Measurement of Central Venous Oxygen Saturation in Patients with Myocardial Infarction

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SUMMARY
Central venous oxygen saturation (CVSO₂) was measured in 31 patients with myocardial infarction. CVSO₂ correlated well with the patients’ clinical course. In those patients not in heart failure, mean ± SEM for CVSO₂ was 70 ± 1%. When heart failure was present, CVSO₂ averaged 56 ± 1%. When both heart failure and shock were present, CVSO₂ averaged 43 ± 1%. In nine patients, serial determinations of arterial oxygen saturation and CVSO₂ were made. In 22 of 26 instances, either a fall in CVSO₂ was accompanied by an increase in the arteriovenous oxygen saturation difference or an increase in CVSO₂ was accompanied by a decrease in arteriovenous oxygen saturation difference. Serial measurements of CVSO₂ appear to be a useful method of monitoring changes in myocardial function in patients with myocardial infarction.

Additional Indexing Words:
Mixed venous oxygen saturation  Heart failure  Cardiac output  Shock
Central venous pressure

MEASUREMENT of cardiac output and intracardiac pressures is used to assess myocardial function in the catheterization laboratory. For patients with myocardial infarction, serial measurements of cardiac output can be helpful in evaluating changes in myocardial function. In most coronary care units, however, the special equipment and technical skills which are used to measure cardiac output may not be readily available. To circumvent this problem, we sought a simple method of assessing changes in cardiac function. Consequently, central venous oxygen saturation (CVSO₂) is now monitored in patients admitted to the Palo Alto-Stanford Coronary Care Unit. CVSO₂ was chosen because it reflects both arterial oxygen saturation and tissue extraction of oxygen.

A retrospective study on the use of CVSO₂ measurements in our Coronary Care Unit was recently carried out and suggested that changes in CVSO₂ correlated with changes in the patient’s clinical status.¹ The purpose of this paper is to report the results obtained on 31 patients studied prospectively in our unit and to offer further validation of the use of changes in CVSO₂ as an index to changes in myocardial function.

Methods
Patients admitted to our Coronary Care Unit have a central venous catheter inserted percutaneously into an antecubital vein and advanced centrally to the superior vena cava. The position

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of the catheter was verified by chest x-rays in half of the 31 patients studied. Central venous pressure (CVP) is measured by a water manometer with zero level being the midthorax. Blood was withdrawn from the catheter by disconnecting the intravenous drip used to keep the catheter patent and withdrawing 3 to 5 cc of fluid and blood. Following this, a heparinized syringe was used to withdraw 3 cc of blood anaerobically. This blood was then rapidly transferred to a glass cuvette. Oxygen saturation was measured on an American Optical reflection oximeter, Model no. 1824. The reproducibility of measurements made with this instrument was found, on duplicate determinations, to be ±2% for oxygen saturations between 40 and 100%. The oximeter was calibrated using standards supplied with the machine and checked against blood oxygen saturation determined by the spectrophotometric method of Huckabee.2

In nine of our patients, arterial blood was also obtained, either by percutaneous puncture of the brachial or radial artery or by placing a catheter in the brachial artery through a surgical incision in patients in shock. In four patients, oxygen consumption was measured by collecting expired air in a lightweight Douglas bag through a low-resistance valve. Collections were taken over a 4 or 5 minute period after an initial period of acclimatization to the mouthpiece. Gas volume was measured with a dry-gas meter. The expired air was analyzed for oxygen and carbon dioxide by the Schollander technique. Measurements were made at least 4 hours apart in each patient.

In all except three patients who were thought to be too ill, nasal oxygen administration was discontinued for 15 minutes before study. This amount of time has been found necessary to return patients to basal oxygen saturation. To obtain nearly basal conditions, no patient was studied within 1 hour of receiving analgesics or when having chest pain. All patients in this study had at least three determinations of CVSO₂. All patients had been at complete rest for at least 10 minutes before a determination was performed. No patient with chronic pulmonary disease, temperature greater than 38 °C, or anemia was included in this study. However, the arteriovenous oxygen saturation differences of one patient with anemia are included, because we believe that although anemia might alter the absolute CVSO₂ value obtained, the direction of change of CVSO₂ and of arteriovenous oxygen saturation difference would not be influenced by the presence of anemia.

Patients who had CVSO₂ values greater than 60% while receiving oxygen had this measurement repeated when they were not receiving oxygen; only CVSO₂ determinations made while patients were breathing room air are included in this report.

