heart failure. There is no doubt that this mass was due to the Waldenstrom's macroglobulineamia as IgM was detected on histological examination of the tumour.

Discrete soft tissue masses are most uncommon in Waldenstrom's macroglobulinaemia, but pulmonary tumours have been described.[2-4] They are usually accompanied by other signs of the disease such as lyphadenopathy, hepatomegaly, splenomegaly and increased serum IgM concentrations.[4] Cardiac disease caused by tumour has not previously been reported. Our patient was unusual in that he had no bone marrow or extramedullary lymphoid tissue metastases at the time of death despite massive tumour load in the heart. In addition, the disease was in remission biochemically as shown by the persistently low monoclonal IgM. This may have been an atypical form of the disease in which IgM was synthesised by the tumour but not secreted into the circulation.

To our knowledge, cardiac disease with tumour metastases in the cardiac tissue has not been described previously in a patient with Waldenstrom's macroglobulinaemia. This possibility should be considered as an alternative to hyperviscosity or amyloidosis as a cause for heart failure in Waldenstrom's macroglobulinaemia.

L A BRAWN
M A PARSONS*
P ALLAMBY*
L E RAMSAY
F E PRESTON†
*Departments of Therapeutics, *Pathology, and †Haematology, Royal Hallamshire Hospital, Sheffield, S10 2RX*

References

1 Deuel TF, Davis P, Avioli LV. Waldenstrom's macroglobulinaemia. *Arch Intern Med* 1983; **143**:986–8.
2 Neiman HL, Wolson AH, Berenson JE. Pulmonary and pleural manifestations of Waldenstrom's macroglobulinaemia. *Radiology* 1973;**107**:301–2.
3 Winterbauer RH, Riggins RCK, Griesman FA, Bauermeister DE. Pleuropulmonary manifestations of Waldenstrom's macroglobulinaemia. *Chest* 1974;**66**:368–75.
4 Rausch PG, Herion JC. Pulmonary manifestations of Waldenstrom's macroglobulinaemia. *Am J Hematol* 1980;**9**:201–9.
5 Forget BG, Squires JW, Sheldon H. Waldenstrom's macroglobulinaemia with generalised amyloidosis. *Arch Intern Med* 1966;**118**: 363–75.

Unchanged concentrations of plasma fibronectin in Alzheimer's disease

In Alzheimer's disease the capacity to remove intracellular and intercellular debris is considered to be impaired.[1,2] It is also claimed that abnormalities attributed to Alzheimer's disease can often be observed in peripheral tissues including skin fibroblasts and the blood.[3] It was therefore thought that an analysis of plasma in relation to repair and maintenance systems might be useful to develop simple tools for the diagnosis of Alzheimer's disease.

In man the concentration of fibronectin in the plasma increases exponentially with age.[4,5] Such changes in plasma fibronectin concentrations are usually associated with the changes in its rates of synthesis, changed proteolytic breakdown, and inefficient scavenging systems.[3,4] Some age related diseases, such as diabetes and atherosclerosis, show prematurely increased concentrations of plasma fibronectin.[6] There is, however, no report on the concentrations of fibronectin in the plasma of patients with Alzheimer's disease. We therefore estimated plasma fibronectin concentrations in such patients.

Plasma samples from seven patients with Alzheimer's disease (six women and one man, aged between 55 and 81 years) were kindly provided by Dr J Vijg, TNO Institute for Experimental Gerontology, Rijswijk, The Netherlands. The clinical diagnosis of the disease in these patients was made by Dr P Eikelenboom (Valerius Clinic, Amsterdam) and was found to be in accordance with the diagnostic criteria for "possible senile dementia of Alzheimer's type", as described by McKhann *et al.*[7] The number of years for which these patients have now been under observation is between seven and 14.

Plasma fibronectin concentration (mg/l) was determined in these samples by a double antibody sandwich ELISA technique, using rabbit anti-human fibronectin antibody as the catching antibody and the same conjugated to horseradish peroxidase as the secondary antibody. For comparison, plasma fibronectin concentrations were also determined in a section of the normal Danish population. For this, venous blood samples from more than 90 apparently healthy volunteers aged between 20 and 82 years were taken in dipotassium edetic acid and using standard methods. Concentrations of fibronectin in plasma were determined as described above. All estimates were made simultaneously in multiple replicates and repeated twice at different times.

In an apparently normal section of the population, an age related exponential increase (from 200 mg/l at 20 years of age to more than 600 mg/l at 80 years) was observed, which is similar to previous estimates.[4,5] The increase observed was significant ($2p < 0.01$), and the extent of the increase was slightly less in men than in women. Patients with Alzheimer's disease had unchanged concentrations of plasma fibronectin compared with their age and sex matched normal counterparts. Many other biochemical processes, such as calcium homeostasis, DNA and protein synthesis, and DNA repair, seem to be changed in Alzheimer's disease, and these changes can be identified in the peripheral tissues.[3,8] Our studies show, however, that impairment of the scavenging system in the nervous tissue of patients with Alzheimer's disease does not occur generally throughout the body.

S I S RATTAN*
L M RASMUSSEN†
P BJERRING‡
P BHATIA*
B F C CLARK*
**Laboratory of Cellular Ageing, Department of Chemistry, Aarhus University; †Institute of Pathology, mune Hospital; ‡Marselisborg Hospital, DK-8000, Aarhus-C, Denmark*

References

1 Selkoe DJ, Bell DS, Podlisny MB, Price DL, Cork LC. Conservation of brain amyloid proteins in aged mammals and humans with Alzheimer's disease. *Science* 1987;**235**:873–7.
2 Wurtman RJ. Alzheimer's disease. *Scientific American* 1985;**252**:48–56.
3 Peterson C, Goldman JE. Alterations in calcium content and biochemical processes in cultured skin fibroblasts from aged and Alzheimer donors. *Proc Natl Acad Sci USA* 1986;**83**:2758–62.
4 Eriksen HO, Clemmensen I, Hansen MS, Ibsen KK. Plasma fibronectin concentration in normal subjects. *Scand J Clin Lab Invest* 1982;**42**:291–5.
5 Labat-Robert J, Robert L. Modifications of fibronectin in age-related diseases: diabetes and cancer. *Arch Gerontol Geriatr* 1984;**3**: 1–10.
6 Labat-Robert J, Leutenegger M, Llopis G, Ricard Y, Dernotte JC. Plasma and tissue fibronectin in diabetes. *Clin Physiol Biochem* 1984;**2**:39–48.
7 McKhann G, Drachman D, Folstein M, Katzman R, Price D, Stadlan EM. Clinical diagnosis of Alzheimer's disease. *Neurology* 1984;**34**:939–44.
8 Li JC, Kaminskas E. Deficient repair of DNA lesions in Alzheimer's disease fibroblasts. *Biochem Biophys Res Commun* 1985;**129**: 733–8.