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Measurement of central and mixed venous-to-arterial carbon dioxide differences in cardiac surgery patients

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Abstract

Background: Measurement of central venous-to-arterial CO_2 difference (p(cv-a)CO_2) as an estimate of mixed venous-to-arterial CO_2 difference (p(v-a)CO_2) has been recommended as an supplementary parameter to identify the inadequacy of tissue oxygenation in septic and postoperative non-cardiac high risk surgery patients. This study investigates the agreement between p(cv-a)CO_2 and p(v-a)CO_2, and explores the relationship of p(v-a)CO_2 with parameters of global and regional tissue oxygenation.
Methods: Simultaneous measurements of p(cv-a)CO_2 and p(v-a)CO_2 were performed in postoperative cardiac surgery patients immediately before and after cardiopulmonary bypass (CPB), and up to 6 hours post CPB. In addition, parameters of global blood flow and tissue oxygenation, i.e. cardiac index, mixed venous oxygen saturation (SvO_2), arterial lactate, and regional blood flow, i.e. gastric tonometry were assessed. Pooled data were used for Bland-Altman and correlation analysis, as appropriate.
Results: Although significantly correlated, p(cv-a)CO_2 and p(v-a)CO_2 showed large limits of agreement (6.7 mmHg, percentage error of 115 %). Correlation analyses revealed no meaningful correlation between p(v-a)CO_2 and CI, SvO_2, arterial lactate, and p(g-a)CO_2 (R^2: 0.013, 0.007, 0.000, 0.006, respectively, with $p > 0.05$ each).
Conclusions: In cardiac surgery patients p(cv-a)CO_2 cannot be used as an estimate of p(v-a)CO_2 with acceptable accuracy. There is no evidence that measurements of p(cv-a)CO_2 or p(v-a)CO_2 could help diagnose global or regional tissue hypoxia in this patient group.

Key words: central-venous-to-arterial carbon dioxide difference, gastric tonometry, mixed venous-to-arterial carbon dioxide difference, arterial lactate, cardiac surgery, mixed venous oxygen saturation, tissue oxygenation, hemodynamic

Introduction

Since the landmark study by Rivers et al. [1] measurement of central venous oxygen saturation ($ScvO_2$) has been extensively used in intensive care and perioperative medicine. $ScvO_2$ serves as an estimate of true mixed venous oxygen saturation (SvO_2) and therefore oxygen delivery/uptake relationship [2]. The physiological limitations of $ScvO_2$, usually measured in the superior venae cava, are well known, and in certain patients large differences between $ScvO_2$ and SvO_2 may occur [3]. Beside this and the fact that Rivers et al. studied severly septic patients during the first 6 hours after hospital admission, $ScvO_2$ meas-

urements have been recommended in perioperative elective cardiac surgery patients [4].

Recent studies in high risk major abdominal surgery and septic shock revealed that a $ScvO_2$ > 70% may not rule out the inadequacy of tissue oxygenation [5,6]. In this aspect the authors recommended the measurement of the central venous-to-arterial CO_2 difference ($p(cv\text{-}a)CO_2$) as a complementary target to identify persistent ischaemic hypoxia [5,6]. $P(cv\text{-}a)CO_2$ is an estimate of mixed venous-to-arterial CO_2 difference ($p(v\text{-}a)CO_2$) and may have the same limitations as the $ScvO_2$, e.g. the exact sampling site, the haemodynamic condition, and the level of sedation. Patients after cardiac surgery exhibit certain problems after hypothermic cardiopulmonary bypass (CPB), namely rewarming, reperfusion, inflammation, and are often haemodynamically compromised to a certain degree. This may have a profound effect on regional and global perfusion in the immediate postoperative period, influencing $p(cv\text{-}a)CO_2$. Thus, whether a widening of the $p(cv\text{-}a)CO_2$ in the face of a $ScvO_2$ > 70% could also serve as a complementary target to identify inadequate global or regional tissue oxygenation in cardiac surgery patients is yet unknown.

