

# Trauma

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MONITORIZATION OF THE ACUTE PHASE RESPONSE IN ISOLATED SEVERE CRANIO-ENCEPHALIC TRAUMA (CET): COMPARISON OF TUMOR NECROSIS FACTOR- $\alpha$  (TNF- $\alpha$ ), INTERLEUKYNE-6 (IL-6), C-REACTIVE PROTEIN (C-RP) AND ANTITRIPSINE 1- $\alpha$ .  
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## OBJECTIVE

The aim of this study was to establish the evolution pattern of (TNF- $\alpha$ ) and (IL-6) and their correlation with c-reactive protein (C-RP) and antitripsine 1- $\alpha$  (AT-1) in acute phase, in patients with isolated severe (CET).

## METHODS

20 consecutive patients, mean age 25 $\pm$ 2 years, 16 males, 4 females with severe isolated (CET) were followed in a prospective study. The mean initial Apache II was 17 $\pm$ 5, ISS 30 $\pm$ 8, GCS 6 $\pm$ 2.

Five determinations of TNF- $\alpha$  (IRMA n.v. < 25 pg/ml), IL-6 (IRMA n.v. < 10 pg/ml), C-RP and antitripsine  $\alpha$ -1 (immunonephelometry), were done at 12 hours intervals during 3 consecutive days.

After the first 24 hours of trauma, all patients were clinically stable, have the kind of ventilatory support and no one received exogenous administration of glucocorticoids.

All patients were in controlled mechanical ventilation without PEEP, enteral nutrition equivalent and similar therapy. There were no differences in the duration of intensive care and mortality rate. Statistical comparison of the results were performed with the chi<sup>2</sup> test. Correlation coefficients were calculated with linear regression analysis.

## RESULTS

Are shown in the following table

	TNF- $\alpha$ pg/ml	IL-6 pg/ml	C-RP mg/%	AT-1 mg/%
	0.54 $\pm$ 0.4	0.25 $\pm$ 0.2	1.08 $\pm$ 0.4	1.67 $\pm$ 0.5
	2.25 $\pm$ 0.8			
	20 $\pm$ 7	21 $\pm$ 9	45 $\pm$ 12	57 $\pm$ 20
	8 $\pm$ 4	9 $\pm$ 5	11 $\pm$ 5	13 $\pm$ 6
	15 $\pm$ 8			
	208 $\pm$ 38	295 $\pm$ 44	327 $\pm$ 50	371 $\pm$ 4
	388 $\pm$ 62			
	24 H. Post-CET	36 H. Post-CET	48 H. Post-CET	60 H. Post-CET
				72 H. Post-CET

IL-6 values increased early and reached maximum at 72 hours after trauma without any relation with TNF- $\alpha$  values. The TNF- $\alpha$  values were lower than the control ones. In all cases with very high serum concentration of C-RP and antitripsine  $\alpha$ -1 without the corresponding rise of TNF- $\alpha$  levels was also noted.

**CONCLUSIONS** This study suggests the possibility of an alternative pathway of IL-6 activation not related with TNF- $\alpha$  activity.

These preliminary results, also suggest the existence of a regulatory pathway for C-RP and antitripsine  $\alpha$ -1 independent of the TNF- $\alpha$  one. However, the size of the sample demand the continuation of the study to confirm the possible existence of alternative pathways.

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## SCORING OF ORGANFAILURE AS AN EARLY PREDICTOR OF OUTCOME IN MULTIPLE TRAUMA PATIENTS

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**Introduction:** Subsequent failure of organ systems is the most common cause of death in intensive care. The aim of this study was to investigate the value of the MOF score for predicting the outcome in patients after multiple injury.

**Methods:** From March 1 to November 15, 1991 we included 99 multiple trauma patients with an Injury Severity Score ISS  $\geq$  18. Patients were prospectively followed up with respect to occurring organ failure and death during intensive care. The daily sum of scoring points was calculated. Groups of survivors and nonsurvivors were compared by students t-test considering a  $p < 0.05$  as statistically significant.

**Results:** 36 patients (36.4%) died after a mean period of 8.5  $\pm$  1.8 days compared with survivors who had a mean stay of 9.4  $\pm$  0.7 days (n.s.). Survivors and non-survivors showed no difference of age (34.0  $\pm$  2.1 vs 39.5  $\pm$  6.6), but were significantly different in ISS (26.8  $\pm$  1.2 vs 40.5  $\pm$  3.1). MOF-Score in sum of all organ systems had significantly more counts in nonsurvivors during the whole period of treatment ( $p < 0.001$ ). In addition, the scores of the cardiovascular and the respiratory system showed significantly differences, whereas liver failure was not different during the first week.

