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**DEVELOPMENT OF PERMEABILITY, HEMODYNAMICS AND GAS EXCHANGE IN SEPTIC vs NON-SEPTIC TRAUMA PATIENTS**

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**Introduction:** In severely injured trauma patients primary permeability defects can commonly be observed. However,ongoing disturbances of permeability may possibly be initiated by inflammatory responses (e.g. sepsis). Therefore we investigated changes in lung microvascular permeability (LMVP), extravascular lung water (EVLW), hemodynamics and gas exchange in ventilator dependend traumatized ICU patients, focusing on the development of septic complications.

**Patients and methods:** 31 artificially ventilated trauma patients (mean age: 35 yrs, ISS > 30) were investigated prospectively from the day of admission to the ICU for up to ten days. Time course of EVLW was measured by the thermal-dye dilution technique on a daily basis. At the same time, blood gas analysis and hemodynamic parameters [mean arterial pressure (MAP), cardiac index (CI), pulmonary microvascular pressure (Pmv)] were obtained. Pmv was derived from pulmonary artery pressure (PAMP) and pulmonary wedge pressure (PCWP) using standard equation. LMVP was determined I. immediately after admission and II 7 days after trauma by computerized gamma szintigraphy. Changes in LMVP are expressed as permeability index (LMVPI). According to their course of illness, patients were assigned to 2 groups: group A (septic) or to group B (non-septic).

**Results:**(table) 16 patients developed septic complications (group A). In that patients, elevated LMVPI and Pmv values already immediately after trauma could be noticed in contrast to non septic patients (group B; 15 pat.) . However, in all patients (A,B) late rises of LMVPI could be seen. Subsequent accumulation of EVLW only did occur in septic patients (B). These patients showed marked disturbances in gas-exchange and hemodynamics as well.

Table

| Time | I | | II | |
|---|---|---|---|---|
| Group | A | B | A | B |
| EVLW (ml/kgBW) | 7.8±0.7 | 7.2±0.7 | 10.5±0.9 | 5.2±0.4 |
| LMVPI (%/h) | 7.5±0.9 | 4.5±0.8 | 9.0±1.1 | 7.2±0.5 |
| CI (l/m2) | 4.8±0.4 | 4.1±0.5 | 5.6±0.6 | 4.0±0.6 |
| MAP (mm Hg) | 85.0±3.5 | 92.0±4.4 | 88.0±3.5 | 98.0±4.3 |
| $P_{mv}$ (mm Hg) | 19.9±2.4 | 12.9±2.3 | 22.7±3.0 | 12.8±4.2 |
| $PaO_2/FiO_2$ | 209.0±23 | 351.0±43 | 235.0±30 | 343.0±36 |

**Discussion and conclusions:** Our results indicate, that disturbances of vascular permeability can be observed very early in all trauma patients. However, in patients, developing septic complications (A), a marked hemodynamic response could be seen as well as pronounced edema formation and disturbances in gas exchange. However, apart from that, in a late observation state (>7 days), inflammatory responses must be assumed even in patients clinically considered not to be septic (B).

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**CARDIAC PERFORMANCE IN MULTIPLE INJURED ICU-PATIENTS WITH SEPTIC SYNDROME** F.G. Haslinger, W. Lingnau, N.J. Mutz

**INTRODUCTION:** Assessment of right ventricular ejection fraction (RVEF) was shown to be a sensitive indicator of right ventricular failure in septic patients. However, these data refere primarily to spontaneously breathing patients. Therefore it was the aim of our study to determine the development of RVEF in artificially ventilated septic ICU-patients, suffering from multiple trauma.

**PATIENTS AND METHODS:** We studied 23 multiple injured (including chest trauma) ventilated ICU-patients (x=30 yrs, mean ISS=19) developing septic syndrome. Using a modified indwelling multifunctional balloontipped catheter (RVEF Thermodilution Catheter ,Model 93A-431-H7,5F Edwards Lab. St. Ana CA.), RVEF as well as Cardiac Index (CI) were evaluated sequentially during 48 hrs at a hourly basis. At the same time, Stroke Volume Index (SVI) End-Systolic Volume Index (ESVI) and End-Diastolic Volume Index (EDVI) were determined.