Therefore we re-analyzed a study published before [7] where simultaneous measurements of $ScvO_2$, SvO_2, $pcvCO_2$, $pvCO_2$, arterial carbon dioxide pressure ($paCO_2$), gastric luminal carbon dioxide pressure ($pgCO_2$), cardiac index (CI), and arterial lactate levels were performed.

The aim of this study was to study the agreement between $p(cv\text{-}a)CO_2$ and $p(v\text{-}a)CO_2$, and explore the relationship of $p(v\text{-}a)CO_2$ with parameters of regional blood flow, i.e. gastric tonometry and global tissue oxygenation, namely SvO_2, and arterial lactate levels.

Material and methods

This is a re-analysis of a prospective observational study in elective cardiac surgery patients, investigating intramyocardial oxygen monitoring [7].

Following approval by the local ethical committee of the University of Lübeck and written informed consent, 28 patients underwent standard CABG. The data of three patients experiencing perioperative complications have been excluded from this analysis and have been published before [8]. Therefore 25 remaining patients were analysed.

Anaesthesia and surgical technique

Anaesthesia was induced with etomidate (0.3-0.5 $mg \cdot kg^{-1}$) and sufentanyl (0,5-1 $\mu g \cdot kg^{-1}$) and maintained with continuous infusions of propofol (5-8 $mg \cdot kg^{-1}h^{-1}$) and sufentanyl (0.5-1 $\mu g \cdot kg^{-1}h^{-1}$). Muscle relaxation was achieved with pancuronium bromid (0.1 $mg \cdot kg^{-1}$). Patients were mechanically ventilated in a volume controlled mode with 100% oxygen throughout the surgical procedure; the respiratory rate was adjusted to achieve normocapnia. Intraoperative fluid management was adjusted to achieve and maintain a central venous pressure between 8 to 12 mmHg and a pulmonary artery capillary occlusion pressure (PAOP) between 15 to 18 mmHg. Volume replacement was performed with Ringer's solution and gelatine polysuccinate, as appropriate. Naso-pharyngeal and rectal temperature was continuously measured during the whole observation time. Standard CABG was performed in moderate hypothermia (32°C naso-pharyngeal) with a membrane oxygenator (Hilite; Medos, Stolberg, Germany) and a roller pump (Stöckert, München, Germany). The pump was primed with 1.430 ml Ringer`s solution, 250 ml of 20% mannitol, and 20 ml 8.4% (1 M) natriumbicarbonate. After median sternotomy and harvesting of the bypass grafts, patients were fully heparinized according to body weight in a dose of 300 IE/kg body weight. Activated clotting time was kept greater than 450 seconds. Aortic and two stage venous cannulation was used and after cross-clamping the heart was arrested using antegrade

cold cristalloid cardioplegia which was repeated every 20 minutes. Mean arterial blood pressure (MAP) during CPB was maintained at 60 to 80 mmHg. Vasopressors (norepinephrine) were applied, if necessary.

Measurements

Intra- and postoperatively all parameters were taken before CPB and every hour up to 6 hours after CPB. All patients were mechanically ventilated as long as clinically appropriate and were sedated with continuous infusion of propofol.

Additionally to standard monitoring with a three lead electrocardiogram, a transcutaneous oxygen sensor, a radial arterial and a central venous line, all patients were equipped with a pulmonary artery catheter for continuous determination of pulmonary artery pressures, automated semicontinuous measurement of cardiac output (CO)/cardiac index (CI), and continuous measurement of mixed venous oxygen saturation (SvO_2) (Vigilance®; Edwards Lifescience, Irvine, USA). Arterial blood samples were drawn for determination of oxygen (paO_2) and carbon dioxide ($PaCO_2$) tension, base excess (BE), arterial bicarbonate, and arterial pH (ABL 505 blood gas analyzer, Radiometer, Copenhagen, Denmark), as well as haemoglobin concentration and arterial lactate concentration. Simultaneously, central venous and mixed venous blood samples were drawn for determination of $pcvCO_2$, $ScvO_2$, and $pvCO_2$. $p(cv\text{-}a)CO_2$ and $p(v\text{-}aCO_2)$ were calculated.

