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Mixed Venous Oxygen Saturation in Critically Ill Septic Shock Patients*

The Role of Defined Events

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Study objective: To investigate the frequency and extent of spontaneous changes ("events") in continuously measured mixed venous oxygen saturation (SvO_2) in septic patients and to determine whether attention to individual event-frequency offers additional information for patient management.

Design: Nonrandomized prospective study.

Setting: General intensive care unit at a university hospital.

Patients: Fifteen patients suffering from septic shock and multiple organ dysfunction syndrome.

Measurements: For the continuous assessment of SvO_2 a fiberoptic pulmonary artery catheter (Baxter Edwards) was inserted in all patients. A certain event was defined as a sudden change in SvO_2 of ≥5 percent lasting for >10 min. All events were grouped as either moderate (≤10 percent changes in SvO_2) or severe events (>10 percent changes). Hemodynamics and inotropic support, oxygenation and ventilatory support, hemoglobin levels and body temperature were determined at the event and compared with the ultimate values registered before the event.

Results: We evaluated 377 events during an observation period of 1,575 h. Patients' mean SvO_2 levels ranged between 72 ± 7 and 82 ± 4. Desaturations below 65 percent (39 out of 377 events) occurred in 11 patients. Overall, 74 percent of all events were moderate and 26 percent were severe. The incidence of events was 5.6 ± 1.5 during 24 h in survivors (n = 10) and 6.3 ± 1.6 during 24 h in nonsurvivors (n = 5). While in survivors only 20 percent of all events were severe events, this portion was significantly higher in nonsurvivors (34 percent; p = 0.03). In 67 percent of all events we observed changes in the registered physiologic parameters or therapeutic interventions probably causing the event. The cause of the remaining 33 percent of all events could not be elucidated.

Conclusions: The SvO_2 of septic shock patients is mainly normal or even supranormal. However, short-term changes in SvO_2 do occur frequently in these patients. Nonsurvivors exhibit a higher frequency as well as a significantly greater severity of events, which may point toward a concealed mismatch of oxygen supply and demand. A high incidence of short-term SvO_2 changes in a septic shock patient may be of diagnostic and prognostic significance. Therefore, we recommend the installation of a computerized alarm-function for the automatic detection and indication of frequent events. ***(Chest 1993; 103:900-06)***

CO = cardiac output; Do_2 = oxygen delivery; SaO_2 = arterial oxygen saturation; SvO_2 = mixed venous oxygen saturation

In patients suffering from septic shock, some evidence exists that oxygen utilization is limited by tissue perfusion.[1,2] An increase in oxygen delivery (Do_2) results in a concomitant increase in oxygen consumption ($\dot{V}o_2$),[3-5] especially in patients exhibiting elevated serum lactate levels. It has been suggested that supranormal levels of Do_2 and $\dot{V}o_2$ may improve outcome of critically ill patients.[6-8] Since mixed venous oxygen saturation (SvO_2) is influenced by and is dependent on this relationship between $\dot{V}o_2$ and Do_2, fiberoptic pulmonary artery catheters, enabling the on-line determination of SvO_2, could be used for on-line monitoring of this relationship, and spontaneous changes in SvO_2 may therefore herald changes in oxygen demand or transport.

The first investigations concerning the clinical use of continuous SvO_2 monitoring reported a limited practicability of this new technique due to technical problems and due to a disappointing correlation of SvO_2 to cardiac output (CO).[9-11] Because of the introduction of more elastic catheter materials and the improvement of the fiberoptic technique, several recent studies show an excellent correlation between the SvO_2 measured *in vitro* and *in vivo*.[12-15]

Despite the clinical controversy,[16-18] whether attention to or management of SvO_2 may change the prognosis of septic shock patients, there is still no information available concerning the frequency of short-term SvO_2 changes occurring during the treatment of these patients.

The aim of this study was therefore to investigate the frequency and extent of sudden SvO_2 changes (*ie*, "events") in patients suffering from septic shock and multiple organ dysfunction syndrome and to determine whether attention to the individual event-frequency offers additional information for the management of these patients.

