**Department of
Epidemiology and Public
Health, University College
London, London
WC1E 6EA**

J M Addington-Hall, *lecturer
in health services research*

Wirral Health Authority

M W Weir, *director of
community medicine and
quality assurance*

**Oncology Unit, Bristol
Hospital for Sick Children,
Bristol**

C Zollman, *senior house
officer*

**Royal Lancaster Infirmary,
Lancaster**

M B McIlmurray, *consultant
physician*

they provided no relevant services. Sixteen districts provided only practical support (physiotherapy, dietary advice, stoma and mastectomy care), one provided only emotional care (counselling, support groups), and 94 provided both types of support. Fifty three provided some complementary therapies (guided imagery, meditation/relaxation, healing, and art therapy) in addition to these services. The numbers of districts providing each service are listed in the table.

Although 111 districts had informal links with independently run cancer support services, usually with palliative care services (51) or local cancer support groups (45), there was limited evidence that these services were compensating for deficiencies in NHS services: no districts without support services had links with independent organisations; in four of those offering only practical support emotional care was provided outside the NHS; and in one both emotional care and complementary therapies were available from independent organisations.

Comment

Our results show that there are wide variations in provision of supportive care for patients with cancer. Most districts provided some practical support, usually stoma and mastectomy care. The full range of support, however, was provided in only 32 districts. This may be an underestimation as many hospital dietetics and physiotherapy departments may have provided services for patients with cancer but been overlooked in the completion of the questionnaire because they were not separate specialist cancer services.

Results suggest that the NHS is already responding to the emotional needs of patients with cancer. The availability of counsellors and support groups may, however, be lower than that suggested by our data as we did not ask about the services' nature and accessibility. Some, for example, may have been available only to patients with breast cancer. Some districts at least were referring patients to routinely available clinical psychology or psychiatric services which may not have been available to, or appropriate for, distressed patients with cancer. Macmillan nurses and hospices were often mentioned, suggesting that some services were orientated towards patients in the last stages of their illness.

More districts provided complementary therapies than we had expected. Clearly, the integration of these

Provision of support services for people with cancer by district health authorities

	Districts reporting provision of service	
	No	%
Practical support services		
Specialist physiotherapy	38	22
Dietary advice	116	68
Stoma care	156	92
Mastectomy care	139	82
None of these	7	4
One	10	6
Two	52	31
Three	69	41
Four	32	19
Emotional care		
Counselling	141	83
Support groups	97	57
None of these	24	14
One	54	32
Two	92	54
Complementary therapies		
Guided imagery	18	11
Relaxation-meditation	41	24
Healing	11	7
Art therapy	18	11
None of these	117	69
One	26	15
Two or more	27	16

non-traditional methods into clinical practice has begun, despite the lack of evidence of benefit and of any clear understanding of their role in cancer care.

Our findings suggest that cancer support services are developing in a fragmented and poorly coordinated way. There is no agreed strategy for providing supportive services for patients with cancer and little evidence on which to draw to decide what should be provided and how it should be organised. Further evaluations of models of supportive care¹ and of specific interventions⁴ are urgently needed. In the meantime we urge those responsible for providing and purchasing services for cancer patients to continue to address the need to provide effective and accessible services aimed at meeting physical and emotional needs.

1 Smith T. Cancer services. *BMJ* 1990;301:1406-7.

2 Watson PG. Cancer rehabilitation: the evolution of a concept. *Cancer Nursing* 1990;13:2-12.

3 McIlmurray MB, Gorst DW, Holdcroft PE. A comprehensive service for patients with cancer in a district general hospital. *BMJ* 1986;292:669-71.

4 Fallowfield L, Roberts R. Cancer counselling in the United Kingdom. *Psychology and Health* 1992;6:107-17.

(Accepted 15 April 1993)

Relation between sialic acid concentrations and the haemostatic system in the elderly

Kazuomi Kario, Takefumi Matsuo

**Department of Internal
Medicine, Awaji-Hokutan
Public Clinic, Hokutan,
Hyogo 656-16, Japan**
Kazuomi Kario, *physician*

**Department of Internal
Medicine, Hyogo
Prefectural Awaji Hospital,
Sumoto, Hyogo 656, Japan**
Takefumi Matsuo, *physician*

Correspondence to:
Dr K Kario.

BMJ 1993;306:1650-1

A recent prospective study showed that mortality due to cardiovascular disease was increased in people with high serum sialic acid concentrations.¹ Recent reports suggest that smoking and lipid concentrations may be confounders,^{2,3} but the precise relation between the serum sialic acid concentrations and death from cardiovascular disease remains uncertain. Fibrinogen is a well known risk factor for cardiovascular disease and, like sialic acid, is an acute phase reactant. Plasma fibrinogen concentrations increase with aging and smoking,⁴ characteristics which are similar to those of sialic acid.^{2,3} The possibility that fibrinogen might be a strong confounder that explains the relation between sialic acid concentrations and cardiovascular death has

not been investigated. We therefore investigated the relation between serum sialic acid concentrations and plasma concentrations of various haemostatic variables and the effect of smoking.