A nasogastric tonometry catheter (TRIP NGS catheter, Tonometric Division, Instrumentarium, Helsinki, Finnland) was placed in the stomach. Correct positioning was verified by auscultation over the gastric region, aspiration of gastric contents and radiologically with the next routine chest X-ray. The tonometer was connected to a Tonocap® (Datex, Helsinki, Finnland). The Tonocap® measures gastric luminal pCO_2 ($pgCO_2$) which is in equilibrium with gastric mucosal pCO_2 by automatic gas capnometry. Briefly, the tonometer balloon is automatically filled with approximately 6 mL air and pCO_2 is measured every 10 min in a recirculating mode [9]. The difference of arterial and gastric pCO_2 ($p(g\text{-}a)CO_2$) was calculated which has been shown to reflect gastro-intestinal stagnant hypoxia secondary to hypovolaemia [10,11]. Negative values of $p(g\text{-}a)CO_2$ were set to zero. There was no enteral feeding as long as the tonometric tube was in place. To rule out intra-gastric CO_2 generation following the buffering of acid with bicarbonate patients received 300 mg ranitidine [12].

Statistics

Agreement between $p(cv\text{-}a)CO_2$ and $p(v\text{-}aCO_2)$, and $ScvO_2$ and SvO_2 were analyzed using the method by Bland and Altman [13]. The percentage error was calculated as 1.96 * SD/mean of reference method [14]. Correlations were assessed by calculating Pearson's coefficient (r) and the coefficient of determination (R^2). All data are presented as mean (SD) unless stated otherwise. A p value less than 0.05 was regarded significant.

Results

The demographic and operative data of the studied 25 patients are listed in table 1. The perioperative course of $p(v\text{-}a)CO_2$ and $p(cv\text{-}a)CO_2$ is shown in fig.1. Pooled data of 127 simultaneous measurements could be analyzed. $P(v\text{-}a)CO_2$ and $p(cv\text{-}a)CO_2$ showed a significant correlation with a correlation index of r = 0.600. The Bland-Altman transformation revealed a bias of $p(cv\text{-}a)CO_2$ of 1.2 (3.4) mmHg, LOA of 6.7 mmHg with a percentage error of 152% (fig. 2). SvO_2 and $ScvO_2$ showed a significant correlation as well. Bias of $ScvO_2$ was -0.3 (7.8) % with LOA of 15.3 %. This resulted in a percentage error of 21,9 % (fig. 3).

Correlation analysis between $p(v\text{-}a)CO_2$ and CI, SvO_2, arterial lactate, and $p(g\text{-}a)CO_2$

Gender [female / male]	7 / 18
Age [years]	69 (8)
Height [cm]	171 (7)
Weight [kg]	77 (14)
LVEF [%]	61 (13)
Number of grafts performed 2-ACVG [n] 2-ACVG + IMA [n] 3-ACVG [n] 3-ACVG + IMA [n] 4-ACVG + IMA [n]	 1 8 5 10 1
Duration of surgery [min]	226 (30)
Cross clamp time [min]	45 (20)
Duration of cardiopulmonary bypass [min]	93 (17)
Duration of ventilation [hours]	13 (5)

Table 1: Demographic and perioperative data

LVEF, left ventricular ejection fraction; ACVG, Aorto coronary venous graft; IMA, Internal mammaria artery graft

