# Assessment of Moderate to Severe Abdominal Blood Loss Using Peripheral to Central Blood Oxygen Saturation

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# ABSTRACT

**Purpose:** There are various definitions and monitoring modalities for hemodynamic status. Each of them has its own advantages and shortcomings. A new hemodynamic index is proposed in this study. This index can be calculated by placing the measured hemoglobin saturation in a formula. Blood samples for this measurement are taken from arterial, antecubital and central venous blood.

**Material and Methods:** We calculated this index in three different groups undergoing laparatomy. The control group consisted of patients who underwent elective surgery. The case group with acute internal abdominal bleeding was divided into two groups. Those with more than 20 ml/kg of blood in their abdominal cavity were designated as the severe case group, while those bleeding less were categorized as moderate. Blood samples were taken from ten patients in each group in stable and unstable conditions.

**Results:** This index differed significantly between dissimilar hemodynamic conditions. The pre-anesthesia value of this index in the control group showed a mean  $\pm$  SD of  $8.5 \pm 3.2$  vs.  $1.6 \pm 0.4$  in the moderate case group vs.  $0.7 \pm 0.08$  in the case group with severe hemodynamic changes (p < .001). The index approximated to the control values as the circumstances improved. After compensation for volume loss, pre-extubation values were not significantly different. These were  $9.6 \pm 2$  in the control group vs.  $8 \pm 2$  in the case group with moderate hemodynamic change vs.  $8 \pm 1.8$  in the severe case group. The likelihood ratio of bleeding increased as this index decreased.

**Conclusion:** As the hemodynamic condition deteriorates, this index decreases significantly. This index is an accurate indicator for predicting hemodynamic changes compared to some other modalities. Further investigations are needed into the prognostic and therapeutic advantages of this index.

Key words: hemodynamics; blood gas analysis; surgical blood loss; patient monitoring

## INTRODUCTION

Hemodynamic status may be defined in various ways. Therefore, different modalities have been used for its monitoring. Each of these practices has some advantages, but does not provide comprehensive information.

Heart rate, cardiac output, arterial or venous blood pressure may reflect hemodynamic status, if the blood flow is considered as the goal. However, these findings are unable to represent the adequacy of the blood flow for tissue oxygenation [1-6].

Mixed venous hemoglobin saturation can provide valuable information about the balance of oxygen delivery and

consumption. The taking of samples with pulmonary artery catheterization and the need to have a base value are among the shortcomings of this modality [7-18].

Various methods use regional venous hemoglobin saturation or tissue oxygenation as a goal for hemodynamic sufficiency. These procedures do not provide enough data regarding the general condition of the body [19-37].

In this study, we define an index to determine the hemodynamic status of our patients.

Adolph Fick described an equation to determine blood flow by measuring overall oxygen uptake and content in the blood:

	Control group $n = 10$	Case group with Moderate change $n = 10$	Case group with Severe change $n = 10$	p value
Age (years)	$38 \pm 6.6$	41± 5.9	$39 \pm 6.3$	ns
BMI	$25 \pm 1.5$	$25\pm0.87$	$25.2 \pm 1$	ns
Gender (male/female)	4	4	4	ns

#### Table 1. Demographic characteristics of patients.

Figures are mean ± SD or frequencies. Means are compared with one-way ANOVA analysis.

Abbreviations; n = number of patients, ns = not statistically significant

$$Q' = \frac{\dot{V}o2}{(Cao2 - Cvo2) \times 10}$$

Q' = cardiac output (L/min)

Vo2 = oxygen consumption (mL O2/min)

Cao2 = oxygen content of arterial blood (mL O2/100 mL blood)

Cvo2 = oxygen content of mixed venous blood (mLO2/100 mL blood)

The same equation is true for an extremity such as a forearm or the central part of the body. Dividing the formulas and ignoring the dissolved portion of oxygen in the blood, we arrive at the following equation:

(Qhand	VVhando2) _ Sao2 - Scentalvo2		
(Qcentral	//Vcentralo2) Sao2 - Shandvo2		
Qhand	= forearm perfusion		
Vhando2	= forearm oxygen consumption		
Qcentral	= central perfusion		
Vcentralo2	= central oxygen consumption		
Sao2	= arterial hemoglobin saturation		
Scentralvo2	= central venous hemoglobin saturation		
Shandvo2	= forearm venous hemoglobin saturation		

The value given by the above formula is denoted as the "Hemodynamic Index" in this paper.

