

## W-1-1

### PLASMAPHERESIS IN INFLAMMATORY POLYNEUROPATHY

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From Nov. 1993 to Nov. 1995, there were 12 cases of recognized inflammatory polyneuropathy treated with plasmapheresis (PP) at our hospital. These include 6 cases of acute inflammatory demyelinating polyneuropathy (AIDP), 4 cases of chronic inflammatory demyelinating polyneuropathy (CIDP), one case of Fisher syndrome and one case of Sjogren syndrome. The patients chosen for PP were under the inclusion criteria of severely disabled, i.e., Grade 4 (bed or chair bound) or Grade 5 (required assisted ventilation), or unremittingly progressive course. Patients of CIDP, Fisher syndrome and Sjogren syndrome were also receiving corticosteroids when PP was conducted. Plasmapheresis was carried out by the double filtration method. The mean flow rate of the plasma was 20 ml/min, and approximately 2.5-3.0 L of plasma was treated in each plasmapheresis treatment. Each patient received a standard course of at least 4 sessions of pheresis. Clinical measures, such as time from onset of disease to time of PP (onset-PP), clinical status (grade) at time of PP, number of sessions of PP, use of corticosteroids, time of recovery, i.e. time for patients to reach Grade 2 (PP-G2) or Grade 4 if patients were on a respirator (PP-G4). PP was judged effective when patients reached Grade 2 within 2 months or were extubated within 7 days in the case of Grade 5. All the patients receiving PP showed beneficial response. Eight patients were judged effective, the other two patients (one AIDP and one CIDP), though improved, were unable to reach Grade 2. Among the complications of PP, only 3 (33%) episodes of symptomatic hypotension were noted in a total of 78 sessions of PP. In conclusion, PP is safe and effective in treating inflammatory polyneuropathy.

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### THE ROLE OF IMMUNOADSORPTION PLASMAPHERESIS IN CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY

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Chronic inflammatory demyelinating polyneuropathy (CIDP) is characterized by progressive muscle weakness due to involvement of the peripheral nerve myelin. Although the cause of CIDP is unknown, present evidence favors a cell-mediated autoimmunity against peripheral nerve myelin. There are increasing evidences that preferential activation of Th1 cell response is central to the pathogenesis of this disease. Six patients with CIDP were treated with at least 4 sessions of immunoadsorption plasmapheresis (IAPP) by using Immusorba PH-350 or TR-350 (Asahi Medical) as an adsorbent column. Combined therapy of IAPP and corticosteroid resulted marked improvement. The function of helper T cell subtype (Th1 and Th2) was analyzed by enzyme-linked immunospot (ELISPOT) assay. Before IAPP, interferon  $\gamma$ , which was produced by Th1 cells, were dominant. After IAPP, IL-4, which was produced by Th2 cells, were dominant. These results suggest that IAPP therapy may remove the disease causative factors and change the T cell subtype function from Th1 dominant to Th2 dominant. These effects may be very important to improve the immunologic state and the suppress the disease activity in CIDP.