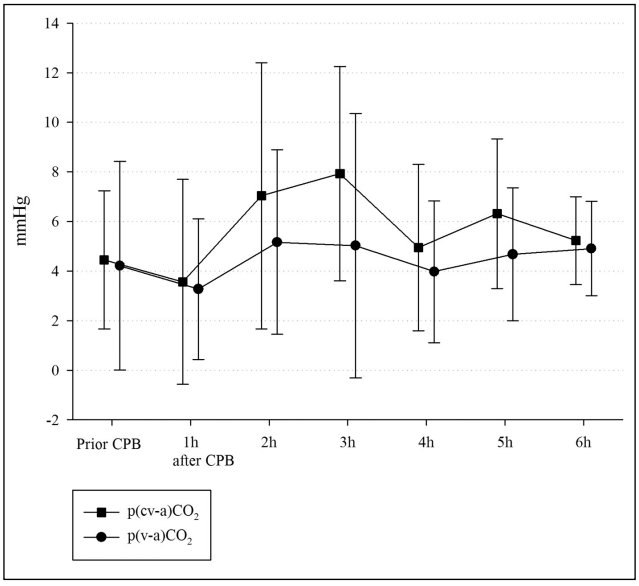

Figure 1: Perioperative course of $p(v\text{-}a)CO_2$ and $p(cv\text{-}a)CO_2$

revealed no meaningful significant correlations (see fig. 4).

Discussion

The main findings of this study are as follows:

- In cardiac surgery patients $p(cv\text{-}a)CO_2$ cannot be used as an estimate of $p(v\text{-}a)CO_2$ with acceptable accuracy.
- There is no reasonable association between $p(v\text{-}a)CO_2$ and CI, SvO_2, or arterial lactate levels, as markers of the adequacy of global tissue oxygenation, and $p(g\text{-}a)CO_2$, as a marker of regional perfusion. Therefore neither $p(v\text{-}a)CO_2$, nor $p(cv\text{-}a)CO_2$ measurement can be recommended to rule out tissue hypoxia in postoperative cardiac surgery patients.

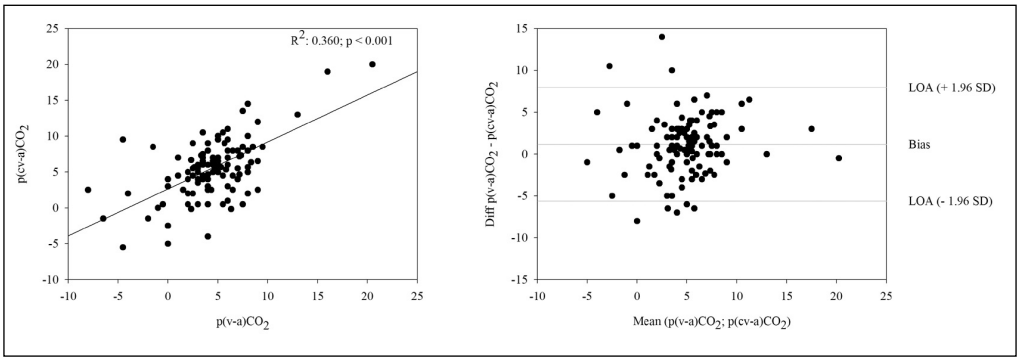

*Figure 2: Correlation and Bland-Altman analyses between p(cv-a)CO_2 or p(v-a)CO_2. p(cv-a)CO_2: Central venous-to-arterial CO_2 difference; p(v-a)CO_2: Venous-to-arterial CO_2 difference; LOA: Limits of agreement (1.96 * SD); p: Significance; R^2: Coefficient of determination (Pearson).*

