n = 27)	Baseline O_2ER ≤ 30% (n = 35)	p Value
Hb concentration (g/L)			
Before transfusion	78.1 ± 7.6 (27)	78.3 ± 16.7 (35)	NS
After transfusion			
15 min	87.0 ± 8.3 (20)	88.5 ± 22.3 (30)	NS
2 hr	86.8 ± 8.94 (19)	87.5 ± 19.6 (32)	NS
Change from baseline			
15 min	+9.24 ± 11.7 (20)	+10.8 ± 9.3 (30)	NS
2 hr	+9.51 ± 12.4 (19)	+9.46 ± 9.4 (32)	NS
O_2ER (%)			
Before transfusion	39.8 ± 9.0 (27)	23.1 ± 4.9 (35)	<0.001
After transfusion			
15 min	33.4 ± 10.2 (20)	23.9 ± 7.8 (26)	<0.001
2 hr	33.3 ± 9.1 (17)	24.7 ± 8.2 (30)	0.001
Change from baseline			
15 min	–5.2 ± 7.8 (20)	+0.7 ± 5.8 (26)	0.004
2 hr	–3.8 ± 8.0 (17)	+1.4 ± 7.0 (30)	0.02

* Data are reported as mean ± SD. Data were not available for all measures; the number of episodes used for each analysis is shown in parentheses. NS = not significant.



Impossibile visualizzare l'immagine. La memoria del computer potrebbe essere insufficiente per aprire l'immagine oppure l'immagine potrebbe essere danneggiata. Riavviare il computer e aprire di nuovo il file. Se viene visualizzata di nuovo la x rossa, potrebbe essere necessario eliminare l'immagine e inserirla di nuovo.

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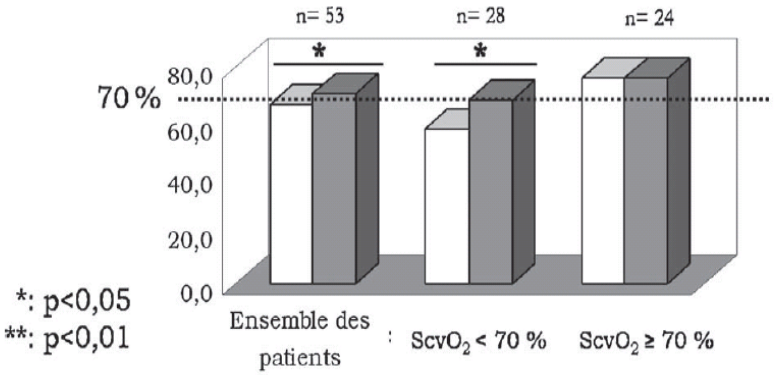
Adamczyk S, Robin E, Barreau O, *et al.*: [Contribution of central venous oxygen saturation in postoperative blood transfusion decision]. *Ann Fr Anesth Reanim* 2009, **28**:522–530.

Threshold value of Hb (g/dl)	Clinical context
10	• Acute coronary syndrome
9	• Ischemic heart disease • Stable heart failure
8	• Age > 75 • Severe sepsis
7	• Others



□ $ScvO_2$ avant TE	(%)	66,4	[57,6-76,2]	57,8	[53,0-59,7]	76,8 [76,1-80,0]
■ $ScvO_2$ après TE	(%)	71,0*	[71,5-72,2]	68,5**	[62,4-70,5]	76,5 [72,5-79,9]
Nombre de culots		2	[2,0-2,2]	2	[2,0-2,0]	2 [2,0-2,0]
Hb avant TE (g/dL)		7,6	[7,3-8,2]	7,6	[7,3-8,5]	7,7 [7,4-8,2]
Hb après TE (g/dL)		9,8**	[9,1-10,4]	9,5**	[8,9-10,0]	10,0** [9,3-10,7]

fusion. The $ScvO_2$ threshold value of 69.5% (sensitivity 82%; specificity 76%) was validated with a receiver operator characteristic (ROC) curve analysis (Figure 1).

groups. Blood transfusion provided a significant and approximately similar increase in hemoglobin concentration for all patients in the four groups but the $ScvO_2$ value increased significantly only in patients with $ScvO_2$ < 70% before blood transfusion (Figure 2 and Table 2).

Venous oxygen saturation as a physiologic transfusion trigger

Benoit Vallet*, Emmanuel Robin and Gilles Lebuffe

Critical Care 2010, 14:213

Conclusion

Physiologic transfusion triggers should progressively replace arbitrary Hb-based transfusion triggers [19]. The same conclusions were drawn by Orlov *et al.* in a recent trial using a global oxygenation parameter for guiding RBC transfusion in cardiac surgery [20]. The use of goal-directed erythrocyte transfusions should render the management of allogeneic red cell use more efficient and should help: 1) in saving blood and avoiding unwanted adverse effects; and 2) in promoting and optimizing the adequacy of this life-saving treatment [16]. These 'physiologic' transfusion triggers can be based on signs and symptoms of impaired global (lactate, SvO_2 or $ScvO_2$) or, even better, regional tissue (EKG ST-segment, DSST or P300 latency) oxygenation; they do, however, have to include two important simple hemodynamic targets: heart rate and MAP or systolic arterial pressure.