TITLE: OXYGEN SUPPLY DEPENDENCY IS NOT A CLINICALLY RELEVANT FINDING IN ADEQUATELY RESUSCITATED CRITICALLY ILL PATIENTS

AUTHORS: Lutz Hannemann, M.D.; Ralf Korell; Brigitte Kuss, M.D.; Konrad Reinhart, M.D.

AFFILIATION: Dept. of Anesth. and Surgical Intensive Care Medicine, Steglitz Medical Center of the Free University of Berlin, Hindenburgdamm 30, 1000 Berlin 45, FRG.

A "pathologic oxygen supply dependency" has been detected in patients suffering from ARDS or sepsis (1). This has been considered as indirect proof of a "covert" tissue oxygen debt and a poor prognostic sign. Bihari (2) demonstrated in his study that all patients whose VO_2 rose after an increase in oxygen delivery (DO_2) died, whereas this was not the case for patients in whom this did not occur. However, the patients in this study generally had a DO_2 of below 400/ml min/kg/m^2. Therefore we were interested in determining whether a "covert" tissue oxygen debt also exists in hyperdynamic critically ill patients and whether this phenomenon is related to patient survival.

Eighty-four patients requiring ventilatory support and PA-catheterization due to sepsis or acute respiratory failure were investigated after informed consent and approval by our local research committee. Before baseline measurements were obtained, all patients were hemodynamically stabilized with adequate volume loading (PCWP > = 15 mmHg) and inotropic support. If a dobutamine (Dob) dose of up to 16 mcg/kg/min was insufficient to achieve a mean arterial pressure (MAP) > =70 mmHg, norepinephrine (Nor) was added. Measurements were taken after patients had been hemodynamically stable for at least 30 min. The patients were then randomly assigned to 4 study groups. In group 1 patients, Dob or Dob + Nor was replaced by dopamine at a dosage titrated to achieve a similar MAP as with the previously applied catecholamines (n = 21). Dopexamine was added at a dosage of 2 mcg/kg/min (n = 22) in group 2 patients, PGI_2 at a dosage of 10 mcg/kg/min (n = 20) in group 3 patients, and hypertonic saline (HTS) 7.5% in 6% hydroxyethylstarch (HES) at a dosage of 2-4 ml/kg/min (n = 21) in group 4 patients.

After these changes in therapy, the DO2 of all patients increased from 732 ± 206 ml/min/m^2 to 859 ± 279 ml/min/m^2 ($p < 0,001$). The VO_2 increased in the survivors (n=46) from 150 ± 32 ml/min/m^2 to 160 ± 34 ml/min/m^2 ($p < 0,001$) but was in the same range as in the nonsurvivors (n=38), where it increased from 153 ± 31 ml/min/m^2 to 166 ± 35 ml/min/m^2 ($p < 0,001$) (Wilcoxon test).
We doubt that this less than 10% increase in VO_2 is indicative of a clinically relevant tissue oxygen debt. The higher rise in VO_2 after an increase in DO_2 that was demonstrated in other studies may have been due to inadequate resuscitation of these patients.

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TITLE : SYSTOLIC BLOOD PRESSURE VARIATION IN POSTOPERATIVE VENTILATED PATIENTS : A SENSITIVE INDICATOR OF LOW PRELOAD STATES

AUTHORS : M. Vrillon, M.D., F. Le Bret, M.D. J. Vrints, M.D., E. Barré, M.D., P. Coriat, M.D., P. Viars, M.D.

AFFILIATION : Department of Anesthesiology. Hôpital Pitié, 75651 Paris Cédex 13 - France

The evaluation of the effective intravascular volume is of great concern in patients admitted in the intensive care unit after a major vascular surgical procedure. As suggested by experimental studies (1), systolic pressure variation (SPV), the difference between maximum and minimum values of systolic blood pressure (SBP) following a single positive breath pressure, may be a fair indicator of hypovolemia. Accordingly, we quantify SPV immediately after a major surgical procedure to compare its reliability relative to other hemodynamic parameters to detect reduced preload states. Transesophageal echocardiography was used to measure end diastolic area (EDa) which was considered in this study as the reference index for left ventricular preload.

METHODS : 20 patients who underwent an abdominal aortic surgery were included in the study. All gave informed consent for the study after approval by our Ethics Committee. All these patients had a preoperative radionuclide ejection fraction (EF). Were excluded patients with EF lower than 50%. Postoperatively patients were admitted in the intensive care unit unresponsive under fentanyl sedation, with a SBP between 90 mmHg and 150 mmHg. At this time pulmonary capillary wedge pressure (PCWP), cardiac output and SBP were recorded. SPV was then determined.

In addition, a TEE short axis view of the LV was recorded. Retrospectively, three consecutive EDa were outlined and averaged by an independent "blinded" observer.

RESULTS : A fair correlation was found between EDa and SPV (r = 0.75). By constrast, no correlation was noted between EDa and either PCWP or cardiac index.

SPV was greater than 12 mmHg only in the 4 patients whose EDa was lower than 10 cm2. In patients whose EDa was greater than 10 cm2 SPV was lower than 11 mmHg.

DISCUSSION : Our study confirms the limitation of PCWP and cardiac index values in revealing post-operative hypovolemia. By contrast, in patients maintained under mechanical ventilation and fentanyl sedation after a major vascular procedure, SPV is an useful monitoring modality that is more sensitive that the other hemodynamic parameters in the diagnosis of latent hypovolemic states.

REFERENCE
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