**RESULTS:** (table)

| hour | 0 | 1 7 | 2 4 | 3 6 | 4 8 |
|---|---|---|---|---|---|
| RVEF(%) | 0.46±0.1 | 0.47±0.1 | 0.49±0.1 | 0.46±0.1 | 0.48±0.1 |
| CI(ml/m2) | 5.8±0.9 | 5.25±0.9 | 5.3±1.0 | 4.8±0.7 | 4.7±0.9 |
| SVI(-/-) | 52.6±9.8 | 53.2±11.8 | 56.4±9.6 | 46.75±9.8 | 53±9.7 |
| ESVI(-/-) | 63.4±19 | 59.3±12 | 60.7±21 | 54±18 | 61.8±27 |
| EDVI(-/-) | 116±21 | 112±17 | 117±24 | 102±19 | 113±35 |

**DISCUSSION AND CONCLUSIONS:** In multiple injured septic patients, cardiac performance will be changed during the first 48 hrs. : Whereas RVEF decreases, a marked EDVI rise does occur. Simultaneously decreasing ESVI enables CI to rise according patient's demand. This will indicate that in artificially ventilated trauma-patients similar mechanisms will take place like in septic internal patients, breathing spontaneously.

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# Cardiovascular assessment I

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**$SvO_2$ MONITORING IN SEPTIC SHOCK.** **M Belghith, S Nouira, JJ Lanore, JP Mira, I Hamy, B Renaud, F Brunet, J Dall'Ava, JF Dhainaut**

Present methods for detecting inadequate tissue oxygenation rely on repeated sampling of arterial and venous blood for measuring O2 saturation, cardiac output, and lactate levels. Although these methods may provide valable informations, the intermittent nature of the data is inadequate for following a hemodynamically unstable patient. Elevated SvO2 associated with increased lactate levels, as a sign of decreased O2 extraction, seems a common finding in untractable septic shock. However, this finding does not imply that SvO2 monitoring is useless in sepsis. Indeed, Heiselman [J Clin Monit 2: 237, 1986] observed that changes in SvO2 reflected changes in the O2 supply-to-demand ratio in a small group of pts with septic shock. The purpose of the study was to evaluate the usefulness of SvO2 monitoring (Oximetrix-3, Abbott; SAT-2, Baxter) in 11 pts (Age: 60 ± 20, SAPS: 21 ± 6) with septic shock as defined by Ziegler [N Engl J Med 324: 429, 1991]. The mean initial SvO2 value was 64%, with no difference between survivors and nonsurvivors. 10 hr later, survivors developed an increase in SvO2 while nonsurvivors did not (p<.01), despite volume expansion and gradual increase of vasoactive support to maintain adequate perfusion pressure and oxygen delivery (DO2). A great instability of SvO2 was only observed in nonsurvivors due to either a rapid decline in DO2 usually related to decréased cardiac output or an increase in oxygen consumption (VO2) related to pyrexia, shivening, agitation or endotracheal suctioning. No correlation was observed between SvO2, DO2, VO2 and lactate levels. All patients with SvO2 ≥75% evidenced an apparently normal VO2 and lactate levels. The classical hyperdynamic septic shock (high DO2, VO2, and lactate levels) was not observed in these pts. In this preliminary study, we conclude that SvO2 monitoring adequately reflects the acute variations in O2 supply-to-demand ratio, and may be a useful tool to monitor the effects of nursing and therapeutic interventions in unstable pts with septic shock.

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PROBLEMS WITH CONTINUOUS $SvO_2$ MONITORING.

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The use of oximeter tipped pulmonary artery catheters can considerably ease the management of critically ill patients. This is because they enable changes in cardiorespiratory performance to be rapidly detected. Initial attempts used two wavelength systems but these were found to suffer from considerable measurement error and drift and three wavelength catheters have become the norm. Early animal studies showed that the drift was small in the short term, and then about ± 3% per day.

There have been several studies in humans that have characterised the behaviour of the OPTICATH over the short term, and it is known that the catheter underreads at saturations of <50% in a non linear manner.

We have studied 30 patients who had OPTICATH( Oximetrix, Mountain View, CA) pulmonary artery catheters inserted as part of their clinical care. All catheters were calibrated in vitro according to the manufacturers instructions prior to insertion. Blood samples were drawn anaerobically from the distal lumen on a daily basis in order to confirm the calibration of the fibre optic system and analysed in a Radiometer OSM-3 6 wavelength spectrophotometer, standardised and operated according to the manufacturers instructions.

Regression analysis showed a highly significant correlation between the co-oximeter and the catheter (r=0.804, 95% C.I. 0.69 to 0.88, P<0.000001). However, there was considerable scatter of the error, SD=8.15, n=62. Daily standardisation of the fibre optic system reduced the scatter, SD=5.6, n=62, and improved the correlation (r=0.893, 95% C.I. 0.82 to 0.93). With daily recalibration the systems accuracy was tolerable. However, the drift of the system appeared unrelated to the duration of the catheter insertion, some catheters not drifting and others showing considerable errors.