Methods

Patients

We studied 15 patients suffering from septic shock after surgery

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Table 1—*Clinical Data of Patients Studied**

Patient	Age, yr	Sex	Diagnosis*	Multiple Organ Failure Score at Entry	Observation Period, h	No. of Events	Outcome
1	57	M	Aortic aneurysm	9	110	37	Expired
2	28	M	Multiple trauma	7	64	13	Discharged
3	22	F	Gestosis, ARI	6	45	14	Discharged
4	33	M	Head injury	7	90	14	Discharged
5	32	F	ARDS, DIC	6	149	38	Discharged
6	80	M	Mesenteric embolism	8	68	21	Discharged
7	21	F	ARDS, gestosis, DIC	8	202	36	Expired
8	25	M	Lobectomy	8	58	17	Expired
9	43	M	Multiple trauma	7	99	32	Discharged
10	66	F	Pancreatitis	10	70	15	Expired
11	25	M	Head injury	7	66	9	Discharged
12	52	M	Lobectomy	7	45	11	Discharged
13	22	M	Multiple trauma	8	192	34	Discharged
14	19	M	ARDS, multiple trauma	8	172	51	Expired
15	18	F	Multiple trauma	7	145	35	Discharged

*ARI, acute respiratory insufficiency; DIC, disseminated intravascular coagulation; ARDS, adult respiratory distress syndrome.

or multiple trauma who were admitted to our general ICU. The patient population studied is shown in Table 1.

Diagnosis of septic shock was made according to the criteria of the ACCP/SCCM Consensus Conference,[19] requiring (1) a systemic response to infection manifested by two or more of the following conditions: temperature greater than 38°C or less than 36°C, heart rate greater than 90 beats per minute, respiratory rate greater than 20 breaths per minute or $PaCO_2$ less than 32 mm Hg, WBC count more than 12,000 cells/mm^3, less than 4000 cells/mm^3 or greater than 10 percent immature forms and (2) hypotension (systolic blood pressure less than 90 mm Hg or a reduction of greater than 40 mm Hg from baseline) despite adequate fluid resuscitation, along with the presence of perfusion abnormalities that may include lactic acidosis, oliguria, or an acute alteration in mental status. Multiple organ dysfunction syndrome was defined as the presence of altered organ function in an acutely ill patient so that homeostasis could not be maintained without intervention.

In order to estimate the extent of organ dysfunction and to compare the severity of disease in different patients, the multiple organ failure score according to Goris et al[20] was assessed for each patient (Table 1).

All patients were sedated (Midazolam [5 to 18 mg/h] in combination with Sufentanil [45 to 150 μg/h]) and were mechanically ventilated (Evita; Dräger) with tidal volumes between 10 and 15 ml/min/kg of body weight.

Monitoring and Measurements

An arterial catheter was placed into the radial artery for invasive blood pressure measurement and arterial blood gas collection. A fiberoptic pulmonary artery catheter enabling the continuous measurement of SvO_2 (SAT-2, Baxter Edwards Laboratories, Irvine, Calif) was inserted through the internal jugular or the subclavian vein. *In vivo* calibration of the catheter SvO_2 with directly measured SvO_2 was performed every 24 h. Inaccurate measurement due to changing hemoglobin levels was avoided by updating hemoglobin levels in the SAT-computer whenever hemoglobin changes more than 10 percent occurred. Incorrect measurements due to catheter malfunction (*eg*, fibrin deposits at the catheter tip) or malposition were indicated by the SAT-computer and deleted from evaluation.

