extracellular glucose concentrations. If this occurs in vivo, microbrial killing by neutrophils may be impaired in patients with acute hyperglycemia.

**P64 Mobilization of leukocytes in patients with severe sepsis and septic shock is associated with increased apoptosis, as detected by Annexin V binding**

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**Objectives:** To clarify whether mobilization of leukocytes during human severe sepsis and septic shock occurs independently of apoptosis.

**Methods:** Over a 6 month period, 33 patients with severe sepsis or septic shock were studied in the intensive care unit. Annexin V binding by leukocytes was determined daily using flow cytometry and FITC-labeled Annexin V. Transient leukocytosis, or peaks in leukocyte counts, were defined as individual increase at least 30% within 2 days, followed by a decrease of at least 30% within the following 2 days.

**Results:** Nine, 14 and 10 peaks in neutrophil, monocyte and lymphocyte numbers, respectively, were observed in 6, 9 and 10 patients; in all of these patients, increased Annexin V binding by neutrophils, monocytes and lymphocytes, respectively, by 69% up to 809% (median 215%), by 32% up to 973% (median 330%), and by 32% up to 4713% (median 224%), respectively, paralleled the neutrophil, monocyte and lymphocyte (in 13/14 episodes) peaks, respectively. During periods in which neutrophil, monocyte and lymphocyte numbers were stable, Annexin V binding was constant as well.

**Conclusions:** In conclusion, mobilization of leukocytes during severe sepsis and septic shock in critically ill patients is associated with increased apoptosis, as determined by Annexin V.

**P65 C5a receptor expression on leukocytes from patients with severe sepsis or septic shock**

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**Background:** Recently animal studies have been presented suggesting that complement factor C5a blockade might be of benefit in patients with severe sepsis/septic shock. In one study the expression of the C5a receptor (CD88) on the granulocytes from septic animals was increased. The aim of the present investigation was to study the CD 88 expression on leukocytes in human sepsis.

**Methods:** Twelve ICU patients fulfilling the ACCP/SCCM criteria for severe sepsis/septic shock were prospectively included into the study as early as possible in their septic course. Blood samples for analyses of leukocyte receptor expression and complement factors were taken on day 1, 3 and 15. The leukocytes were isolated from heparinised whole blood and labelled with CD88 antibodies. As controls leukocytes from 20 healthy individuals were used. The samples were analysed by the use of flow cytometry and results were presented as mean fluorescence intensity (MFI). The complement proteins C3a and terminal complement complex (TCC) were analysed in EDTA plasma by capture ELISA techniques. Levels of TNF-α, IL-6, IL-8, IL-10, IL-1ra, and MCP-1 were analysed in EDTA plasma with ELISA technique.

**Results:** On day 1 10/11 patients had increased levels of C3a, 1144 ± 138 ng/ml, (mean ± SE) (normal range: 92–268) and 11/11 of TCC 146 ± 46 AU/ml (normal range: 12–56). CD88 expression on the granulocytes in the control group was 63 ± 4. In comparison with the controls, the patients with severe sepsis/septic shock had significantly lowered values: on day 1 37 ± 5 (P < 0.001), on day 3 45 ± 8 (P < 0.05), and on day 15 51 ± 8 (P < 0.05). The granulocyte expression of CD88 on day 1 correlated negatively to APACHE-II score at inclusion (r = −0.59, P < 0.05). Besides a weak correlation to IL-1ra, there were no significant correlations to the other cytokines. In the patient group, the CD88 expression on the monocytes did not change during the observation time and did not differ from that in the control group.

**Conclusion:** Although a transient increase at an earlier stage of the septic course cannot be excluded, our results demonstrate that the expression of the C5a receptor on granulocytes — at the time when diagnosis can clinically be made — is low, despite an activation of the complement system. Our result suggest that C5a blockade in human sepsis might be of a more limited value than that found in animal experiments.

**P66 Terminal complement complex in porcine septic shock with substantial capillary leak syndrome**

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**Introduction:** In septic shock with capillary leak syndrome (CLS), it has been suggested that hemodilution, and capillary leakage of protein may account in part for reduced levels of complement pro-

**References:**