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Central Venous Oxygen Saturation: A Useful Clinical Tool in Trauma Patients

THOMAS M. SCALEA, M.D., RONALD W. HARTNETT, M.D., ALBERT O. DUNCAN, M.D., NABIL A. ATWEH, M.D., THOMAS F. PHILLIPS, M.D., SALVATORE J. A. SCLAFANI, M.D., MICHELE FUORTES, M.D., AND GERALD W. SHAFTAN, M.D.

An accurate method of estimating acute blood loss is essential in the evaluation of injured patients. Central venous oxygen (CVO_2) saturation has been shown to be a sensitive and reliable correlate of blood loss in an animal model but its clinical validity is unproven.

We evaluated 26 consecutive patients with an injury mechanism suggesting blood loss but who were deemed stable after initial evaluation. Vital signs (pulse, blood pressure, pulse pressure, urine output, CVP) and CVO_2 saturation were serially measured. Blood loss was estimated by direct intracavitary collection or serial hematocrits and acute transfusion requirements.

Despite stable vital signs, ten patients (39%) had CVO_2 saturations under 65%. These patients had more serious injuries, significantly larger estimated blood losses, and required more transfusions than those patients with CVO_2 saturation greater than 65%. Linear regression analysis demonstrated the superiority of CVO_2 saturation to predict blood loss with a p value <0.005 relative to any of the normally followed parameters.

CVO_2 saturation is a reliable and sensitive method for detecting blood loss. It is a useful tool in the evaluation of acutely injured patients.

Accurate and relatively simple monitoring is essential when managing acutely injured patients. This becomes essential when substantial occult blood loss is possible. The Advanced Trauma Life Support (ATLS) course teaches that tachycardia and a narrowed pulse pressure are the earliest reliable parameters that change following acute blood loss (1). We have demonstrated that central venous oxygen (CVO_2) saturation is a more reliable and accurate measure of blood loss in an animal model (2). Recently, we began using central venous oxygen saturation as one of our parameters to help to gauge the amount of blood loss in the evaluation of injured patients. We have now reviewed our results in the first 26 patients to see if central venous oxygen saturation is as sensitive and reliable in the clinical setting as it was in the laboratory.

METHODS

All patients were evaluated in the major trauma receiving area of our Emergency Department. Their initial assessment and management was carried out according to standard ATLS guidelines as was deemed appropriate for their mechanism of injury. All patients had two large-bore peripheral intravenous lines established and crystalloid resuscitation begun. Initial vital signs (pulse, blood pressure, pulse pressure, respiratory rate) were measured and recorded as per our normal ED procedure.

Patients who were deemed stable after their initial assessment and first measurement of vital signs but had sustained a mechanism of injury consistent with the possibility of significant blood loss then had central venous pressure (CVP) lines inserted. Line placement was verified by chest X-ray performed immediately after insertion. These lines were placed as early as possible in the resuscitation and ongoing evaluation. Arterial and central venous blood gases were initially measured and then were repeated according to clinical need.

Blood pressure (BP), pulse rate (HR), pulse pressure (PP), central venous pressure (CVP), and respiratory rates (RR) were serially measured every 20 minutes. Urine was collected over the first hour of hospital admission. Blood loss (EBL) was estimated by direct intracavitary collection when possible, i.e., chest tube drainage in the case of penetrating chest trauma or initial suction canister aspiration of free intra-abdominal blood at laparotomy in the case of abdominal trauma. When direct measurement of blood loss was not possible, blood loss was estimated by serial hematocrits and/or transfusion requirements over the first 6 hours of hospitalization. Patients were transfused according to clinical need and/or to maintain hematocrit at 35 mg%.

Results were tabulated and linear regression analysis was utilized to correlate EBL versus both initial and repeat clinical parameters as well as central venous oxygen saturation.

From the Surgical ICU, Kings County Hospital, and the Division of Surgical Critical Care, Departments of Surgery and Radiology, SUNY, Health Sciences Center at Brooklyn, Brooklyn, New York.

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Address for reprints: Thomas M. Scalea, M.D., Department of Surgery, Box 40, 450 Clarkson Avenue, Brooklyn, NY 11203.

RESULTS

Twenty-six patients were treated during the period of March 1 to June 1, 1987. There were 19 males and seven females. The mean age was 26.1 years (range, 14–46 years). No patient gave a history of any chronic medical problems. Twenty-one patients were victims of penetrating trauma. Ten patients sustained single stab wounds, two sustained multiple stab wounds, eight suffered single gunshot wounds, and one suffered multiple gunshot wounds. Of the five patients with blunt mechanisms of injury, two were in pedestrian-motor vehicle accidents, one was in a motor-vehicle accident and two had fallen from a height.