All patients were examined by one of the authors twice daily, or oftener if they were in shock or if there was a dramatic change in their clinical state, and a determination was made as to whether or not the patient was in heart failure or shock. This determination was made without prior knowledge of the CVSO₂ of the patient. CVSO₂ blood was obtained and measured independently by members of the Coronary Care Unit nursing staff, usually within 1 hour of examination of the patient by one of the authors. All arterial punctures and collections of expired air were performed by one of the authors after clinical assessment of the patient's condition had been made. Specimens of arterial blood were handled in the same manner as venous blood. Statistical comparisons between the different groups of patients studied were performed using an unpaired t test.

Group Studied

Patients admitted to our Coronary Care Unit were studied if their physician gave permission for them to be examined by one of the authors. Thirty-one patients with a diagnosis of acute myocardial infarction established by clinical history and either definite electrocardiographic changes (the presence of diagnostic Q waves with S-T and T-wave changes and the evolutionary electrocardiographic changes commonly associated with acute myocardial infarction) or suggestive electrocardiographic changes (the presence of changing S-T and T-wave abnormalities) accompanied by significant, but transient, elevation of serum glutamic oxalacetic transaminase (SGOT), creatinine phosphokinase (CPK), or lactate dehydrogenase (LDH) are included in this report. Patients were divided into three groups:

Group I: Eleven patients with uncomplicated myocardial infarction.

Group II: Fifteen patients with myocardial infarction and congestive heart failure at some time during their hospital course. The presence of a protodiastolic gallop, pulmonary rales, elevated CVP (greater than 12 cm H₂O), or peripheral edema were taken to indicate failure.

IIA: Group II patients when not in failure (10 patients).

IIB: Group II patients when in failure.

Group III: Four patients with myocardial infarction, congestive heart failure, and shock. The same criteria as in group II were used for heart failure. In addition, shock was felt to be present.

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when the systolic blood pressure was 90 mm Hg or less and peripheral signs of decreased perfusion were present.

In addition to these patients, the arteriovenous oxygen saturation differences obtained in one patient with myocardial infarction and anemia are included.

Results

Group I

Sixty-one CVSO₂ measurements were made in the 11 patients in this group. Mean CVSO₂ ± standard deviation (SD) was 70 ± 7%. In figure 1 is presented the mean ± standard error of the mean (SEM) for this group. Only four CVSO₂ values in four patients were below 60%, and in each of these patients, CVSO₂ determinations before and after the low values were greater than 60%.

Group II

One hundred fourteen CVSO₂ determinations were made on these 15 patients; 32, when no evidence of heart failure was present (group IIA), and 82 when clinical evidence of heart failure was present (group IIB). Only five of the 32 CVSO₂ determinations in group IIA were less than 60% and the mean value ± SD was 66 ± 8%. In group IIB mean ± SD CVSO₂ was 56 ± 10%. Twenty-five of

![Figure 1](image1)

**Figure 1**

Central venous oxygen saturation (CVSO₂) in patients with no clinical evidence of heart failure. Mean ± SEM CVSO₂ for the group was 70 ± 1%. Only four values were less than 60%.

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![Figure 2](image2)

**Figure 2**

Central venous oxygen saturation (CVSO₂) in patients with clinical evidence of heart failure. Mean ± SEM was 66 ± 1% when not in heart failure (group IIA) and 56 ± 1% when heart failure was present (group IIB).
the 82 CVSO₂ values were greater than 60% while the patients were in clinical heart failure. However, 10 of these values were obtained within the 24 hours preceding the disappearance of all clinical evidence of failure. In figure 2 the means ± SEM(σ) for group IIA and B are presented. Ten patients were studied both in and out of failure. Figure 3 gives the CVSO₂ values obtained in patients who developed signs of heart failure while CVSO₂ was being monitored. The values presented are the last value obtained before and the first value obtained after a change in the patient's clinical state. In seven of eight patients who developed failure, CVSO₂ fell coincident with the development of failure. In eight patients, signs of failure abated while CVSO₂ was being monitored. In four of these eight patients CVSO₂ rose coincident with improvement in their clinical state. In two of these patients, CVSO₂ fell, and in two it did not change even though their clinical state improved. In group II patients, only nine of 15 had elevated CVP, while 13 of 15 had CVSO₂ of less than 60%. Barratt-Boyes and Wood⁴ have shown that in normal individuals, oxygen saturation of superior vena caval blood averaged 76.8 ± 1.0%. Thus, although a number of our patients had CVSO₂ values greater than 60% when they were in heart failure, none of these patients had a normal CVSO₂ when in heart failure.

