of endogenous dopamine in the visual cortex.[5] Photic stimulation, in our patients brought about by television, might exacerbate a latent dopaminergic deficiency in the visual cortex due to alcohol withdrawal and lead to seizure.

The presenting feature in all three patients was that they had had a seizure when watching or adjusting the television set. We suggest that when a patient presents after a television induced seizure the possibility of alcoholism should be considered, because management is completely different from that of idiopathic photosensitive epilepsy.

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Protein C values in coronary artery disease

The many risk factors that have been identified for coronary artery disease include a hypercoagulable state, one large study showing that high concentrations of fibrinogen and factors VIIIc and VIIc are better predictors of death from cardiovascular disease than is the serum cholesterol value.[1] Defective fibrinolytic activity has also been implicated in the aetiology of early onset coronary artery disease,[2] and protein C is a recently described regulator of fibrinolysis. Deficiency of protein C has been associated with venous thrombotic disease[3] but concentrations of the protein have not been reported in patients with coronary artery disease. We have therefore assayed protein C values in a group of young patients with coronary artery disease; those with known risk factors for the disease (diabetes mellitus and hyperlipidaemia requiring drug treatment) were excluded.

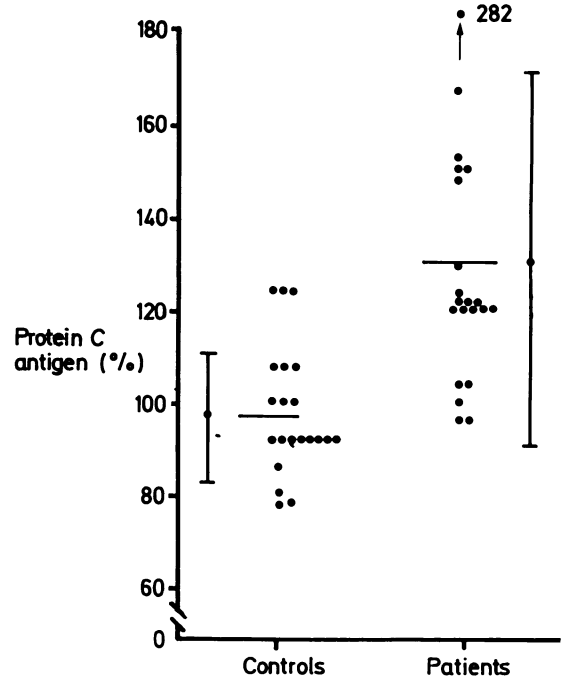
Patients, methods, and results

We studied 21 men aged less than 50 who had angiographic evidence of coronary artery disease. All coronary angiograms made at the John Radcliffe Hospital between 1 January 1979 and 31 December 1983 were reviewed. Angiography in each case was to assess whether patients with angina pectoris were suitable for surgical intervention. Seventy one patients had a positive angiogram, but those who had undergone surgery within the past six months, lived more than 30 miles (48 km) away, or were considered by their cardiologist to be psychologically unsuitable for study were excluded, as were three patients with hyperlipidaemia requiring drug treatment. None had diabetes mellitus. All 21 patients included in the study had documented severe coronary artery disease with complete occlusion of at least one coronary artery in seven cases and almost complete occlusion in 12. Myocardial infarction had been documented in four patients, and 13 had undergone coronary artery surgery. Twenty one healthy non-smoking men aged 25-48 years served as controls.

All patients were well, and none had been in hospital over the past 12 months. Thirteen continued with antianginal treatment, although only four suffered from exertional angina. No patient was taking coumarin anticoagulants, and only two were smokers (fewer than 10 cigarettes a day) at the time of study.

Subjects were studied between 1 June and 31 August 1983, being seen between 0830 and 1030 after an overnight fast. Blood was drawn after 30 minutes' supine rest and samples assayed for prothrombin ratio, kaolin cephalin clotting time, full blood count, fasting blood lipid concentrations, and protein C antigen and activity values.[3 4] Lastly, plasminogen activator concentrations were assayed on euglobulin lysis plates before and after a standard venous occlusion stress test.[5] Protein C antigen and activity values were expressed as a percentage of normal (pooled plasma obtained from 40 normal subjects).

Full blood count, prothrombin ratio, and kaolin clotting time were normal in all subjects, and four patients had borderline hyperlipidaemia. Protein C antigen values were normal or increased in all patients studied, eight of them having values higher than in the controls (figure). Protein C activity results were similar: the mean activity in the patients was 115% (SD 27%) and in the controls 96·6% (SD 16·5%). Fibrinolytic potential in response to venous occlusion was poor in 10 of the 21 patients and two of the controls, but the high protein C values were not related to defective fibrinolysis.



Protein C antigen values in men aged < 50 with proved coronary artery disease and in controls. Bars are means and SD.

Comment

Protein C is the proenzyme of a serine protease concerned in the regulation of coagulation and which is made in the liver in the presence of vitamin K. The protease is a powerful anticoagulant which inactivates factors V and VIIIc and stimulates the fibrinolytic pathway.[3] Both protein C deficiency and defective fibrinolysis have been independently identified as risk factors for venous thrombotic disease,[3 5] and defective fibrinolysis has been linked with early onset coronary artery disease.[4] None of our patients with coronary artery disease had a deficiency of protein C; indeed, eight of them had values higher than in the controls. These high values, however, were unrelated to an individual's fibrinolytic potential and remain unexplained.

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