Mean time from injury to initial E.D. arrival was 28 min (range, 10–90 min), as estimated by the Emergency Medical Technicians in the 24 patients who arrived by ambulance and by the patients in the two cases who walked into the ED.

The Emergency Medical Technicians estimated blood loss at the scene. Their estimates ranged from 0 to 2 units with a mean of approximately 200 cc. This was not included in our estimates of blood loss. Twelve patients had IV lines placed in the field and they received an average of 350 cc of crystalloid (range, 100–1,000 cc) before arrival at the hospital.

Of the 24 patients who were transported by ambulance, mean systolic blood pressure in the field was 110 mm Hg (range, 60–160 mm Hg) and mean heart rate was 102 beats per minute (range, 78–140 beats per minute). Pneumatic antishock garments were inflated in six patients. Their mean systolic blood pressure was 80 mm Hg (range, 60–100 mm Hg) and their mean pulse was 110 beats per minute (range, 82–140 beats per minute). Upon presentation to the Emergency Department, mean systolic blood pressure was 122 mm Hg (range, 95–170 mm Hg) and mean pulse rate was 100 beats per minute (range, 74–120 beats per minute). Mean initial systolic blood pressure of those patients in the PASG was 110 mm Hg (range, 95–160 mm Hg) and mean pulse rate was 110 beats per minute (range, 80–120 beats per minute).

The mean time from ED admission to central line placement and central venous oxygenation saturation measurement averaged 24 minutes (range, 5–45 minutes). Choice of CVP site was at the discretion of the resident placing the line. Twenty-two CVP lines were placed via a right internal jugular approach, two via a right subclavian approach, and two via a left subclavian approach. An attempt at line placement was defined as a single pass of the needle. Nineteen CVP lines were successfully placed in one attempt, five required two attempts, and one required three attempts. One patient required four attempts and had a left subclavian line inserted after three attempts at a left internal jugular approach. No other patients required a change in the site of their central access. The tip of all of the CVP lines was in either the superior vena cave or right atrium on the initial chest X-ray after lines placement. There were no complications from CVP placement.

No patient had arterial hypoxia. All 24 patients transported by ambulance were treated with supplemental oxygen. Mean pO_2 was 110 (range, 85–160) and saturations varied between 95% and 99%. Likewise, no patient had metabolic acidosis on initial presentation. Arterial pH varied between 7.38 and 7.45. Base excess was between −3 and 2.

Despite stable vital signs, ten patients (39%) had a CVO_2 saturation under 65%. Their mean CVO_2 saturation was 56% (range, 42%–64%). Five of these patients had blood loss estimated by direct intracavitary collection and the other five had blood loss estimated by serial hematocrits and transfusion requirements. The injuries identified are tabulated in Table I. The mean estimated blood loss in this group of patients was 1,100 cc (range, 800–1,600 cc). Nine of these ten patients required transfusion within the first 12 hours of admission and all ultimately required blood transfusion. The mean transfusion requirement was 8.4 units (range, 6–12 units) within the first 24 hours of admission.

Seven of ten patients with initial venous desaturation had venous gas measurements repeated. Three of them had persistent venous desaturation and the other four had resolved their desaturation. The three who had persistence of their low venous saturations had a mean EBL of 1,250 cc. The four in whom the venous desaturation resolved had less blood loss with a mean EBL of 920 cc. Three patients did not have repeat CVO_2 determinations as they required emergent laparotomies and were anesthetized at the time their venous gas was to be repeated. They all remained hemodynamically stable until the time of their operation.

Sixteen patients had initial CVO_2 saturations greater than 65%. Their mean CVO_2 saturation was 73.4% (range, 66–81%). Eleven patients had blood loss estimated by direct intracavitary collection and five patients had blood loss estimated by serial hematocrits and transfusion requirements. Injuries for this group of patients are also tabulated in Table I. It is interesting to note that four of the 16 patients (25%) had no injury of

TABLE I
Injuries

	$CVO_2 < 0.65$ (10 pts)	$CVO_2 > 0.65$ (16 pts)
HTX > 800 cc	4	0
HTX < 800 cc	0	5
Mult. fractures	2	0
Pelvic fractures	2	0
Major vascular	2	0
Kidney/liver/spleen	1	2
Bowel	1	4
Heart	0	2
No injury	0	4

significance identified despite a mechanism suggesting the possibility of blood loss.

No patient of the 16 suffered a significant vascular injury as opposed to the two patients with low CVO_2 saturation who had major vascular injuries. Of the five patients with hemothoraces, none were greater than 800 cc. All four patients with low CVO_2 saturations who had hemothoraces bled more than 800 cc from their chest tubes.