Cardiac output was measured every 2 h by the thermodilution technique. Measurements were performed in triplicate by using 10-ml room temperature aliquots of Ringer's solution and were then averaged. Body temperature was measured by means of the pulmonary artery catheter thermistor (blood temperature). Blood samples were drawn together with the CO determinations and arterial and mixed venous oxygen contents were directly measured with an oximeter (OSM 3). Derived cardiopulmonary parameters were calculated according to standard formulas.[21]

Study Protocol

The study period started with the onset of septic shock and multiple organ dysfunction syndrome and lasted as long as multiple organ dysfunction syndrome persisted (which was equal to a multiple organ failure score equal to or more than 5) or until the patient expired. We defined eight treatment intervals lasting 3 h each, starting with 0:00 to 3:00 AM and ending with 9:00 PM to 12 PM.

After approval by the institutional ethics committee, continuously measured SvO_2 values were recorded throughout the entire study period. Cardiopulmonary performance was assessed routinely every 2 h and whenever changes in the patient's condition or hemodynamics occurred. Arterial and mixed venous blood gas analyses were done every 2 h and furthermore when demanded by the managing physician.

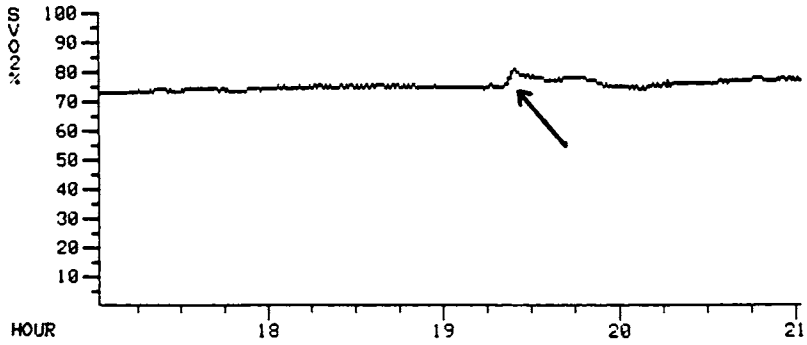

FIGURE 1. Typical moderate event (*arrow*) with an increase in SvO_2 from 74 to 80 percent lasting for about 40 min.

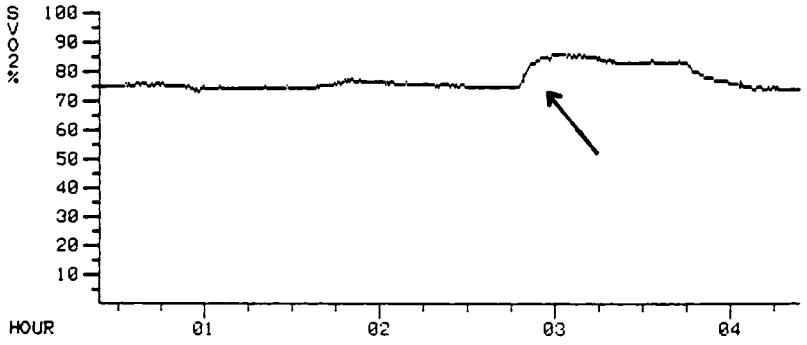

FIGURE 2. Typical severe event *(arrow)* with an increase in SvO_2 from 74 to 85 percent lasting for about 75 min.

A sudden fall or rise of greater than or equal to 5 percent below or above the initial value, lasting for more than 10 min, was defined as an *event*. All observed events were grouped according to their magnitude as moderate events (sudden fall or rise of SvO_2 of 10 percent or less [Fig 1]) and severe events (sudden fall or rise of more than 10 percent [Fig 2]) and were evaluated separately. At or immediately after each event, simultaneous arterial and mixed venous blood samples, followed by the determination of CO were accomplished. Cardiac output, inotropic support (dobutamine), hemoglobin level, oxygenation (SaO_2), ventilatory support with the level of positive end-expiratory pressure (PEEP) and body temperature were recorded.