We selected patients undergoing laparatomy and classified them according to the amount of blood measured in their abdominal cavity. Then we compared this new index and some other known modalities in these groups before and after resuscitation.

We aimed to show whether or not this index was an accurate quantitative indicator of the severity of blood loss. In addition, we wanted to know if this index improved after volume compensation compared to other modalities.

## MATERIAL AND METHODS

We selected patients undergoing laparatomy. Some of them were candidates for elective surgery with no abdominal bleeding, who were considered as the control group. Others were admitted for acute internal abdominal bleeding. Only patients who gave informed consent after obtaining institutional review board approval were included. We excluded conditions that could interfere with the results (the flowchart illustrates the selection process).

We took radial artery, antecubital and central venous blood samples before resuscitation and anesthesia. These samples were used to find the initial hemodynamic index.

Patients with abdominal bleeding were divided into two groups according to the amount of blood in their abdominal cavity.

We defined the fluid treatment goal for the elective group as 10 cc/kg/h of crystalloid solution infusion. For emergency cases, we aimed to reach a systolic blood pressure of more than 100 mmHg with infusion of crystalloids and compensating the blood deficit of the severe case group with one third of initial blood loss with packed red blood cells. Those in the moderate case or control group who needed blood transfusion or any patients requiring vasopressors were excluded from the study. Similar blood samples were taken before the reversal of anesthesia to calculate the final hemodynamic index.

We utilized NCSS 2000 statistical software to find the sample size. According to a pilot study and applying one-way ANOVA power analysis, 10 patients in each group (control, case group with moderate hemodynamic change and case group with severe hemodynamic change) were needed.

We conducted this study in two trauma centers from January 2006 until January 2007.

We used SPSS 15 as our statistical software for analyzing the data. Comparison among means is performed with oneway ANOVA and the post hoc test for difference confirmation. To determine the sensitivity and specificity of this test, we used receiver-operating characteristics (ROC curve) and plotted the likelihood ratio curve of the index.

#### RESULTS

The demographic and hemodynamic characteristics of patients are given in Tables 1, 2 and 3.

There was no significant difference between the groups in terms of age, body mass index and gender (*Tab. 1*).

Pre-anesthesia hemodynamic characteristics including systolic blood pressure, respiratory rate, hemodynamic index, central venous oxygen saturation and hemoglobin level showed significant differentiation between groups (*Tab.* 2).

Post-resuscitation data showed significant differences in hemoglobin level and central venous oxygen saturation (*Tab. 3*).



	Control group (1) n=10	Case Group with Moderate change (2) n=10	Case Group with Severe change (3) n=10	p value	post-hoc test evaluation
Initial Systolic Blood Pressure (mmHg)	$126 \pm 8.8$	$122 \pm 12.7$	110 ± 7.8	0.003	1 # 3 – 2 # 3
Initial Heart Rate	$94 \pm 11$	93 ± 6	$104 \pm 1$	ns	
Initial Respiratory Rate	11 ± 1.4	$14 \pm 2$	19 ± 3.6	<.001	1 # 2 # 3
Initial Temperature (°C)	$37 \pm 0.18$	$37 \pm 0.18$	$37 \pm 0.17$	ns	
Initial Hemodynamic Index	8.5 ± 3.2	$1.6 \pm 0.4$	$0.7 \pm 0.08$	<.001	1 # 2 # 3
Initial Central Venous Pressure (cm H <sub>2</sub> O)	6.3 ± 1.33	6.2 ± 1.2	4.8 ± 1.2	ns	
Initial Central Venous Saturation (%)	85 ± 4	75 ± 5	71± 3	<.001	1 # 2 - 1 # 3
Initial Hemoglobin level (g/dL)	$12.3 \pm 0.8$	9.6 ± 1	$7 \pm 1$	<.001	1 # 2 # 3

Table 2. Pre-anesthesia hemodynamic characteristics of patients.

Figures are mean  $\pm$  SD. Means are compared with one-way ANOVA analysis. Significant differences between groups evaluated with post-hoc test are depicted by #.

Abbreviations; n = number of patients, ns = not statistically significant

Table 3. Post resuscitation hemodynamic characteristics of patients.