Two patients with normal CVO_2 saturation had cardiac injuries identified. Both of these patients suffered anterior chest stab wounds and had their cardiac injuries identified by diagnostic pericardial window. Neither patient demonstrated any evidence of cardiac tamponade. They both had a minimal amount of blood within the pericardium, good wall motion and normal CVP measurements just before the induction of anesthesia. Neither patient had a hemothorax and thus they were judged to have suffered insignificant blood loss.

The mean estimated blood loss in the group of patients with CVO_2 saturations greater than 65% was 390 cc (range, 50–800 cc). Only six of these patients ever required blood and only four of these six required their blood within the first 24 hours of admission. The mean transfusion requirement in this group of 16 patients was 1.0 units (range, 0–6 units) and was 2.3 units in the six patients requiring transfusions. Nine of these 16 patients had repeat venous blood gases. Two patients who had initial CVO_2 saturations of 68% and 72% had a dramatic decrease on repeat determination to 54% and 60%, respectively. Repeat hematocrits had dropped 8 mg% in each of these patients. Both were found to have significant injuries requiring 4 units of blood acutely in one patient and 6 units in the other patients. The other seven patients had stable venous saturations. Seven patients had their workup completed expeditiously and were determined to have not suffered any blood loss of significance. They did not have repeat blood gases drawn.

Linear regression coefficients tabulated in Table II demonstrate the superiority of CVO_2 saturation to predict EBL with a p value <0.005.

DISCUSSION

Early recognition of hemorrhage is the cornerstone of the evaluation and resuscitation of acutely injured patients. While mechanism of injury is helpful, many patients with a mechanism or location suggesting the possibility of significant blood loss turn out to have insignificant injuries with a minimal blood loss. In general, serial vital signs are used to gauge the severity of blood loss.

TABLE II
Parameters vs. EBL

	r^2 Values		
	Initial	Repeat	Δ
HR	0.199	0.012	0.17
BP	0.004	0.037	0.001
CVP	0.202	0.024	0.049
PP	0.005	0.015	0.011
Urine	0.012		
CVO_2	0.436		

The Advanced Trauma Life Support course teaches that tachycardia is the earliest sign of hemorrhage, followed by a narrowed pulse pressure and loss of capillary refill (1). Heart rate is one of the central components of cardiac output and an increased heart rate will help to compensate for a decreased preload and aids in maintaining peripheral oxygen delivery and consumption. Yet heart rate can be a nonspecific sign, as many factors other than blood loss such as anxiety will effect heart rate. Pulse pressure will vary from patient to patient and factors such as ambient temperature and the ingestion of vasodilators such as alcohol and/or drugs may alter capillary refill.

Other compensatory mechanisms exist to maintain peripheral oxygen consumption. Vasoconstriction limits blood flow to nonessential tissues and intense vasoconstriction may limit the necessity of an increase in heart rate. Finally, an increase in tissue oxygen extraction may occur. This would allow oxygen consumption to remain normal or even increase in the face of decreased hemoglobin and preload.

As there are many compensatory mechanisms that potentially contribute to maintaining oxygen consumption, it would be extremely useful to know which one of these is the most sensitive marker of blood loss and if there is variability between different individuals. Peripheral post-capillary venous pressure and cuff-occluded rate of rise of peripheral venous pressure have been proposed to be both sensitive and reliable but have not gained any real acceptance in clinical medicine (3). Mixed venous oxygen saturation has been demonstrated to be a useful early indication of decreased tissue perfusion in cardiac surgery (4). We have previously demonstrated that both mixed venous and central venous oxygen saturation are the most reliable and sensitive indicators of acute blood loss in a canine model of blood loss (2). In our series, this was true both in an anesthetized as well as awake canine model of hemorrhage.

Questions still existed as to whether these laboratory results were reproducible in humans and whether the information should alter patient management. In addition, the safety of central venous catheter insertion in potentially volume-depleted patients remained unclear. This study was undertaken to answer these questions. Our data demonstrate that central venous oxygen saturation is more reliable and sensitive than the vital signs commonly used in the initial assessment of patients following trauma. This was true at a p-value <0.005. The linear regression coefficient of CVO_2 saturation versus blood loss was 0.436, which is considerably less than the

r value of 0.92 that we reported in our laboratory model. Linear regression coefficients were considerably less for all parameters in the clinical series as opposed to the laboratory study. The laboratory model was carefully controlled and every cc of blood loss was measured. This obviously was not possible in the clinical series. We could not measure blood loss at the scene. In addition, there was a small time difference in CVO_2 saturation measurement and blood collection in some patients. For instance, the patients with abdominal trauma had CVO_2 saturations measured in the Emergency Department and blood loss measured in the operating room. Also serial hematocrits and transfusion requirements can only estimate blood loss.