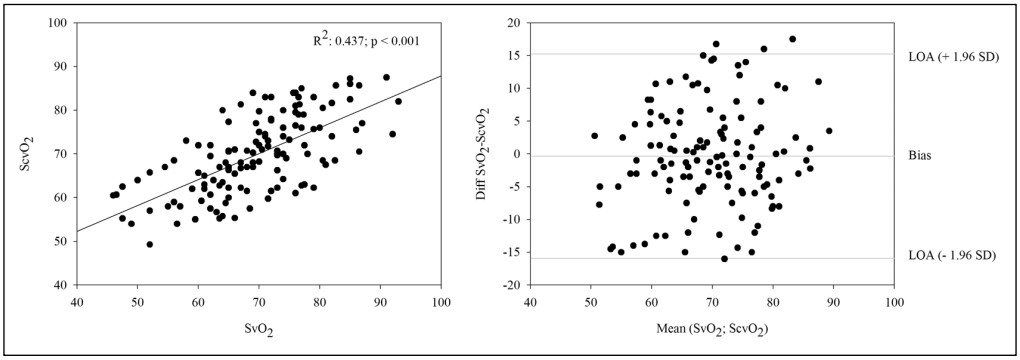

*Figure 3: Correlation and Bland-Altman analyses between $ScvO_2$ and SvO_2. $ScvO_2$: Central venous oxygen saturation; SvO_2: Mixed venous oxygen saturation; LOA: Limits of agreement (1.96 * SD); p: Significance; R^2: Coefficient of determination (Pearson).*

These results are in contrast with some of the existing literature. In a heterogenous group of critically ill patients Cuschieri et al. showed a strong agreement between p(v-a)CO_2 and p(cv-a)CO_2 measurements [15]. In addition, they revealed a high correlation between p(cv-a)CO_2 and CI. There are possible reasons for these different results. Unfortunately, p(cv-a)CO_2 mostly represents venous carbon dioxide from the upper part of the body, even if the tip of the central venous catheter is located near the right atrium. Cerebral CO_2-production is very different between awake and sedated states, not necessarily reflecting different states of cardiac performance [2,16]. In addition, CO_2-production from the lower part of the body might increase very much during reperfusion after hypothermic CPB. This will result in false low CO_2-differences when p(cv-a)CO_2 is measured to estimate p(v-a)CO_2. In our opinion p(cv-a)CO_2 measured as an estimate of p(v-a)CO_2 should be used very cautiously, at least in cardiac surgery patients.

There is clear experimental evidence, that stagnant tissue hypoxia is associated with a widening of the venous-to-arterial pCO_2-difference, depending on the region of interest [17-19]. In the present study no clear relationship between p(v-a)CO_2 and markers of global tissue oxygenation, as SvO_2 and arterial lactate levels, or CI could be demonstrated. One reason could be the inevitable inaccuracy of blood gas analyzers, accounting for up to