**Conclusion:** Scoring systems are able to show early differences between survivors and nonsurvivors after multiple injury. The value for prediction of outcome in individuals is controversial and should be stated very carefully.

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## INCREASING SMOKE EXPOSURE INCREASES TISSUE OXYGEN DEMANDS IN THE EARLY POST BURN PERIOD

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Smoke inhalation injury is the main cause of morbidity and mortality in the burn victim. Advances in the treatment of cutaneous burn injury greatly exceeds the advances in the treatment of inhalation injury. Our purpose was to determine the effect of increasing smoke exposure on oxygen demands. We maintained filling pressures and adequate urine output, on oxygen delivery, DO<sub>2</sub>, oxygen consumption, VO<sub>2</sub>, and oxygen extraction, PVO<sub>2</sub>, in a mild smoke inhalation injury, COHgb 20% (n=6) vs a moderate smoke inhalation injury, COHgb 40% (n=6) both combined with a 15% third degree burn in adult sheep (40-50kg) monitored for 24 hrs post injury. Physiologic data is shown below.

	BASELINE		1 HOUR		6 HOUR		24 HOUR	
	20% COHgb	40% COHgb	20% COHgb	40% COHgb	20% COHgb	40% COHgb	20% COHgb	40% COHgb
C.I.	6 $\pm$ 6.2 $\pm$	6 $\pm$ 6.1 $\pm$	6.6 $\pm$ 5.9 $\pm$	6 $\pm$ 5.9 $\pm$				
l/min/M <sup>2</sup>	.3	.4	.4	.5	.4	.5	.4	.5
WEDGE mmHg	8 $\pm$ 8 $\pm$	7 $\pm$ 7.5 $\pm$	8 $\pm$ 8.2 $\pm$	8 $\pm$ 7 $\pm$				
VO <sub>2</sub> l/min/M <sup>2</sup>	250 $\pm$ 260 $\pm$	350 $\pm$ 450 $\pm$	260 $\pm$ 350 $\pm$	250 $\pm$ 380 $\pm$				
DO <sub>2</sub> l/min/M <sup>2</sup>	50	40	70*	100*	60	50*	60	50*
PVO <sub>2</sub>	700 $\pm$ 720 $\pm$	720 $\pm$ 700 $\pm$	600 $\pm$ 650 $\pm$	620 $\pm$ 610 $\pm$				
	50	70	70	50	70*	50*	40*	50*
	46 $\pm$ 39 $\pm$	39 $\pm$ 36 $\pm$	45 $\pm$ 38 $\pm$	45 $\pm$ 30 $\pm$				
	3	2*	2*	3*	3	2	2	2*

There was not a significant difference between filling pressures, urine output, or oxygen delivery between the two groups. There was, however, a significant increase in VO<sub>2</sub> at 24 hrs in the 40% COHgb group. Lung histology revealed minimal alveolar injury and a compared inflammation in both groups. However, airway injury was markedly accentuated with the increased smoke. We conclude that the additional smoke induced airways injury results in increased systemic oxygen demands, most likely due to mediators released from the injured airways.

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## INTESTINAL PERMEABILITY AFTER SEVERE TRAUMA AND HAEMORRHAGIC SHOCK IS INCREASED, WITHOUT RELATION TO SEPTIC COMPLICATIONS

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Polytrauma and haemorrhagic shock may initiate a cascade of events finally leading to ARDS and multiple organ failure (MOF). Altered barrier function of the gut, associated with bacterial translocation and endotoxaemia, is thought to play a major role in this cascade. Increased intestinal permeability has been shown to occur after thermal injury and was correlated with subsequent infections. We investigated whether gut permeability after severe trauma and shock is altered and studied its relation to septic complications and endotoxaemia.

**Methods:** 7 controls, 11 polytraumatized and 8 patients with a ruptured abdominal aortic aneurysm (AAA) were studied. Gut permeability was assessed on the second day after hospital admission by lactulose/mannitol assay. From eleven patients arterial blood was withdrawn at 0, 6, 24 and 48 hours from admission for endotoxin determination by a quantitative limulus assay. Patients were divided according to development of infectious complications.