Subjects, methods, and results

We studied 138 healthy Japanese men aged over 60 years (mean age 78 years) who were selected from among men undergoing annual health examinations. Subjects with abnormal results on routine laboratory tests (C reactive protein, blood urea nitrogen, creatinine, transaminases, etc) were excluded. Fifty subjects had hypertension and 48 were smokers. Blood samples were collected after an overnight fast, and serum sialic acid concentrations were measured by an enzymatic assay.⁵ Serum total cholesterol and triglyceride concentrations were determined by enzymatic assays using cholesterol esterase, cholesterol oxidase, and glycerol-3-phosphate oxidase. The clotting activity of plasma fibrinogen and factor VII was determined by automated one-stage clotting assays. Plasma concentrations of antithrombin III, heparin cofactor II, plasminogen,

Correlations between serum sialic acid concentrations and plasma fibrinogen, factor VII activity, heparin cofactor II, and antithrombin III levels in elderly men. ● Smokers, + Non-smokers

and α_2 -plasmin inhibitor were determined by chromogenic methods. The following variables were determined by enzyme linked immunoassay (ELISA): lipoprotein(a) (Biopool, Sweden), factor VII antigen, and D-dimer (Diagnostica Stago, France), complex of tissue plasminogen activator and its inhibitor-1, active tissue plasminogen activator inhibitor-1 antigen, plasmin- α_2 -plasmin inhibitor complex (Teijin Co, Japan), and thrombin-antithrombin III complex (Behringwerke AG, Germany). Student's *t* test was used for comparing mean values, and Spearman's correlation coefficients were calculated for the different variables.

There was a strong positive correlation between serum sialic acid concentrations and those of plasma fibrinogen and heparin cofactor II in these elderly men (figure). Serum sialic acid concentrations also showed weak but significant positive correlations with the following variables: factor VII antigen ($r=0.223$, $p<0.01$), complex of tissue plasminogen activator and its inhibitor ($r=0.282$, $p<0.001$), plasminogen ($r=0.356$, $p<0.001$), α_2 -plasmin inhibitor ($r=0.291$, $p<0.001$), thrombin-antithrombin III complex ($r=0.203$, $p<0.02$), plasmin- α_2 -plasmin inhibitor

complex ($r=0.232$, $p<0.01$), triglycerides ($r=0.200$, $p<0.02$), and uric acid ($r=0.171$, $p<0.05$), but not with body mass index and other variables. The mean (SD) plasma fibrinogen concentration was higher in smokers than in non-smokers (3.25 (0.74) v 2.92 (0.53) g/l, $p<0.01$), and the six subjects with the highest serum sialic values were smokers, but the difference in mean serum sialic acid concentrations between smokers and non-smokers was not significant (0.58 (0.09) v 0.55 (0.06) g/l, $p>0.05$).

Comment

Our study confirmed the results of previous reports that serum concentrations of sialic acid show a weak positive correlation with triglyceride values.² In addition, sialic acid concentrations showed significant correlations with the plasma concentrations of pro-coagulant factors (fibrinogen and factor VII antigen), activation markers of coagulation (thrombin-antithrombin III complex and plasmin- α_2 -plasmin inhibitor complex), protease inhibitors (antithrombin III and heparin cofactor II), and fibrinolytic variables (plasminogen and α_2 -plasmin inhibitor). All these factors are closely related to each other by various compensatory feedback mechanisms. In particular, strong positive correlations were found with plasma fibrinogen concentrations and with heparin cofactor II values. These close positive correlations may be due to the fact that sialic acid, fibrinogen, and heparin cofactor II are all acute phase reactants. Alternatively, an increase in highly sialylated fibrinogen may be expressed as an increase in serum sialic acid concentrations. Smoking increased concentrations of both fibrinogen and sialic acid in this study. Our results thus indicate that fibrinogen may perhaps be the confounder that explains the apparent relation between high serum sialic acid concentrations and death from cardiovascular disease.

This study was partly supported by grants from the Foundation for the Development of the Community (Tochigi, Japan), the Japanese Medical Association (Hyogo, Japan), and the Japanese Shipbuilding Industry Association (Tokyo, Japan).

- 1 Lindberg G, Eklund GA, Gullberg B, Rastam L. Serum sialic acid concentration and cardiovascular mortality. Results from 20-5 years follow up of 26 693 men and 27 692 women. *BMJ* 1991;302:143-6.
- 2 Lindberg G, Rastam L, Gullberg B, Eklund GA, Tornberg S. Serum sialic acid concentration and smoking: a population based study. *BMJ* 1991;303:1306-7.
- 3 Wakabayashi I, Sakamoto K, Toshimoto S, Masui H. Relation of serum sialic acid to lipid concentrations. *BMJ* 1992;305:562-3.
- 4 Meade TW. Hypercoagulability and ischemic heart disease. *Blood Reviews* 1987;1:2-8.
- 5 Sugahara K, Sugimoto K, Nomura O, Usui T. Enzymatic assay of serum sialic acid. *Clin Chim Acta* 1980;108:493-8.

(Accepted 15 April 1993)

ONE HUNDRED YEARS AGO

THE COMPULSORY SECLUSION OF INEBRIATES

Two inimitable object lessons, illustrative of the crying need for the involuntary internment of inebriates, are presented in the report in the daily journals of two recent police cases in the metropolis. The notorious Jane Cakebread, the veteran heroine of some 252 police-court appearances for inebriate exploits in defiance of the law, after a month's enforced abstinence in prison, was liberated on Friday week. On the afternoon of the same day she was guilty of a fresh inebriate breach of the peace,

so was treated to another month's incarceration. The other case was that of a drunken mother, who was charged with having, while drunk, taken her four-year-old scarlet-fever-stricken infant through the pouring rain to a public house, to the peril of the company at the bar. When will the Government of the day, by imperative legislation, protect the community and the wretched victims themselves from the domination of a disease which so effaces womanhood in the person of these involuntary and miserable offenders against the law? (*BMJ* 1893;ii:260.)