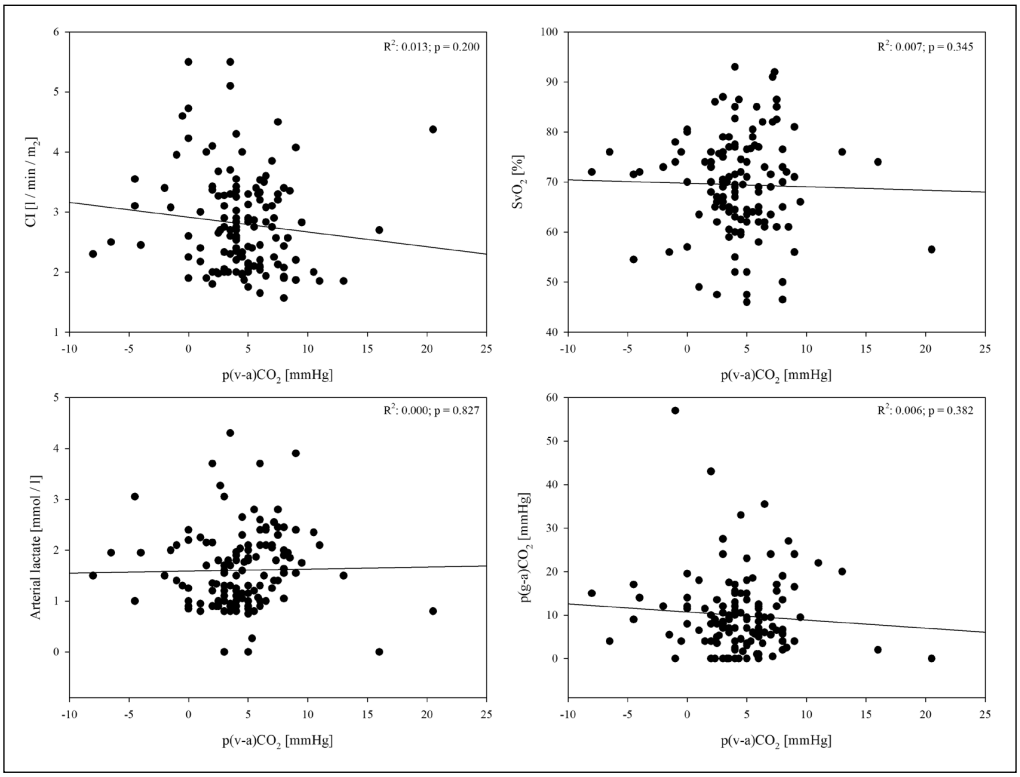

Figure 4: Correlation between p(v-a)CO_2 and CI, SvO_2, arterial lactate levels, and p(g-a)CO_2. p(v-a)CO_2: Venous-to-arterial CO_2 difference; CI: Cardiac index; SvO_2: Mixed venous oxygen saturation; p(g-a)CO_2: Gastric-to-arterial CO_2 difference; p: Significance; R^2: Coefficient of determination (Pearson).

10% bias. In contrast to laboratory studies in clinical investigations much less parameters effecting CO_2-production, eg. exact body temperature, level of sedation, and alveolar ventilation, can be controlled for. Especially body temperature is a very crucial point in postoperative cardiac surgery patients. There is a high interindividual variability in the rate of rewarming of peripheral tissue beds after hypothermic CPB, resulting in a varying degree of CO_2-production. These factors might even be worse in clincial routine, where eg. different blood gas analyzers are used, possibly increasing the inaccuracy.

Vallée et al. demonstrated in patients with septic shock that some degree of tissue hypoxia might be present even with $ScvO_2$ values > 70% [5]. In their study these patients could be identified by a p(cv-a)CO_2 of more than 6 mmHg [5]. Futier et al. reported on patients undergoing major abdominal high risk surgery [6]. 15% of the patients showed a ScvO2 > 71%, but developed major complications. A p(cv-a)CO_2 of more than 5mmHg had the most predictive threshold value in these patients [6]. That patients with a $ScvO_2$ > 70% still have an inadaquacy of regional or global tissue oxygenation may be due to the inevitable limitations of the parameter [20-22]. For example, stagnant tissue hypoxia in the lower part of the body may be unrecognized. While $ScvO_2$ measurements are an established monitoring parameter in septic patients [1], many questions have not been answered for other patient groups [21,23,24]. Sander et al. studied cardiac surgery patients and revealed a reasonable correlation and bias between SvO_2 and $ScvO_2$ measurements, but large limits of aggreement [23]. They conclude, that under certain conditions

these parameters may differ significantly. We confirmed these results with the present study showing a high, significant correlation, and a low bias, but large LOA between the two parameters. Perz et al. demonstrated that in elective cardiac surgery patients a low (< 60 %) and a supranormal (> 77 %) $ScvO_2$ were associated with an unfavourable outcome, while patients with normal values (61-76 %) showed no complications. Values for $p(cv\text{-}a)CO_2$ were not reported [25].

Thus, up to now $ScvO_2$ monitoring should be used very cautiously, especially in cardiac surgery patients.

In the present study no reasonable relationship between $p(v\text{-}a)CO_2$ and lactate or $p(g\text{-}a)CO_2$ could be demonstrated. While arterial lactate is a well accepted routine marker of global tissue oxygenation [26], gastric tonometry has been critizised of being unspecific to tissue hypoxia in critically ill patients [27]. In addition, interventional trials aimed at improving an abnormal gastric mucosal pH, showed no impact on outcome [28]. Therefore, routine use of gastric tonometry is not recommended [29]. Nevertheless, gastric tonometry has been proven to be an early warning sign of low regional oxygen delivery [27,30-32]. As neither lactate nor $p(g\text{-}a)CO_2$ increased in parallel with widening of $p(v\text{-}a)CO_2$, this parameter cannot be recommended for routine use in cardiac surgery patients, until further information about thresholds, sensivity, and timing are elaborated.