Continuous recordings of SvO_2 were investigated by two physicians not directly involved in the management of the patients. To investigate whether a physiologic change or a therapeutic intervention may have led to a certain SvO_2 alteration, we compared the ultimate values of the SvO_2 determinants recorded before the event with the values at the time of the event. Changes of greater than or equal to 5 percent in SaO_2, of greater than or equal to 10 percent in hemoglobin, and CO of greater than or equal to 2 μg/kg/min in the dobutamine dosage, of greater than or equal to 2 cm H_2O in PEEP and of greater than or equal to 0.5°C in body temperature were considered significant and were assumed to be a possible cause of the event.

Events caused by direct patient care such as endotracheal suction or patient turning or by diagnostic procedures such as bronchoscopy were excluded from evaluation.

Statistical analysis was done using the χ^2 test and a p value less than 0.05 was considered significant. Data are shown as mean value ± SD.

RESULTS

Ten male and five female patients suffering from septic shock were investigated. Ten were discharged from the hospital as survivors, whereas five patients died ultimately, resulting in a mortality rate of 33 percent (Table 1). All patients had multiple organ dysfunction syndrome, and the average multiple organ failure score[20] at study entry was 7 ± 0.7 for survivors and 8.6 ± 0.9 for nonsurvivors, respectively.

We observed and evaluated 377 events in these 15 critically ill septic patients during a cumulative observation period of 1,575 h. Actual numbers of events observed in individual study patients are shown in Table 1. The calculated incidence of short-term SvO_2 changes during an observation period of 24 h was 5.6 ± 1.5 events in survivors and 6.3 ± 1.6/24 h events in nonsurvivors. This trend toward a higher incidence of events in nonsurvivors did not reach statistical significance.

The patients' mean SvO_2 levels at the events ranged from 72 ± 7 (patient 1) to 82 ± 4 (patient 7), with a mean duration of events within a range from 16 ± 7 (patient 10) to 29 ± 19 (patient 11) min (Table 2).

Fifty-five percent of all observed events occurred during 6:00 AM and 6:00 PM and of these, 29 percent occurred during 6:00 AM and 9:00 AM. The remaining 45 percent of all events occurred during 6:00 PM and 6:00 AM, and more than two thirds of these occurred between 3:00 PM and 9:00 PM. However, most events that occurred during these nursing shift change periods (around 7:00 AM and 7:00 PM) were minor events. Absolute numbers and severity of events according to daytime are shown in Figure 3 for survivors and in Figure 4 for nonsurvivors.

Seventy-four percent of all SvO_2 changes were moderate events (36 percent were decreases and 38 percent, increases) and 26 percent of all SvO_2 changes were severe (14 percent decreases and 12 percent increases); 5.5 percent of all events were greater than 15 percent (Table 3).

Nonsurvivors exhibited a significantly higher number of severe events than survivors. While in nonsurvivors 34 percent of all events were severe events (in the range of >10 percent), this portion was only about

Table 2—*Number and Duration of and Mean Mixed Venous Oxygen Saturation at all Observed Events*

Patient*	No. of Events	Duration†	Mean SvO_2‡
1	37	25 ± 14	72 ± 7
7	36	19 ± 11	82 ± 4
8	17	24 ± 14	76 ± 6
10	15	16 ± 7	80 ± 5
14	51	27 ± 14	78 ± 7
2	13	16 ± 6	79 ± 5
3	14	26 ± 16	79 ± 5
4	14	26 ± 16	81 ± 4
5	38	17 ± 10	75 ± 5
6	21	23 ± 9	72 ± 6
9	32	29 ± 15	72 ± 5
11	9	29 ± 19	74 ± 5
12	11	18 ± 13	77 ± 6
13	34	25 ± 16	79 ± 6
15	35	24 ± 13	74 ± 6

*Patients 1,7,8,10 and 14 were nonsurvivors and patients 2,3,4,5, 6,9,11,12,13 and 15 were survivors.
†Mean duration ± SD of events (minutes).
‡Mean SvO_2 ± SD at all observed events.