While deficiencies exist in the experimental design, we practice medicine in the ED and operating room and not within a controlled laboratory model of disease. A decrease in CVO_2 saturation reliably predicted blood loss and severity of injuries. Patients with low CVO_2 saturation had large hemothoraces, multiple fractures including pelvic fractures, and significant vascular injuries. One had liver and kidney injuries and another had a splenic injury. Although both remained stable, repeat CVO_2 saturations and hematocrits fell and both required blood transfusions.

It is particularly disturbing that while all 26 patients had normal vital signs on admission, a significant portion of them had serious injuries and ongoing blood loss that required immediate attention. Some may argue that all of these patients eventually would have manifested clinical signs of ongoing volume loss. While it is true that these patients would have ultimately developed hemodynamic instability, the earliest possible detection of ongoing blood loss prompts treatment that limits hypoperfusion and prevents the potential sequelae of subacute shock (5). Our clinical data support the concept that central venous oxygen saturation can aid in this early detection of blood loss. Central venous catheterization has been considered dangerous in the acute trauma setting and catheters have generally been placed electively as monitoring lines. While we emphasize that these lines were not utilized for resuscitation, the ability to measure CVO_2 saturation makes them attractive. When questioned, the attending physicians and chief residents uniformly felt that a low CVO_2 saturation heightened their suspicion of occult blood loss. This allowed them to triage these patients to a higher priority, forced a more expeditious workup, and may have resulted in earlier operative intervention in selected cases. While it might have been a purer study to blind clinicians to the results of venous gases, we did not feel we could deny them data that might impact on patient care.

Complete mixing of venous return does not occur until venous blood reaches the pulmonary artery. This potentially jeopardizes the validity of using right atrial or superior vena caval oxygen saturations as an estimate of mixed venous saturation. However, in our animal model, central venous saturations correlated extremely well with blood loss and mirrored changes in mixed venous saturation (2). In addition, Ito et al. have described a numerical relationship between the central venous and mixed venous saturation (6).

Central venous saturation is a reliable and accurate method of detecting blood loss in the acute setting. We would caution that its utility is unproven once resuscitation is started. Isovolemic hemodilution may have an effect on the relationship between venous saturation and peripheral perfusion. In addition, hypothermia, shock, and blood transfusions all may all affect both the distribution of blood flow and the cells' ability to extract oxygen. We have utilized serial venous saturations only in a few patients and the data are insufficient to reach a conclusion. In addition, factors other than blood loss may contribute to venous desaturation. Arterial hypoxia or low cardiac output states such as cardiac tamponade or myocardial depression from a cardiac contusion may result in a low CVO_2 saturation. Our patients were young, had sustained penetrating trauma almost exclusively, and were evaluated soon after their trauma. The utility of CVO_2 as a predicator of blood loss in the general trauma population remains unproven.

We had no complications from CVP catheter placement. Our CVP lines were placed by second- and third-year residents under the direct supervision of chief residents and/or attendings. This is a relatively small group of patients and there is little question that there eventually will be complications if enough CVP lines are placed. It is unclear, though, whether the complication rate will be any higher in this group of patients than it is in any other group. At this point, we feel that the added information that is gained from knowing the CVO_2 saturation justifies the potential risks.

CONCLUSIONS

Central venous oxygen saturation is a safe, rapid, and reliable method of detecting blood loss in patients who have suffered penetrating trauma. It is more sensitive and specific than the commonly used clinical parameters. It is a useful clinical tool for triage and is a useful addition to the initial evaluation of stable but potentially seriously injured patients. Its use in patients who have suffered blunt trauma or who have received a significant amount of crystalloid for resuscitation will require further study.

REFERENCES

1. American College of Surgeons' Committee on Trauma: *Advanced Trauma Life Support Course Instructor Manual,* Chicago, 1985.
2. Scalea, T. M., Holman, M., Fuortes, M., et al.: Central venous blood oxygen saturation: An early accurate measurement of volume during hemorrhage. *J. Trauma,* **28:** 725, 1988.

3. Sheldon, C. A., Balik, E., Dhanalal, K., et al.: Peripheral post capillary venous pressure: A new hemodynamic parameter. *Surgery,* **92:** 663, 1982.
4. Jamieson, W. R., Turnbull, K. W., et al.: Continuous monitoring of mixed venous saturation in cardiac surgery. *Canadian J. Med.,* **25:** 538, 1982.
5. Shoemaker, W. C.: Controversies in the pathophysiology of post-operative adult respiratory distress syndrome. *Surg. Clin. No. Amer.,* **65:** 931, 1985.
6. Ito, K., Ramirez-Schon, G., Nehta, J. G., et al.: A simplified technique for determining mixed venous blood gases. *Surg. Forum,* **30:** 1–3, 1979.