**Results:** L/M ratio's were significantly increased in both patients groups as compared to controls (0.012  $\pm$  0.005): trauma 0.069  $\pm$  0.034 and AAA 0.098  $\pm$  0.093,  $p < 0.005$ , by Wilcoxon. No significant difference was found between nine infected patients (0.059  $\pm$  0.032) and ten patients without subsequent infections (0.101  $\pm$  0.080).

Endotoxaemia was demonstrated in 8 out of 44 blood samples and only so in blood taken at 0 and 6 hours from admission, but never in samples withdrawn at 24 and 48 hours, while in this period gut permeability was shown to be increased.

**Conclusions:** Gut permeability is markedly increased after trauma and shock, without a positive association to subsequent infectious complications. Endotoxaemia could only be demonstrated prior to, but not at the time of or after the measurement of increased permeability.

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**DEVELOPMENT OF PERMEABILITY, HEMODYNAMICS AND GAS EXCHANGE IN SEPTIC vs NON-SEPTIC TRAUMA PATIENTS**  
 M. M. Seyr, W. Furtwaengler, N.J. Metz

**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 dependent 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 1. immediately after admission and 7 days after trauma by computerized gamma scintigraphy. Changes in LMVP are expressed as permeability index (LMVP<sub>I</sub>). 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 LMVP<sub>I</sub> 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 LMVP<sub>I</sub> 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	
	A	B	A	B
EVLW (ml/kgBW)	7.8±0.7	7.2±0.7	10.5±0.9	5.2±0.4
LMVP <sub>I</sub> (%/h)	7.5±0.9	4.5±0.8	9.0±1.1	7.2±0.5
CI (l/m <sup>2</sup> )	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 <sub>mv</sub> (mm Hg)	19.9±2.4	12.9±2.3	22.7±3.0	12.8±4.2
PaO <sub>2</sub> /FIO <sub>2</sub>	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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## Cardiovascular assessment I

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**SvO<sub>2</sub> 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 O<sub>2</sub> saturation, cardiac output, and lactate levels. Although these methods may provide valuable informations, the intermittent nature of the data is inadequate for following a hemodynamically unstable patient. Elevated SvO<sub>2</sub> associated with increased lactate levels, as a sign of decreased O<sub>2</sub> extraction, seems a common finding in untractable septic shock. However, this finding does not imply that SvO<sub>2</sub> monitoring is useless in sepsis. Indeed, Heiselman [J Clin Monit 2: 237, 1986] observed that changes in SvO<sub>2</sub> reflected changes in the O<sub>2</sub> supply-to-demand ratio in a small group of pts with septic shock. The purpose of the study was to evaluate the usefulness of SvO<sub>2</sub> 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 SvO<sub>2</sub> value was 64%, with no difference between survivors and nonsurvivors. 10 hr later, survivors developed an increase in SvO<sub>2</sub> while nonsurvivors did not (p<.01), despite volume expansion and gradual increase of vasoactive support to maintain adequate perfusion pressure and oxygen delivery (DO<sub>2</sub>). A great instability of SvO<sub>2</sub> was only observed in nonsurvivors due to either a rapid decline in DO<sub>2</sub> usually related to decreased cardiac output or an increase in oxygen consumption (VO<sub>2</sub>) related to pyrexia, shivering, agitation or endotracheal suctioning. No correlation was observed between SvO<sub>2</sub>, DO<sub>2</sub>, VO<sub>2</sub> and lactate levels. All patients with SvO<sub>2</sub> ≥75% evidenced an apparently normal VO<sub>2</sub> and lactate levels. The classical hyperdynamic septic shock (high DO<sub>2</sub>, VO<sub>2</sub>, and lactate levels) was not observed in these pts. In this preliminary study, we conclude that SvO<sub>2</sub> monitoring adequately reflects the acute variations in O<sub>2</sub> 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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**CARDIAC PERFORMANCE IN MULTIPLE INJURED ICU-PATIENTS WITH SEPTIC SYNDROME** F.G. Haslinger, W. Lingnau, N.J. Metz

**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 refer 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	17	24	36	48
RVEF(%)	0.46±0.1	0.47±0.1	0.49±0.1	0.46±0.1	0.48±0.1
CI(ml/m <sup>2</sup> )	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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**PROBLEMS WITH CONTINUOUS SvO<sub>2</sub> 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<0.00001). 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=82, 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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