a separate section of our unit was reserved for positive patient use only;
a suitable number of dialysis machines was reserved for the same patients;
a rest room and a check room were reserved for positive patients as well;
a dialysis machine was reserved for patients not yet screened (usually new patients or acute-phase patients);
guest patients were accepted only if recently tested for HCV antibodies.

The patient population varied between 44 and 54 during the study. Dialysis length varied between 3 and 4.5 h and no filter was reused. All patients were tested monthly for alanine transferase. Statistical analysis was performed using chi-square test. The policy regarding basic precautions, such as cleaning and disinfection of surfaces and machines, use of gloves and no sharing of tools among patients, did not change before and during our study.

The number of new seroconversions was zero in 1992, in 1993 and in 1994. Seroprevalence fell to 18% in 1992, 13% in 1993 and 7% in 1994 because of transfer, transplantation, or death of seropositive patients. The rate of transfusion, defined as the number of blood transfusions per patient and per year, did not change significantly during our study. It was 1.1 (56/51) in 1991, 1.7 (73/44) in 1992, 0.8 (37/45) in 1993 and 1.2 (64/54) in 1994. The prevalence in haemodialysis staff was zero.

Compared with the gold standard, the use of reverse-transcription polymerase chain reaction (RT-PCR), to detect the presence of viraemia in serum, second generation assays are not ideal for diagnosing HCV infection. Nevertheless, they are economical and are effective to detect infected patients. Our procedure does not guarantee absolute protection against HCV infection, because it does not exclude the risk arising from negative patients in early phase of infection or those recently transfused. However, the incidence of new seroconversions was zero from the beginning of the infection control procedure to September 1995.

An infection control strategy based on the identification and isolation of positive patients, screening of blood donors and erythropoietin therapy, is successful in hindering the spread of HCV infection. Nevertheless, they are economical and are effective to detect infected patients. Our procedure does not guarantee absolute protection against HCV infection, because it does not exclude the risk arising from negative patients in early phase of infection or those recently transfused. However, the incidence of new seroconversions was zero from the beginning of the infection control procedure to September 1995.

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levels > 30 mg/dl and four of them (57%) suffered vascular disease. The interpretation of these rather interesting results concerning the correlation of altered Lp(a) levels during HD and vascular disease needs further investigation based on the Apo(a) phenotype [7].

Our findings that are in agreement with those of Schumacher et al. [3], indicate that the HD procedure seems to worsen the abnormal Lp(a) metabolism in haemodialysis patients, possibly due to cyclic Lp(a) transportation from the blood vessels wall deposits. We have not studied the possible role of heparin administration (unfractionated and low-molecular-weight) on Lp(a) alterations during haemodialysis, but as far as we know there is no study reporting similar effect. Furthermore, recent reports provide strong evidence that many cytokines can alter lipid metabolism, indicating a possible role of the immune system in atherosclerosis [8,9]. The HD procedure results in cytokines release and IL-6 has been reported to be an activator of the synthesis of acute-phase proteins by hepatocytes [5]. Since Lp(a) has been considered as an acute-phase protein [10], the possible influence of cytokines on Lp(a) alterations during haemodialysis needs further investigation using a larger group of patients haemodialyzed with more biocompatible membranes.

### Table 1. Uncorrected and corrected mean (± SD or range) plasma lipids, apolipoproteins and Lp(a) levels post-HD in comparison to the pre-HD values

<table>
<thead>
<tr>
<th></th>
<th>Pre-HD</th>
<th>Post-HD</th>
<th>P</th>
<th>Corrected</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Uncorrected</td>
<td>Corrected</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>195 ± 41.22</td>
<td>213.3 ± 42.1</td>
<td>&lt;0.02</td>
<td>195 ± 36.1</td>
<td>NS</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>123 ± 49.42</td>
<td>139 ± 59.5</td>
<td>NS</td>
<td>121 ± 51.3</td>
<td>NS</td>
</tr>
<tr>
<td>HDL</td>
<td>42.0 ± 10.15</td>
<td>49.0 ± 10.3</td>
<td>&lt;0.001</td>
<td>44.7 ± 9.76</td>
<td>NS</td>
</tr>
<tr>
<td>LDL</td>
<td>131.5 ± 36.05</td>
<td>136 ± 35.4</td>
<td>NS</td>
<td>128.6 ± 34.9</td>
<td>NS</td>
</tr>
<tr>
<td>Apo A1</td>
<td>165.7 ± 45.27</td>
<td>178 ± 31.9</td>
<td>NS</td>
<td>164 ± 30.1</td>
<td>NS</td>
</tr>
<tr>
<td>Apo B</td>
<td>113 ± 39.35</td>
<td>120 ± 32.2</td>
<td>NS</td>
<td>109 ± 27.9</td>
<td>NS</td>
</tr>
<tr>
<td>Lp(a)</td>
<td>15.4 (4–128)</td>
<td>30 (4–200)</td>
<td>&lt;0.05</td>
<td>27.4 (3.6–174)</td>
<td>&lt;0.05</td>
</tr>
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All results in mg/dl.

Serum amyloid A protein levels in haemodialysis patients

Sir,

Long-term deleterious consequences of haemodialysis (HD) have caused much concern during the past few years. After the description of a syndrome now known as dialysis arthropathy, it has become evident that an increasing proportion of patients may develop problems, especially related to joints [1]. Indeed, haemodialysed patients on treatment for more than 15–20 years have signs of arthropathy. The pathogenesis of this condition has become better understood since it was observed that a great proportion of patients suffering from arthropathy had amyloid deposits consisting of β₂-microglobulin (β₂M), especially in joints and periarticular tissues. The exact nature of how and why β₂M is precipitated remains obscure [2]. Serum amyloid A (SAA) is one of the most sensitive low-molecular-weight acute-phase proteins, produced primarily by the liver as a result of tissue injury, infection, or inflammation. SAA is secreted in monomeric forms and associates with high-density lipoprotein, but its physiological function is not known. It is taken up by various tissues and degraded by cells of the mononuclear phagocyte system.

In the present study we investigated serum levels of SAA, β₂M, and creatinine, and compared them with diatylic age in haemodialysis patients. This study was performed on 18 (10 male, 8 female aged 33.2 ± 4.3 years) end-stage renal disease (ESRD) patients treated by HD (mean dialytic age 42.3 months). Dialysis was performed twice or three times per week for 4 h with polysulphone dialysers. All patients were given calcitriol (0.25 μg/day), rHuEpo, and other medications as required. Electromyographs of all patients were taken. SAA was measured by a commercial human SAA immunoassay kit (Cytoscreen, BioSource, USA) with ELISA