Stable PO₂ during Hemofiltration in Spite of a Drop in Leucocytes

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Dear Sir,

The pathogenesis of dialysis-induced hypoxemia is still not fully clarified [1]. Several mechanisms have been proposed, of which however only two deserve special attention. Craddock et al. [2] have emphasized the importance of the initial leucocytopenia for the development of the hypoxemia. These authors suggested that the blood-membrane interaction during hemodialysis (HD) induces a sequestration of leucocytes in the pulmonary vascular bed, which would then impair the oxygen diffusion. On the other hand, Aurigemma et al. [3] have claimed that the loss of carbon dioxide during acetate HD, which they regularly observed, when they measured the PCO₂ in the inlet and outlet of the dialyzer might be the crucial pathogenetic factor causing hypoxemia. They concluded that the resulting hypocapnia would lead to hypoventilation and thereby to a consecutive fall of the arterial oxygen tension. As recent investigations have shown that a decrease of the PO₂ will not occur during a hemofiltration (HF) treatment [4], we studied the PO₂ and the leucocyte counts at different times in 6 patients being chronically treated by conventional postdilution HF. Furthermore, in order to evaluate possible influences of different membranes, the studies were conducted with two different hemofilters. During the first investigation a capillary filter with polyamide fibers was applied (Gambro® fiber hemofilter FH 202), for the second study we used a high-flux dialyzer with a cuprophane membrane (Gambro® Lundia Major High Flux 1.36 m²).

The results, which are summarized in figure 1, clearly show that patients on both hemofilters maintain a stable PO₂ throughout a HF treatment. However, whereas the polyamide filter did not induce any fall in the leucocytes, such a drop could be regularly observed when applying the cuprophane filter. The differences observed at 30 min are highly significant ($2\alpha < 0.01$, Wilcoxon test).

Although these results, which were obtained during HF treatment, are not necessarily transferable to HD, they indicate that the leucopenia, which is observed after the onset of HD as well as HF, when cuprophane membranes are used, is not very crucial for the development of the hypoxemia. It appears rather likely that the loss of PCO₂,

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Polyamide Cuprophane

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Fig.1. Percentage change of $p < \frac{3}{4}$ and leucocytes during hemofiltration. B = Begin; E = end. n = 6, $2\alpha < 0.01$. Data represent mean values $\pm$ SD.

which is eliminated during HF, plays the dominant role in dialysis-induced hypoxemia.

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