F.R.G.) of $r = 0.95$, as well as between the former method and a radioimmunoassay method (CIS-SORIN, Biom-edicina, 13040 Saluggia, Italy) of $r = 0.99$.

The lack of correlation between serum ferritin and serum iron on one hand and the inverse relationship between serum ferritin and iron-binding capacity on the other, reported by Fortier et al. (1) and by Lipschitz et al. (2), has also been reported by us in patients on chronic hemodialysis and after kidney transplantation (3, 4). Figure 1 shows the inverse correlation between serum ferritin (IRMA method, Hoechst-Behringwerke) and transferrin measured immunoturbidimetrically with a centrifugal analyzer (ENI-GEMSAEC; Electro-Nucleo,neen, Fairfield, NJ 07006) in 192 patients on chronic dialysis and after kidney transplantation when the data are plotted semilogarithmically as presented by Lipschitz et al. (2) and Fortier et al. (1). We found that the equation $y = (b/x) + a$, a hyperbolic function, gave the best coefficient of correlation (Figure 2). The same hyperbolic relationship was found between serum ferritin and intestinal iron absorption and is also well known for creatinine and inulin clearance (5). Figure 2 seems to indicate that ferritin is the better discriminator in iron deficiency and iron overload, which seems logical because transferrin is a transport protein and therefore cannot reach a zero value, whereas ferritin is a storage protein and therefore closely related to the actual iron content.

References

Horst D. Koehn
Günter Wider
Peter M. Bayer
Adolf Mostbeck
Institut f. Nuklearmedizin und Zentralabteilung, Wilhelminenspital Vienna, Austria

**IGD Myeloma or δ Heavy-Chain Disease?**

To the Editor:

I read with interest the Case Report of an unusual IGD myeloma (1). Cases of monoclonal IGD with "unreactive" light-chain determinants, although previously reported (2), should be thoroughly studied to rule out a yet-unreported δ heavy-chain disease.

So far, only γ, α, and μ heavy-chain diseases have been described (3–5). These diseases were helpful in elucidating the structure and genetic control of immunoglobulins (6). The clinical features of the three kinds of heavy-chain diseases are quite different. It will be interesting to know whether the clinical findings of the postulated δ heavy-chain disease resemble other heavy-chain diseases or multiple myeloma. The fact that a δ heavy-chain disease case has not yet been found could be due to the scarcity of IgD-secreting cells in humans. Indeed, only about 150 cases of IgD myeloma have been reported so far (7). It is also likely that such cases have not been recognized.

Every effort should be made to characterize a monoclonal IgD with respect to light-chain type. About 90% of monoclonal IgD are of λ type, and A light chains are often difficult to identify with the commonly available antisera. The following case report illustrates this fact.

A 41-year-old woman presented to the hospital with pain in her back and rib cage. She was anemic (hemoglobin 116 g/L, hematocrit 35%), and her serum calcium (147 mg/L) and blood urea nitrogen (800 mg/L) concentrations were above normal. Serum protein electrophoresis in agarose gel showed a faint, homogeneous band in the γ region. Multiple osteolytic lesions were found on roentgenographic examination, and a smear of bone marrow aspirate revealed 50% abnormal plasma cells. Immunoelectrophoresis revealed an IgD arc with a monoclonal configuration.

The IgD concentration was 1.48 g/L (normal < 0.2 g/L). Both the serum and the urine contained monoclonal, free λ light chains (λ Bence Jones protein) that migrated in the β region on agarose-gel electrophoresis.

Repeated immunoelectrophoretic analyses failed to show any reaction of the IgD with several antisera to κ and λ light chains. Column-chromatographic isolation of the IgD did not help to identify the light-chain type. The possibility of a δ heavy chain was ruled out by analytical ultracentrifugation, which showed a 7 S homogeneous protein, and by immunoelectrophoresis in sodium dodecyl sulfate, which did not show evidence of free heavy chains or their fragments.

In routine immunoelectrophoretic analysis the serum is often reacted with anti-κ and anti-λ sera and with either a polyvalent antisera (anti-IgG + IgA + IgM) or specific antisera to the three major immunoglobulins. In such assays the presence in serum of even large amounts of δ or ε heavy chains would be always unrecognized. Perhaps routine testing of serum with anti-IgD and anti-IgG sera in double-diffusion analysis (which requires less anti-serum) than conventional immunoelectrophoresis would document a δ or ε heavy-chain disease.

References
Is Serum or Heparinized Plasma Better for Assay of Aminotransferases?

To the Editor:

In recent years standardization of the determination of the catalytic activities of enzymes occurring in serum has been an important problem in clinical chemistry. In 1972 the German Society (1) and in 1974 the Scandinavian Society (2) published recommendations for the determination of the catalytic activities of aspartate and alanine aminotransferases.

The expert panel on enzymes of the Committee on Standards of the IFCC also described an provisional recommendation for the determination of aspartate aminotransferase (3, 4) (L-aspartate:2-oxoglutarate aminotransferase, EC 2.6.1.1; AST).

Bergmeyer et al. (5) published in 1978 an extensive study on the optimization of methods for AST and alanine aminotransferase (L-alanine:2 oxoglutarate aminotransferase, EC 2.6.1.2; ALT). Their assay conditions for determining the catalytic concentration of AST in human serum are in full agreement with the provisional recommendation of the expert panel on enzymes (3, 4).

Some years ago we investigated the influence of heparin on the estimation of aminotransferases (6), and showed that, although heparin scarcely affects the AST determination, the ALT activity in plasma is about 70% of the activity in serum, when the enzyme determinations were carried out according to the Scandinavian recommendations (2). We have now investigated the influence of heparin on the assay of AST and ALT, using the methods described by Bergmeyer et al. (5).

Blood was taken from normal individuals by venipuncture. After centrifugation, we assayed the plasma (heparinized; 25 kilo-int. units/L blood) or serum sample as soon as possible for aminotransferase activities. All catalytic activities were determined at 340 nm in a spectrophotometer (Model 25; Beckman Instruments, Inc.) at 30 °C. The assay conditions are described in Table 1. The reaction was started by addition of 2-oxoglutarate, and the decrease in absorbance at 340 nm was read every minute for 5 min. All catalytic activities were calculated from the mean change in absorbance during the first 5 min and expressed as U/L.

Table 2 shows the activities of AST and ALT in plasma and serum determined according to the Scandinavian recommendations and by the assay methods described by Bergmeyer et al. As far as the Scandinavian recommendations are concerned the results obtained for the measurement of the catalytic activities of AST and ALT are quite the same as published earlier (6): the ALT activity in plasma is lower than that in serum and, although there is a significant difference in catalytic activity of AST between serum and plasma, this difference does not seem to be clinically important. When, however, the assay methods of Bergmeyer et al. are used, there is no difference in catalytic activity of AST and ALT, whether in plasma or serum; thus, either sample source can be used to determine the catalytic activities of the aminotransferases.

It is not totally clear to us why there is this difference in results between the Scandinavian recommendations and the method described by Bergmeyer et al. The volume fraction of sample is lower in the assay method of Bergmeyer et al., which means a lower concentration of globulin. The concentrations of buffer and L-alanine are also higher than in the Scandinavian method, which means the ionic strength will be higher. Possibly the hydration shell has been changed to such an extent that no turbidity can arise when plasma is used for measuring AST activity.

References