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### INTRAOPERATIVE SHOCK AND ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS) AFTER LIVER TRANSPLANTATION: THE ROLE OF RIGHT VENTRICULAR FUNCTION

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The present study was performed in 9 adult cirrhotic patients who underwent orthotopic liver transplantation (OLTx), 4 of whom suffered from severe intraoperative hypotensive episodes and subsequent postoperative ARDS. We evaluated right ventricular (RV) contractility and the hemodynamic changes during all phases of OLTx (basal, hepatectomy, anhepatic, 5 min and 30 min after reperfusion, 24, 48, and 72 hours after OLTx) using a rapid response thermodilution pulmonary artery catheter (Swan-Ganz, 93A-431H-7.5F, Baxter). In addition to right ventricular ejection fraction (RVEF)(Vincent JL et al. Intensive Care Med 12:33 1986), we also studied time-independent maximum elastance ($E_{max}$)(Sagawa K: Circulation 63:1223 1981), expressed as the ratio between the end-sistolic pressure and the RV end-sistolic volume: MPAP/RVESV. All 4 pts before anhepatic phase had hypovolemic shock with a significant reduction of RVEF and $E_{max}$ ($p<0.05$, Student t test for paired data) and reduced systemic vascular resistance index (SVRI)($p<0.05$), which were corrected by blood transfusion and norepinephrine infusion. ARDS was subsequently observed 48 hours later in the same pts associated with increased pulmonary vascular resistance index (PVRI)($p<0.001$) causing a significant reduction of RVEF and $E_{max}$ ($p<0.05$). At this stage an inverted ratio ventilation (IRV) was applied associated with the continuous infusion of prostaglandins ($PGE_1$, 0.2-0.4 γ/kg/hr, PROSTIN VR, Upjohn) with rapid resolution of ARDS and improvement of right ventricular function.

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### LACK OF CORRELATION BETWEEN PULMONARY CAPILLARY WEDGE PRESSSURE AND LEFT VENTRICULAR PRELOAD IN SEPTIC SHOCK.

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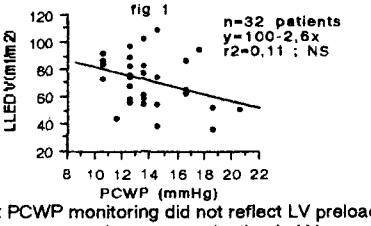
We compared Pulmonary Capillary wedge Presure (P.C.W.P) obtained by a Swan-Ganz catheter, and Left ventricular (LV) preload evaluated by two-dimensional echocardiography in 32 patients hospitalized for an acute episode of circulatory failure occuring in a septic context. Measurements of LV end-diastolic volume (LVEDV) were performed from one apical four-chamber view, applying the prolate ellipsoid, single-plane, area lenght formula. Measurements used for the study were simultaneously obtained when the patients were considred as hemodynamically stable, a status obtained by fluid loading (13 ml/kg of plasma expanders, 32 cases), Dopamine infusion (15±7 µg/kg/min 21 cases) and/or Dobutamine infusion (14±5µg/kg/min,21 cases). At this time a total lack of correlation between PCWP and LVEDV was observed (fig 1). Moreover LVEDV remained low in a subgroup of 7 patients despite fluid loading (42±7 cm3/m2) when compared with the 25 remaining patients (74±14cm3/m2) ; Whereas, PCWP was significantly higher in this subgroup (15±3 mmHg) compared with the remaining (13±2 mmHg) ($p<0.05$).




We concluded that PCWP monitoring did not reflect LV preload in septic shock, and we suggest that some reduction in LV compliance might occur in this context.

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### PULMONARY ARTERY MONITORING IS NOT ADEQUATE TO EVALUATE THE EFFECTS OF DOBUTAMINE ON RIGHT VENTRICLE IN COPD PATIENTS.

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The administration of catecholamines may alter the contractile sequence of the right ventricle (RV),preventing the normal transmission of intraventricular pressure into the pulmonary artery. In this study, we considered a group of acutely decompensated COPD patients and analyzed the effects of dobutamine (DBT) on the pressure wave transmission from RV to the pulmonary artery.