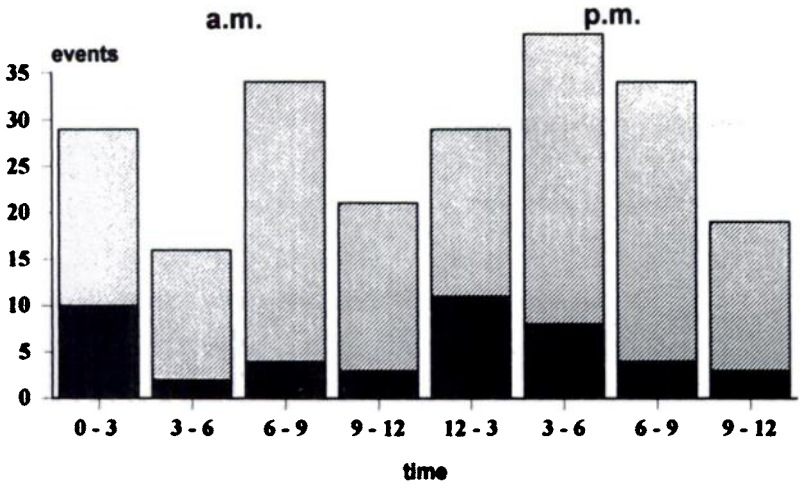

FIGURE 3. Incidence of events according to daytime and severity in survivors (solid bars indicate severe events and hatched bars indicate moderate events).

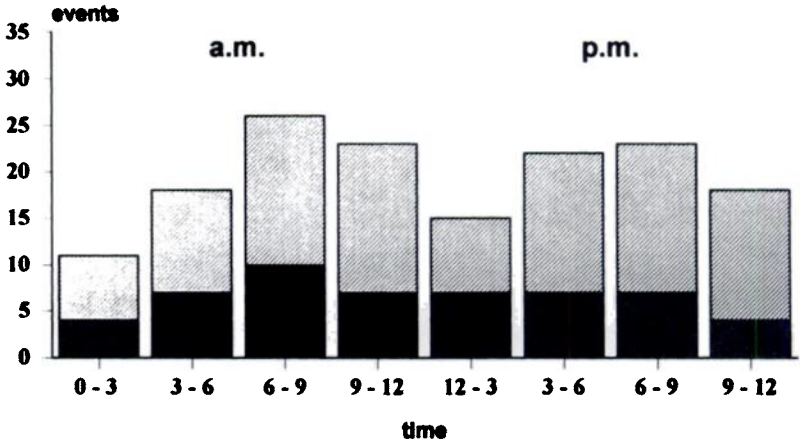

FIGURE 4. Incidence of events according to daytime and severity in nonsurvivors (solid bars indicate severe events and hatched bars indicate moderate events).

20 percent in survivors ($p = 0.03$).

Severe desaturations below 65 percent occurred in 11 patients (39 of 377 events) with a duration between 11 and 60 min. The remaining four patients (three survivors and one nonsurvivor) never suffered from severe desaturations. Table 4 shows the actual number of severe desaturations for all individual patients, as well as the lowest SvO_2 values and the duration of these events.

The number and severity of events as well as the

Table 3—*Number,* Severity, and Potential Cause of All Events in Individual Patients*

	SvO_2												
	Decrease			Increase			Related to Changes in						
Patient†	>15	>10	>4	>4	>10	>15	CO	Dobutamine	SaO_2	Hemoglobin	PEEP	Body Temperature	Unknown
1	. . .	6	10	16	5	. . .	11	4	2	2	1	10	12
7	2	3	16	12	2	1	10	9	. . .	12	3	6	14
8	2	2	4	4	4	1	3	3	. . .	5	. . .	3	7
10	1	2	6	5	1	. . .	2	5	2	2	3	1	6
14	3	8	14	16	7	3	14	10	5	11	6	8	18
2	. . .	1	5	7	. . .	. . .	3	1	. . .	2	. . .	2	4
3	. . .	1	6	5	2	. . .	6	3	2	—	1	5	1
4	. . .	. . .	8	6	. . .	. . .	5	. . .	. . .	3	. . .	3	5
5	2	4	11	14	6	1	9	4	. . .	6	2	5	10
6	1	3	7	6	4	. . .	7	3	. . .	4	. . .	4	6
9	. . .	3	14	12	3	. . .	5	3	4	6	1	5	13
11	1	1	4	3	. . .	. . .	3	. . .	. . .	4	. . .	3	2
12	1	. . .	2	5	1	2	2	1	. . .	4	1	1	4
13	. . .	2	17	14	1	. . .	5	3	. . .	5	2	6	14
15	. . .	2	13	17	3	. . .	6	5	. . .	9	3	4	8