	Control group (1) n = 10	Case group with Moderate change (2) n = 10	Case group with Severe change (3) n = 10	p value	post-hoc test evaluation
Final Hemodynamic Index	$9.6 \pm 2$	$8 \pm 2$	$8 \pm 1.8$	ns	
Final Central Venous Pressure (cm $H_2O$ )	$6.6\pm0.96$	$7.2 \pm 1.26$	$7.6 \pm 1.57$	ns	
Final Central Venous Saturation (%)	$82\pm5$	$82 \pm 3$	78 ± 2	<.05	1 # 3 - 2 # 3
Final Hemoglobin level (g/dl)	$11.7\pm0.9$	$9 \pm 0.7$	8 ± 1.2	<.001	1 # 2 - 1 # 3
Blood Loss (cc)	0	$665 \pm 200$	$2000\pm440$	<.001	1 # 2 # 3

Figures are mean  $\pm$  SD. Means are compared with one-way ANOVA analysis. Significant differences between groups evaluated with post-hoc test are depicted by #.

Abbreviations; n = number of patients, ns = not statistically significant

Figure 1 and 2 illustrate hemodynamic index error bars in stable and unstable conditions.

We used receiver-operating characteristics (ROC curve) and constructed a final diagram (*Fig. 3*) to show the likelihood (sensitivity/1-specificity) of abdominal bleeding according to the hemodynamic index.

## DISCUSSION

The ability to identify the hemodynamic condition of a patient would be of great value. Therapeutic and prognostic approaches can be based on this finding. Judgments in this area require a comprehensive understanding of different aspects of hemodynamic status. A physician can predict the mechanical properties of circulation by considering cardiovascular monitoring values such as arterial or venous blood pressures, echocardigraphic indices and heart rate. However, some of these modalities are expensive, invasive or difficult to perform, and besides they cannot reflect oxygenation of the tissues [1-6].

Venous hemoglobin saturation, which can be carried out on a pulmonary artery blood sample (mixed venous) to indicate the general condition of the body [7- 18] or on jugular venous samples to show brain circulation are important modalities. However they still have shortcomings, such as their being invasive and requiring a patient-dependent basic value for further comparisons. Central venous oxygen saturation can also be used for this purpose [19-37].

Physiologic reduction of peripheral perfusion and maintenance of vital organ circulation (brain and heart) is the basis for this study. In a normal person, peripheral venous hemoglobin saturation has a value between the arterial and central venous hemoglobin saturations. This figure will become closer to or even lower than the central samples as the hemodynamic status deteriorates. We intended to find an index that was easy to determine, inexpensive and indicative

Figure 1. Pre-anesthesia hemodynamic index error bar plot.



Abbreviation: SE= Standard Error

*Figure 3.* Likelihood ratio (sensitivity/1-specificity) of abdominal bleeding according to the hemodynamic index values and patient group.



of the severity of hemodynamic status. With this is mind, we rearranged the Fick equation and compared two parts of the body with each other.

Laboratory errors may be attenuated by this means as samples from different parts of the same patient are examined using an identical instrument.

This research could demonstrate that the proposed index differs significantly between dissimilar hemodynamic conditions and will approximate to the control values as the circumstances are corrected.

Our study also showed that this index accurately indicated the severity of abdominal bleeding. There is no need for a base value.

Figure 2. Post resuscitation hemodynamic index error bar plot.



Abbreviation: SE= Standard Error

Central venous oxygen saturation showed significant differentiation in unstable conditions, but did not approximate to the control values after compensation. This finding may be due to differences in its base value, the need for more time for correction, or its inaccuracy for response to this therapy. Hemoglobin level has a trend similar to central venous oxygen saturation.

At the end of anesthesia, our patients were in their most stable hemodynamic condition. The effects of our drugs were minimal; they were resuscitated and were not under stress. Therefore, we decided to take final samples at this stage.

This index may be used to predict the severity of hemodynamic status. Its could be usefully applied in other conditions where the volume of blood loss is not measurable (such as pelvic fracture or massive external bleedings). Young patients do not usually show exaggerated changes in blood pressure or heart rate even in conditions where they have lost a great amount of their blood volume. This index may be an accurate modality for such circumstances.

Many confounding factors were eliminated by the inclusion and exclusion criteria. Therefore the results of this paper cannot be applied to patients in general until appropriate studies confirm the validity of this index.

Further research is needed to prove whether there is a linear correlation between this index and the severity of hemodynamic changes. In addition, supplementary investigations may demonstrate the prognostic or therapeutic value of this index.

# CONCLUSION

We rearranged the Fick equation and arrived at the following formula:

_(Qhand/Vha ndo2)	Sao2 - Scentalvo2		
(Qcentral/Vcentralo2)	Sao2 - Shandvo2		

Samples were taken from radial artery, antecubital and central veins.