**METHOD**. Eleven COPD patients entered our ICU for ARF and were intubated and mechanically ventilated. Pulmonary artery was cannulated and a RV ejection fraction/volumetric Swan-Ganz catheter (Baxter) was placed for hemodynamic monitoring. Systolic pulmonary artery pressure (SPP) was measured as usual while the RV systolic pressure (RVSP) was obtained through the proximal port advancing the Swan-Ganz catheter after cardiac output determination. Pressure waves were recorded and data at the end of expiration was directly measured on the strip chart record. Measures were collected at basal and during the administration of DBT 10 ug/Kg/min. Student's t-test was used for statistical analysis.

**RESULTS**. The administration of DBT produced a non-significant increase in SPP from 31 ± 10 to 35 ± 13 mm Hg, while the RVSP rised significantly from 33 ± 10 to 46 ± 12 mm Hg [$p<0.001$]. At basal, the gradient between RVSP and SPP was negligible [ 2 ± 2 mm Hg; range -2;5 ] and did not always show the same direction. On the other hand, during DBT administration RVSP was constantly and significantly higher than SPP [ 11 ± 6 mm Hg; range 2;22; $p<0.02$ ]. Cardiac index increased from 2.5 ± 7 to 4.2 ± 1 L/min/m² [$p<0.001$] and stroke volume index from 31 ± 10 to 36 ± 11 ml/m²[n.s.].

**DISCUSSION**. In all subjects the administration of DBT caused the appearance of a systolic gradient between RVSP and SPP not evident at basal conditions. This gradient is an absolute limitation to the use of SPP in estimating RV intracavitary pressure. We conclude that in COPD patients, the use of pulmonary artery pressure monitoring to evaluate RV status during DBT administration, may be misleading.

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### ACCURACY OF THE MEASUREMENT OF TOTAL BLOOD VOLUME WITH THE COLD SYSTEM

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Bedside monitoring of total blood volume (TBV) has become possible with the recent introduction of an integrated fiberoptic monitoring system (COLD Z-021, Pulsion Medizintechnik, Munich, FRG), which calculates TBV from the blood concentration (C) of the nontoxic dye indocyaninegreen (ICG) within 4 minutes after injection. The aim of this investigation was to compare the COLD TBV estimate $TBV_{COLD}$ with the standard TBV measurement using Evans Blue (EB; $TBV_{EB}$) and additionally with TBV calculated by photometrically measured plasma concentration $C_{ICG}$ of ICG ($TBV_{ICG}$).

The experiments were performed in 11 anesthetized, paralyzed and mechanically ventilated pigs (mean body weight 31 ± 4 kg) during normovolemia, hypovolemia (blood withdrawal up to 50 ml/kg) and hypervolemia (retransfusion of the withdrawn blood plus 30 ml/kg 10% hydroxyethyl starch) and in 5 animals during additional β-stimulation (5-10 µg/kg/min dobutamine). A catheter for the injection of the indicator dyes EB and ICG was placed centralvenously, a fiberoptic thermistor catheter (4F PV 2024, Pulsion) was advanced into the aorta. Samples for the determination of $C_{EB}$ and $C_{ICG}$ were drawn from an arterial line in the femoral artery.

Linear regression analyses were applied for comparison of $TBV_{EB}$, $TBV_{ICG}$[1-16min := interval of sampling after injection and evaluation], and $TBV_{ICG}$[1-4min] with $TBV_{COLD}$ yielding the following equations:

$TBV_{COLD} = 0{,}78 \cdot TBV_{EB} - 30$ (ml), $r = 0{,}90$;

$TBV_{COLD} = 0{,}76 \cdot TBV_{ICG}$[1-16min] - 11 (ml), $r = 0{,}89$;

$TBV_{COLD} = 0{,}86 \cdot TBV_{ICG}$[1-4min] - 14 (ml), $r = 0{,}86$.

The results demonstrate, that $TBV_{COLD}$ correlates very well with $TBV_{EB}$ and $TBV_{ICG}$. $TBV_{EB}$ and $TBV_{ICG}$ are about 10-14% higher than $TBV_{COLD}$, if central instead of whole body hematocrit is used for calculation. Taking this fact into account, there still remains a systematic difference of about 10% between $TBV_{COLD}$ and both $TBV_{EB}$ and $TBV_{ICG}$[1-16min]. This difference occurs because determination of $TBV_{ICG}$[1-16min] and $TBV_{EB}$[2-17min] is based on longer mixing times of the indicators during which these also distribute in almost stagnant blood pools as spleen and the gastrointestinal tract. In conclusion $TBV_{COLD}$ represents actively circulating blood volume and qualifies for TBV bedside measurement.

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