*Absolute numbers stated in all fields.

†Patients 1,7,8,10, and 14 were nonsurvivors and patients 2,3,4,5,6,9,11,12,13, and 15 were survivors.

suspected causes of the events observed in individual patients are shown in Table 3. Factors most often associated with sudden short-term changes in SvO_2 were as follows: changes in CO (observed in 24 percent of all events) often due to modification of inotropic support (in 14 percent), blood loss, or correction of hemoglobin levels (20 percent). In 18 percent of events, we identified a change in body temperature as a possible cause. By contrast, changes in PEEP (6 percent) and SaO_2 (4 percent) were seldom related to short-term SvO_2 alterations. In other words, in 67 percent of all sudden events, we documented a physiologic change or a therapeutic intervention probably responsible for a special SvO_2 change. In two thirds of these instances a change in a single parameter was found, and in one third, simultaneous changes in more than one determinant were found. For the remaining 33 percent of all registered events, a definitive cause could not be identified.

Table 4—*Number, Duration, and Severity of Desaturation (<65%) in Nonsurvivors and Survivors*

Patient	Outcome	SvO_2 <65%*	SvO_2, Range	Duration, min
1	Expired	9	60 ± 5 (48-64)	21 ± 6
7	Expired	0	. . .	. . .
8	Expired	4	59 ± 5 (53-63)	15 ± 4
10	Expired	2	64, 32†	15, 25†
14	Expired	6	62 ± 6 (54-64)	20 ± 8
2	Survived	0	. . .	. . .
3	Survived	0	. . .	. . .
4	Survived	0	. . .	. . .
5	Survived	3	57, 64, 63†	20, 15, 12†
6	Survived	2	64, 64†	30, 17†
9	Survived	6	61 ± 3 (56-64)	30 ± 16
11	Survived	2	62, 64†	11, 15†
12	Survived	3	58, 60, 61†	15, 18, 17†
13	Survived	1	64†	60†
15	Survived	1	54†	12†

*Absolute number.
†Actual values stated.

Discussion

The study of oxygen kinetics in critically ill patients may be difficult because of therapeutic interventions (*eg*, administration of PEEP, volume expanders, and drugs) and unstable physiologic conditions. The clinical value of continuous SvO_2 monitoring is based on the opportunity to gain on-line monitoring of the balance between oxygen supply and demand in case of changes in SaO_2, CO, hemoglobin, and $\dot{V}O_2$.[22,23] Some reports reveal that aggressive attention of SvO_2 is of prognostic significance. Baele et al[12] could show that continuous SvO_2 monitoring is an accurate and valuable warning system for deterioration in cardiopulmonary function and an indicator of the effects of various therapeutic maneuvers in critically ill patients. The authors concluded that a severe SvO_2 depression below 60 percent in the presence of adequate gas exchange indicates either inadequate cardiac performance or high tissue oxygen consumption. Sumimoto et al[24] reported that SvO_2 was a better predictor of hyperlactatemia and survival than cardiac index in patients with acute myocardial infarction and might be associated with an increased oxygen demand and an impaired oxygen transport system in critically ill patients. By means of discriminant analyses, the authors found only SvO_2 to be a significant variable associated with survival.