The value given by the above formula was denoted the "hemodynamic index".

We selected 30 patients in three different groups of hemodynamic conditions. This index differed significantly in these groups.

The index was an accurate indicator of the severity of abdominal blood loss. Receiver operating characteristics depicted high sensitivity and specificity for this index as bleeding exaggerated. This finding was illustrated as an increasing likelihood ratio of bleeding as the index decreased. In addition, it approximated to control values after our therapy, whereas central venous oxygen saturation and hemoglobin level did not.

The results given in this paper cannot yet be generalized until their validity is confirmed by other studies.

#### REFERENCES

1. Lazor MA, Pierce ET, Stanley GD, Cass JL, Halpern EF, Bode RH, Jr. Evaluation of the accuracy and response time of STAT-mode continuous cardiac output. J Cardiothorac Vasc Anesth. 1997;11(4):432-6.

2. Shoemaker W, Zelman W, Wo C, Chen D, Kamel E, Chen L, Ramicone E, Berlzberg H, Thangaturia D, Lamb P. [Prognosis of severe trauma outcome using noninvasive monitoring]. Anesteziol Reanimatol. 2003;(6):8-13.

3. Shoemaker WC, Thangathurai D, Wo CC, Kuchta K, Canas M, Sullivan MJ, Farlo J, Roffey P, Zellman V, Katz RL. Intraoperative evaluation of tissue perfusion in highrisk patients by invasive and noninvasive hemodynamic monitoring. Crit Care Med. 1999;27(10):2147-52.

4. Shoemaker WC, Wo CC, Chan L, Ramicone E, Kamel ES, Velmahos GC, Belzberg H. Outcome prediction of emergency patients by noninvasive hemodynamic monitoring. Chest. 2001;120(2):528-37.

5. Vignon P. Hemodynamic assessment of critically ill patients using echocardiography Doppler. Curr Opin Crit Care. 2005;11(3): 227-34.

6. Bellomo R, Uchino S. Cardiovascular monitoring tools: use and misuse. Curr Opin Crit Care. 2003;9(3):225-9.

7. Buheitel G, Scharf J, Hofbeck M, Singer H. Estimation of cardiac index by means of the arterial and the mixed venous oxygen content and pulmonary oxygen uptake determination in the early post-operative period following surgery of congenital heart disease. Intensive Care Med. 1994;20(7):500-3.

8. Gothgen I. Heat-induced changes in PO2 and PCO2 of blood. Acta Anaesthesiol Scand. 1984;28(4):447-51.

9. Kazarian KK, Sharma P, Caron N, Arias J, Del Guercio LR. Significance of mixed venous oxygen reserve in traumatic shock. Surg Forum. 1977;28:16-9.

 Kazarian KK, Del Guercio LR. The use of mixed venous blood gas determinations in traumatic shock. Ann Emerg Med. 1980;9(4):179-82.

11. Kirubakaran C, Gnananayagam JE, Sundaravalli EK. Comparison of blood gas values in arterial and venous blood. Indian J Pediatr. 2003;70(10):781-5.

12. Krantz T, Warberg J, Secher NH. Venous oxygen saturation during normovolaemic haemodilution in the pig. Acta Anaesthesiol Scand. 2005;49(8):1149-56.

13. Marx G, Reinhart K. Venous oximetry. Curr Opin Crit Care. 2006 Jun;12(3):263-8.

14. Noll ML, Byers JF. Usefulness of measures of Svo2, Spo2, vital signs, and derived dual oximetry parameters as indicators of arterial blood gas variables during weaning of cardiac surgery patients from mechanical ventilation. Heart Lung. 1995;24(3):220-7.

15. O'Connor TA, Hall RT. Mixed venous oxygenation in critically ill neonates. Crit Care Med. 1994;22(2):343-6.

16. Reinhart K, Bloos F. The value of venous oximetry. Curr Opin Crit Care. 2005;11(3):259-63.

17. Siggaard-Andersen O, Fogh-Andersen N, Gothgen IH, Larsen VH. Oxygen status of arterial and mixed venous blood. Crit Care Med. 1995;23(7):1284-93.

18. Siggaard-Andersen O, Gothgen IH. Oxygen and acid-base parameters of arterial and mixed venous blood, relevant versus redundant. Acta Anaesthesiol Scand Suppl. 1995;107:21-7.

19. Bloos F, Reinhart K. [The value of central venous O(2) saturation for assessment of tissue oxygenation] Dtsch Med Wochenschr. 2004;129(48):2601-4.