There are limitations of the study.

The studied patients group is very small, and the study is a re-analysis of an older study. Although the data were prospectively collected, they were not intended to evaluate the relationship between $p(v\text{-}a)CO_2$ and $p(cv\text{-}a)CO_2$. Therefore no definite conclusions can be drawn.

No patients showed an unfavourable course. As can be seen in the Bland Altman diagram, a wide range of $p(v\text{-}a)CO_2$ and SvO_2 values were observed. But these did not seem to have an effect on outcome.

We conclude that in cardiac surgery patients $p(cv\text{-}a)CO_2$ cannot be used as an estimate of $p(v\text{-}a)CO_2$ with acceptable accuracy. There is no evidence that measurements of $p(cv\text{-}a)CO_2$ or $p(v\text{-}a)CO_2$ could help diagnose global or regional tissue hypoxia in this patient group. Unless more information on the relationship between central-venous parameters with other parameters of tissue oxygenation and especially the therapeutic implications of pathological values of these parameters are known, $p(cv\text{-}a)CO_2$ or $p(v\text{-}a)CO_2$ and $ScvO_2$ should be interpreted very cautiously.

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References

1. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001; 345: 1368-1377
2. Shepherd SJ, Pearse RM. Role of central and mixed venous oxygen saturation measurement in perioperative care. Anesthesiology 2009; 111: 649-656
3. Heinze H, Hanke T, Misfeld M. A mysterious difference between central and mixed venous oxygen saturation after cardiac surgery. Applied Cardiopulmonary Pathophysiology 2008; 12: 20-22
4. Carl M, Alms A, Braun J, Dongas A, Erb J, Goetz A, Goepfert M, Gogarten W, Grosse J, Heller AR, Heringlake M, Kastrup M, Kroener A, Loer SA, Marggraf G, Markewitz A, Reuter D, Schmitt DV, Schirmer U, Wiesenack C, Zwissler B, Spies C. S3 guidelines for intensive care in cardiac surgery patients: hemodynamic monitoring and cardiocirculatory system. Ger Med Sci; 8: Doc12
5. Vallee F, Vallet B, Mathe O, Parraguette J, Mari A, Silva S, Samii K, Fourcade O, Genestal M. Central venous-to-arterial carbon dioxide difference: an additional target for goal-directed therapy in septic shock? Intensive Care Med 2008; 34: 2218-2225