However, in patients suffering from multiple organ dysfunction syndrome, significant desaturation as an indicator of potential tissue hypoxia is rarely observed. The SvO_2 levels in our patients were mainly normal or even supranormal (Table 2) during our investigation with only 39 cases of severe desaturation below 65 percent (Table 4). Additionally, these cases of desaturation were observed in survivors as well as in nonsurvivors without a significant difference in severity and duration. Therefore, some authors suggested[25,26] that continuous measurement of mixed venous oxygen saturation could be of limited value particularly in the hyperdynamic septic state, because of mainly normal or even supranormal SvO_2 levels.[16-18] However, we do not assume that the major concern while monitoring SvO_2 in septic shock patients is evidence of significant desaturation as an indicator of potential tissue hypoxia. We believe that the major concern is the observation that short-term changes in SvO_2 do occur, although the maintenance of stable SvO_2 determinants has been a therapeutic goal in our patients. Since nonsurvivors exhibit a higher frequency as well as a significantly greater severity of events than survivors, this observation may point toward a concealed mismatch between oxygen supply and oxygen demand in patients who ultimately succumb and may therefore be of diagnostic and prognostic significance. Thus, not absolute SvO_2 values but changes or variations in SvO_2 may be of major importance and offer additional information for the treatment of these patients. Furthermore, this might also explain the fact that the incidence of sudden events in our patients suffering from septic shock is much higher than that reported by Jastremski et al[27] who continuously measured SvO_2 in a mixed patient population suffering from various diseases. Due to our data, we cannot agree with the conclusion of Jastremsky et al that in critically ill patients SvO_2 changes greater than 10 percent lasting for 5 min often have no clinical importance.

In our septic shock patients, 33 percent of all sudden events were missed by routine clinical monitoring without the on-line determination of SvO_2. This finding is in opposition to the observations of Boutros and Lee.[28] These authors investigated the impact of continuously measured SvO_2 on clinical decision-making in a blinded manner. The authors defined reductions

of SvO_2 below 60 percent or increases above 80 percent, as well as changes of more than 15 percent lasting longer than 10 min as an "occurrence." They concluded that no significant change in SvO_2 occurred without simultaneous changes in the clinical picture or in commonly available measurements. Unfortunately, Boutros and Lee[28] did not give any details concerning the underlying diseases of their patients. Furthermore, hyperdynamic septic shock patients seldom exhibit SvO_2 levels below 60 percent and rarely show SvO_2 changes greater than 15 percent. We suggest that the definition of an occurrence used by Boutros and Lee[28] is inappropriate for investigations in septic shock patients.

In 67 percent of all sudden events, we identified a physiologic change or a therapeutic intervention as being responsible for the changing SvO_2 (Table 3). Of these, the most important factors associated with SvO_2 changes are alterations in blood flow often due to inotropic support, blood loss, or changing hemoglobin levels. The onset of hyperthermia or the initiation of physical cooling of the patient were two other important factors associated with short-term SvO_2 changes. Although PEEP improves arterial oxygenation, its use may result in a fall in CO, with the possible net effect of a deterioration of Do_2.[29] However, changes in PEEP were seldom (in 6 percent) related to sudden alterations in SvO_2. In only 4 percent, changes in SaO_2 may have been responsible for sudden SvO_2 changes; 33 percent of the events were only detected by on-line SvO_2 determination and were missed by our routine clinical monitoring. The first reason for this lack of information is a limitation of our study protocol. We assessed hemodynamics and oxygenation routinely every 2 h. For any event occurring between two measurements, we did not have the exact control values of the SvO_2 determinants in order to compare them with the values at the event. In all these events, no immediate counterregulation by the managing physician was necessary or was initiated to reestablish the initial SvO_2 levels. Therefore, the second reason might be that a stable course of SvO_2 reflects a constant proportion of Do_2 and $\dot{V}o_2$ because of the mathematical coupling between the determinants of SvO_2 and the $\dot{V}o_2/Do_2$ relationship.[30] Thus, even minor deviations from the stable SvO_2 course may represent an early sign of spontaneous short-term changes in oxygen demand. Villar et al[31] demonstrated that large fluctuations in $\dot{V}o_2$ and Do_2 could occur spontaneously in critically ill patients, even in patients with normal blood lactate levels. In their study, neither an experimental intervention nor any therapeutic effort to change Do_2 was performed during the study period, and the authors suggested that these changes probably reflected primary changes in O_2 demand. This statement is in accordance with our finding that spontaneous short-term changes in SvO_2 do occur frequently in critically ill septic shock patients. Since SvO_2 reflects the balance of oxygen supply and oxygen demand, a main cause of the spontaneous SvO_2 changes we observed may have been spontaneous short-term changes in oxygen demand.