20. Gopinath SP, Cormio M, Ziegler J, Raty S, Valadka A, Robertson CS. Intraoperative jugular desaturation during surgery for traumatic intracranial hematomas. Anesth Analg. 1996;83(5):1014-21.

21. Gopinath SP, Valadka AB, Uzura M, Robertson CS. Comparison of jugular venous oxygen saturation and brain tissue Po2 as monitors of cerebral ischemia after head injury. Crit Care Med. 1999;27(11):2337-45.

22. Howard L, Gopinath SP, Uzura M, Valadka A, Robertson CS. Evaluation of a new fiberoptic catheter for monitoring jugular venous oxygen saturation. Neurosurgery. 1999;44(6):1280-5.

23. Kadoi Y, Saito S, Kawahara F, Goto F, Owada R, Fujita N. Jugular venous bulb oxygen saturation in patients with preexisting diabetes mellitus or stroke during normothermic cardiopulmonary bypass. Anesthesiology. 2000;92(5):1324-9.

24. Kadoi Y, Saito S, Goto F, Fujita N. Decrease in jugular venous oxygen saturation during normothermic cardiopulmonary bypass predicts short-term postoperative

neurologic dysfunction in elderly patients. J Am Coll Cardiol. 2001;38(5):1450-5.

25. Kadoi Y, Saito S, Goto F, Someya T, Kamiyashiki S, Fujita N. Time course of changes in jugular venous oxygen saturation during hypothermic or normothermic cardiopulmonary bypass in patients with diabetes mellitus. Acta Anaesthesiol Scand. 2001;45(7):858-62.

26. Kadoi Y, Saito S, Yoshikawa D, Goto F, Fujita N, Kunimoto F. Increasing mean arterial blood pressure has no effect on jugular venous oxygen saturation in insulin-dependent patients during tepid cardiopulmonary bypass. Anesth Analg. 2002;95(2):266-72.

27. Kadoi Y, Fujita N Increasing mean arterial pressure improves jugular venous oxygen saturation in patients with and without preexisting stroke during normothermic cardiopulmonary bypass. J.Clin.Anesth. 2003; 15: 339-44.

28. Kadoi Y, Saito S, Goto F, Fujita N. The effect of diabetes on the interrelationship between jugular venous oxygen saturation responsiveness to phenylephrine infusion and cerebrovascular carbon dioxide reactivity. Anesth Analg. 2004;99(2):325-31.

29. Kadoi Y, Saito S, Takahashi K, Fujita N, Goto F. Jugular venous oxygen saturation during mild hypothermic versus normothermic cardiopulmonary bypass in elderly patients. Surg Today. 2004;34(5):399-404.

30. Kim MB, Ward DS, Cartwright CR, Kolano J, Chlebowski S, Henson LC. Estimation of jugular venous O2 saturation from cerebral oximetry or arterial O2 saturation during isocapnic hypoxia. J Clin Monit Comput. 2000;16(3):191-9.

31. Madsen P, Olesen HL, Klokker M, Secher NH. Peripheral venous oxygen saturation during head-up tilt induced hypovolaemic shock in humans. Scand J Clin Lab Invest. 1993;53(4):411-6.

32. Miyoshi S, Morita T, Kadoi Y, Goto F. Analysis of the factors related to a decrease in jugular venous oxygen saturation in patients with diabetes mellitus during normothermic cardiopulmonary bypass. Surg Today. 2005;35(7):530-4.

33. Okano N, Owada R, Fujita N, Kadoi Y, Saito S, Goto F. Cerebral oxygenation is better during mild hypothermic than normothermic cardiopulmonary bypass. Can J Anaesth. 2000;47(2):131-6.

34. Robertson CS, Gopinath SP, Goodman JC, Contant CF, Valadka AB, Narayan RK. SjvO2 monitoring in headinjured patients. J Neurotrauma. 1995;12(5):891-6.

35. Sheinberg M, Kanter MJ, Robertson CS, Contant CF, Narayan RK, Grossman RG. Continuous monitoring of jugular venous oxygen saturation in head-injured patients. J Neurosurg. 1992;76(2):212-7.

36. Sutton RN, Wilson RF, Walt AJ. Differences in acidbase levels and oxygen-saturation between central venous and arterial blood. Lancet. 1967;2(7519):748-51.

37. Valadka AB, Gopinath SP, Contant CF, Uzura M, Robertson CS. Relationship of brain tissue PO2 to outcome after severe head injury. Crit Care Med. 1998;26(9):1576-81.