6. Futier E, Robin E, Jabaudon M, Guerin R, Petit A, Bazin JE, Constantin JM, Vallet B. Central venous O_2 saturation and venous-to-arterial CO_2 difference as complementary tools for goal-directed therapy during high-risk surgery. Crit Care; 14: R193
7. Misfeld M, Heinze H, Sievers HH, Kraatz EG. Intramyocardial oxygen monitoring in coronary artery bypass surgery. J Clin Monit Comput 2004; 18: 93-101
8. Kraatz E, Uhlig T, Heinze H. The connection between myocardial oxygen partial pressure and intragastric CO2 – three cases. Anästhesiol Intensivmed Notfallmed Schmerzther 1998; 33 (Suppl 2): S106-108
9. Creteur J, De Backer D, Vincent JL. Monitoring gastric mucosal carbon dioxide pressure using gas tonometry: in vitro and in vivo validation studies. Anesthesiology 1997; 87: 504-510
10. Chapman MV, Mythen MG, Webb AR, Vincent JL. Report from the meeting: Gastrointestinal Tonometry: State of the Art. Intensive Care Med 2000; 26: 613-622
11. Schlichtig R, Mehta N, Gayowski TJ. Tissue-arterial PCO2 difference is a better marker of ischemia than intramural pH (pHi) or arterial pH-pHi difference. J Crit Care 1996; 11: 51-56
12. Bams JL, Kolkman JJ, Roukens MP, Douma DP, Loef BG, Meuwissen SG, Groeneveld AB. Reliable gastric tonometry after coronary artery surgery: need for acid secretion suppression despite transient failure of acid secretion. Intensive Care Med 1998; 24: 1139-1143
13. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986; 1: 307-310
14. Critchley LA, Critchley JA. A meta-analysis of studies using bias and precision statistics to compare cardiac output measurement techniques. J Clin Monit Comput 1999; 15: 85-91
15. Cuschieri J, Rivers EP, Donnino MW, Katilius M, Jacobsen G, Nguyen HB, Pamukov N, Horst HM. Central venous-arterial carbon dioxide difference as an indicator of cardiac index. Intensive Care Med 2005; 31: 818-822
16. Pearse R, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennett ED. Changes in central venous saturation after major surgery, and association with outcome. Crit Care 2005; 9: R694-699
17. Vallet B, Teboul JL, Cain S, Curtis S. Venoarterial CO(2) difference during regional ischemic or hypoxic hypoxia. J Appl Physiol 2000; 89: 1317-1321
18. Neviere R, Chagnon JL, Teboul JL, Vallet B, Wattel F. Small intestine intramucosal PCO(2) and microvascular blood flow during hypoxic and ischemic hypoxia. Crit Care Med 2002; 30: 379-384
19. Groeneveld AB. Interpreting the venous-arterial PCO2 difference. Crit Care Med 1998; 26: 979-980
20. Dueck MH, Klimek M, Appenrodt S, Weigand C, Boerner U. Trends but not individual values of central venous oxygen saturation agree with mixed venous oxygen saturation during varying hemodynamic conditions. Anesthesiology 2005; 103: 249-257
21. Varpula M, Karlsson S, Ruokonen E, Pettila V. Mixed venous oxygen saturation cannot be estimated by central venous oxygen saturation in septic shock. Intensive Care Med 2006
22. Marx G, Reinhart K. Venous oximetry. Curr Opin Crit Care 2006; 12: 263-268
23. Sander M, Spies CD, Foer A, Weymann L, Braun J, Volk T, Grubitzsch H, von Heymann C. Agreement of central venous saturation and mixed venous saturation in cardiac surgery patients. Intensive Care Med 2007
24. Pearse RM, Hinds CJ. Should we use central venous saturation to guide management in high-risk surgical patients? Crit Care 2006; 10: 181
25. Perz S, Uhlig T, Kohl M, Bredle DL, Reinhart K, Bauer M, Kortgen A. Low and „supranormal" central venous oxygen saturation and markers of tissue hypoxia in cardiac surgery patients: a prospective observational study. Intensive Care Med 37: 52-59
26. Bakker J. Lactate: May I have your votes please? Intensive Care Med 2001; 27: 6-11
27. Hamilton MA, Mythen MG. Gastric tonometry: where do we stand? Curr Opin Crit Care 2001; 7: 122-127
28. Gomersall CD, Joynt GM, Freebairn RC, Hung V, Buckley TA, Oh TE. Resuscitation of critically ill patients based on the results of gastric tonometry: a prospective, randomized, controlled trial. Crit Care Med 2000; 28: 607-614

29. Antonelli M, Levy M, Andrews PJ, Chastre J, Hudson LD, Manthous C, Meduri GU, Moreno RP, Putensen C, Stewart T, Torres A. Hemodynamic monitoring in shock and implications for management. International Consensus Conference, Paris, France, 27-28 April 2006. Intensive Care Med 2007; 33: 575-590
30. Kolkman JJ, Otte JA, Groeneveld AB. Gastrointestinal luminal PCO2 tonometry: an update on physiology, methodology and clinical applications. Br J Anaesth 2000; 84: 74-86
31. Mythen MG, Webb AR. Gastrointestinal tonometry comes of age? Br J Anaesth 1998; 81: 667-668
32. Marik P. Gastric tonometry: the canary sings once again. Crit Care Med 1998; 26: 809-810

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