The reason for this fluctuation in oxygen demand is not completely understood. Weissman et al[32] studied the effect of routine intensive care interactions on the metabolic rate and demonstrated that a variety of stimuli could alter the $\dot{V}o_2$ of patients. These were not necessarily painful stimuli but involved minimal external stimulations, such as connecting the instrumentation for indirect calorimetry. In our study, the maximum frequency of defined events occurred during the time intervals from 6:00 to 9:00 AM and 3:00 to 9:00 PM. Although we excluded SvO_2 depressions due to manuevers like endotracheal suction or patient turning[12] from the evaluation, these are the time spans when most patient care is performed.

The measurement of a stable course of SvO_2 within the normal range might not reliably rule out a flow-limited regional oxygen consumption in septic shock patients. This was demonstrated by Dahn et al[33] using concurrent measurements of SvO_2 and splanchnic venous saturation in severely ill septic patients. While SvO_2 was normal (70.5 percent), the authors registered a reduced saturation of splanchnic venous blood (55.6 percent), which points toward an insufficient oxygen delivery to the splanchnic vasculature. Furthermore, the authors demonstrated a parallel course of SvO_2 and splanchnic venous saturation, where splanchnic venous saturation levels might track at substantially lower levels than SvO_2, especially during patient care maneuvers. Other authors have previously commented that, although patients with burns and sepsis have substantially increased splanchnic oxygen demand, the requirement generally is met by increasing regional blood flow as opposed to an increased oxygen extraction fraction.[34,35] However, Dahn et al[33] reported an extremely high splanchnic oxygen consumption up to 45 percent to 55 percent of total body oxygen consumption in septic shock patients. This fact, together with the observation that the higher total body or splanchnic oxygen consumption, the greater the likelihood that increased oxygen extraction becomes an important mechanism to maintain oxygen uptake, leads to a reduction in splanchnic venous oxygen saturation. If the assumption of Dahn et al[33] proves to be correct, all events with decreasing SvO_2 observed in our study might have been paralleled by a simultaneous event in splanchnic venous saturation at a lower saturation level. Since we found a significantly higher incidence of severe spontaneous SvO_2 alterations in nonsurvivors than in survivors (Table 3), we speculate that these patients may more often suffer from an inad-

equate oxygen delivery to consumption relationship.

Conclusion

Mixed venous oxygen saturation of patients suffering from septic shock is high-normal or even supranormal. However, short-term changes in SvO_2 do occur in septic shock patients. Since nonsurvivors exhibit a higher frequency as well as a significantly greater severity of sudden SvO_2 events, we recommend the installation of a computerized alarm-function for the automatic detection and indication of frequent events. When employed in conjunction with the other indicators of tissue oxygenation available in an ICU, SvO_2 can therefore be useful as a guide for both prognosis and urgency of therapy.[22]

In 33 percent of all registered events, we were unable to identify the therapeutic intervention or physiologic change possibly responsible for the sudden SvO_2 alteration. We speculate that the fluctuations in SvO_2 demonstrated in our study are often caused by fluctuations in tissue oxygen demand without a concomitant change in oxygen delivery. Further studies combining on-line SvO_2 monitoring with indirect calorimetry are warranted to complete the puzzle of SvO_2 and oxygen kinetics in septic shock patients.[23]

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