**Group III**

Twenty-five CVSO₂ values were obtained on group III patients. Mean ± SD was 43 ± 7%. In figure 4 the mean ± SEM for group III patients is presented. In patients who did not receive oxygen therapy, CVSO₂ averaged 40%. No CVSO₂ value was greater than 60%. In group III patients, all four patients had an elevated CVP and all had a CVSO₂ value less than 45%.

Serial oxygen consumptions were obtained daily in three patients, and three times in
Serial oxygen consumption measurements in four patients with myocardial infarction.

One day in one patient. In each of these patients oxygen consumption remained relatively constant (fig. 5). These data support our contention that these patients were studied in a basal state.

In figure 6, the mean and SEM for the CVSO₂ values obtained in the different groups of patients are compared. There was a slight but significant difference in CVSO₂ values between groups I and IIA. When heart failure was clinically apparent (group IIB), there was a significant decrease in mean CVSO₂. When heart failure and shock were present (group III), there was a further significant fall in CVSO₂.

**Discussion**

To be of value in a Coronary Care Unit, CVSO₂ determinations should meet three criteria: (1) CVSO₂ values should reflect the patient's clinical status. (2) Changes in CVSO₂ should reflect changes in myocardial function. (3) The measurement of CVSO₂ should be easy to obtain without significant risk to the patient.

The results of this study, as well as that previously performed, support our contention that CVSO₂ values reflect the patient's clinical status. As clinical evidence of myocardial dysfunction increased with the development of heart failure and then heart failure and shock, CVSO₂ decreased. In group I patients, only four of 61 CVSO₂ values were less than 60%. When heart failure was present (group IIB), 57 of 82 CVSO₂ values were less than 60%. When both heart failure and shock were present (group III), 19 of
25 CVSO₂ values were 45% or less and all 25 CVSO₂ values were less than 60%.

The second criteria to be met is that changes in CVSO₂ should reflect changes in myocardial function. Valentine and associates demonstrated that oxygen consumption remains relatively constant in a basal state, and that changes in arteriovenous oxygen differences are inversely related to changes in cardiac output following myocardial infarction. We have studied patients in a basal state only. Therefore, when arteriovenous oxygen difference widens, cardiac output must decrease and when arteriovenous oxygen difference decreases, cardiac output must increase. In figure 7 the changes in CVSO₂ are plotted against the changes in arteriovenous (A-V) oxygen saturation difference obtained in nine patients who had simultaneous arterial and venous samples drawn for measurements of oxygen saturation. The changes in CVSO₂ and in A-V oxygen saturation difference were calculated using the last CVSO₂ value and A-V oxygen saturation difference value obtained in each patient as the reference point. In the nine patients studied, 35 A-V oxygen saturation differences were obtained. Thus 26 comparisons were made between the changes in A-V oxygen saturation differences and the changes in CVSO₂. In figure 7 it can be seen that in nine instances in which CVSO₂ increased, A-V oxygen saturation difference decreased, while in no instance did CVSO₂ increase while A-V oxygen saturation difference also increased. In 13 instances a fall in CVSO₂ was associated with an increase in A-V oxygen saturation difference, while in only three instances was a fall in CVSO₂ associated with a decrease in A-V oxygen saturation difference. In one instance, CVSO₂ fell without a change in A-V oxygen saturation difference. Thus, our data extend the findings of Valentine and associates by showing that a fall in CVSO₂ is accompanied by an increase in A-V oxygen saturation difference, while a rise in CVSO₂ is accompanied by a decrease in A-V oxygen saturation difference. Of the nine patients studied with arterial and venous samples, two were not in heart failure, four were in heart failure, and three were in heart failure and shock. Although these groups are small, it appears that the changes in CVSO₂ can be used as a guide to changes in myocardial function even when severe myocardial dysfunction is present.

The third criteria that must be met if CVSO₂ measurements are to be of use in a coronary care unit is that they should be safely and easily obtained. The experience of our unit suggests that nursing personnel can be taught to insert central venous catheters safely into critically ill patients and that CVSO₂ measurements can be easily and accurately obtained by nurses.

Measurement of CVSO₂ cannot be used alone to predict arterial oxygen saturation or to predict or calculate cardiac output. However, the results of this study indicate that as myocardial function deteriorates, CVSO₂ falls. When CVSO₂ is less than 60%, clinical evidence of heart failure is usually present. When CVSO₂ is less than 45%, myocardial dysfunction has usually progressed to the point where both heart failure and shock are present.

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References

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