In adults, it is well known that hypoxemia in acetate hemodialysis (AH) with a conventional membrane is secondary to two main mechanisms: blood-membrane interaction followed by reversible lung damage \([1,2]\) with hypoxemia shortly after initiation of the extracorporeal blood circulation on the one hand, and hypoventilation by carbon dioxide loss through the dialyser \([3, 4]\), with hypoxemia later in the session on the other hand. In children, the effects of acetate (acetate 35 mmol/l, Na 140 mmol/l) hemodialysis with a high-permeable membrane \([5]\), on acid-base status, are not documented.

Four children (mean weight 24 ± 1.2 kg) were studied during twelve AH with a high-permeable membrane (F 40, polysulfone, 0.65 m², Fresenius) (fig. 1). There is no hypoxemia before the first hour of session time. All children have carbon dioxide loss, but only a few have hypoxemia. So, two groups of sessions versus AH can be isolated: group I \((n = 6)\): carbon dioxide loss with hypoxemia (significant fall in \(pO_2\), with a minimal drop at 70%); group II \((n = 6)\): carbon dioxide loss with acidosis and normoxemia (no significant fall in \(pO_2\), with a maximal drop at 95%). The \(p50\), e.e. the \(pO_2\) at which hemoglobin is half saturated with oxygen at \(pH 7.40\) is assessed at the beginning \((0 \text{ min})\) and the end \((180 \text{ min})\) of the session. There is (fig. 1) a significant increase of \(p50\) in the group II. All the blood samples are drawn from the arterial blood of the A-V fistula, and measurements are made on the whole blood using an automated apparatus (cooxy-meter, tonometer).

AH with a high-permeable membrane induced not only carbon dioxide loss in children, but also acid-base

\[
\begin{align*}
\text{%} & \\
100-90 & - 80-70-60-\% \\
100-90 & - 80-70-60-\% \\
7.32-7.35 & - 7.38 \cdot 7.41 - 7.44-7.47 \cdot 7.50
\end{align*}
\]
Fig. 1. Effects of AH on pCO₂, pO₂, pH and p50. pCO₂ and pO₂ are expressed as a percentage of the predialysis value to reduce the effects of the variability between patients. Results are given as mean ± SEM at selected intervals after beginning of dialysis. Blood flow (120 ml/min) and dialysate flow (500 ml/min) are the same for (6 values).

All children. °,* Significance (Student’s t test, p < 0.001) within groups in respect to O min, and between groups at the same time, respectively. Group I = carbon dioxide loss, with hypoxemia (6 values); group II = carbon dioxide loss, acidosis, normoxemia. Variation. In group I, the carbon dioxide loss is not associated with acidosis, so ventilation depression occurred with hypoxemia. In group II, carbon dioxide loss is masked by severe acidosis which stimulated the ventilation, so hypoxemia did not occur. This fall of pH with ventilatory stimulation (superior to the depression of ventilation caused by the fall of pCO₂) is also proposed in hemofiltration (where high-permeable membranes are used) to explain stable pO₂ [6]. This fall of pH is the result of the net base equilibrium during the treatment time (sum of acetate administered and metabolized, minus bicarbonate and organic anion losses from the blood to the dialysate). The p50, a blood-to-tissue oxygen transfer, is better in group II; the combination of pO₂ and pH variations induced an increase in p50. Only in bicarbonate hemodialysis (and not in acetate hemodialysis) with conventional membranes in adults, an increase of p50 is demonstrated [7]. These potential variations (pO₂ and p50) are of course one of the factors for good or bad intradialytic tolerance to AH in children. If high-permeable membranes are used in children, various effects of AH on acid base-status are produced: carbon dioxide loss on one hand, acidosis (with decreased hemoglobin affinity to oxygen) or hypoxemia on the other. This two different biological statuses must be known for adequate treatment of intolerance symptoms in children during the session (respectively bicarbonate or oxygen administration).

References


