is routine. Our observations (table III) suggest that patients with tumours of the gut or lung should be the most informative to study. Timed sequential urine collections should be obtained to quantify this proteinuria, measure its constituents, confirm its prevalence, verify the prognostic importance, and determine the incidence. We further recommend that an assay sensitive at low concentrations should be used because the sensitivity of the turbidometric assay is poor.²³ Nevertheless, this assay is used routinely and thus our findings are clinically applicable.

Possibly a subclinical nephritis or other form of renal damage related to malignancy is reflected by these low concentrations of proteinuria. When our data are replicated oncologists might quantify their patients' proteinuria, which may prove to be an additional prognostic feature.

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SHORT REPORTS

Studies on platelets before and after stroke in a patient with essential thrombocythaemia

Only limited data on the aggregation of platelets before stroke are available; we investigated the behaviour of platelets three days before and four days after acute stroke in a patient with thrombocythaemia.

Case report

The patient was a 72 year old man who had had a stroke nine months earlier and been prescribed aspirin 300 mg once weekly. Two months later he had developed a mild left hemiparesis and had signs reflecting disorganisation of the aortic valve. A computed tomogram of the head showed diffuse cerebral atrophy but no focal lesion. The platelet count was raised ($766 \times 10^9/l$), and results of subsequent laboratory investigations were consistent with essential thrombocythaemia. We concluded that he probably had emboli in the right cerebral hemisphere and prescribed aspirin 1200 mg and dipyridamole 400 mg daily.

He remained well for seven months, when he presented with a focal fit affecting the left arm. Cardiac signs were unchanged, there were no signs of subacute

bacterial endocarditis, and cultures of blood yielded negative results. The platelet count was $906 \times 10^9/l$. Aspirin and dipyridamole were withdrawn and phenytoin sodium 200 mg nightly was started. The focal epileptic fit was thought to have been related to cortical scarring after his previous strokes.

Thirteen days later he returned as an outpatient, feeling well and having had no further neurological symptoms. His platelet count was $852 \times 10^9/l$, and detailed platelet studies were performed. Three days later he was readmitted to hospital, having collapsed and developed a right hemiplegia. Initially he seemed to recover, and four days after admission blood was taken for platelet studies; the platelet count had fallen to $557 \times 10^9/l$. His condition deteriorated rapidly shortly afterwards, and he died a day later. Consent for a necropsy was refused.

Platelet studies—Aggregation of platelets was measured in stirred citrated whole blood and heparinised whole blood with a platelet counting technique.¹ Results were compared with those for 20 apparently healthy men (mean age 71, range 61-83). The figure shows that the patient's platelets were hyperaggregable before the stroke and hypoaggregable afterwards. Resistance of aggregation to epoprostenol was assessed in plasma rich in platelets prepared from heparinised blood with 1 mmol sodium arachidonate/l as the aggregating agent.² The results were compared with those for 14 apparently healthy men (mean age 70, range 61-83). A concentration of 10 nmol/l epoprostenol was sufficient to prevent completely the aggregation of platelets (and the release reaction that accompanies it) in plasma from the controls; in contrast, 25 nmol/l, the highest concentration of epoprostenol used, did not affect aggregation of platelets in plasma taken from the patient either before or after his stroke.

Platelet aggregation (percentage of single platelets lost) occurring in (a) stirred citrated blood; (b) stirred heparinised blood; and (c) citrated blood to which adenosine diphosphate (0.5 µmol/l) was added (arrowed). ●=Patient before stroke; ○=patient after stroke; ■ and vertical bars=mean (SD) value in 20 controls.

Comment

Although there have been many studies of aggregation of platelets after stroke,³ few data exist on aggregation immediately before stroke. The patient reported on here had hyperaggregable platelets before the stroke and hypoaggregable platelets (and a lower platelet count) after it. The hyperaggregable platelets may possibly have participated in the formation of thrombus and not been available in the circulation after the stroke; alternatively, natural protective mechanisms may have operated to reduce the aggregability of platelets.

Although aggregation of platelets was reduced after the stroke, it continued to be resistant to the inhibitory effects of epoprostenol. Resistance to inhibitory prostaglandins in thrombocythaemia has recently been reported by others.⁴ That this abnormality was present both before and after stroke in our patient suggests that they are not major determinants of the extent of aggregation of platelets in whole blood. Nevertheless, the resistance of aggregation to inhibition by epoprostenol may be relevant in vivo.

Preston *et al* found that patients with high platelet counts and neurological symptoms improved considerably with treatment with aspirin and dipyridamole.⁵ The fact that in our patient the acute stroke occurred only 16 days after treatment was withdrawn may be important; aspirin and dipyridamole may have been providing protection by reducing the aggregability of platelets.

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Alcohol intoxication and severity of injury in victims of assault

Though an association between alcohol intoxication and aggression has been generally established,¹ surprisingly little evidence of direct cause and effect exists with regard to the increasing problem of violence in inner cities.² Further, although victims of many crimes of violence have been found to be intoxicated,³ data linking the degree of intoxication to the severity of injury are not available. We therefore investigated the association.

Patients, methods, and results

We interviewed and examined 470 consecutive victims of assault who attended an inner city accident and emergency service in 1986. Their reported intake of alcohol in the 12 hours immediately before the assault was recorded by trained interviewers. Blood ethanol concentrations corresponded to this alleged intake ($r=0.699$) in the 362 patients (77%) who consented to testing. The rate of admission to hospital was not significantly different in patients who consented to the investigation and those who did not; the patients who did not consent may have suspected that drug abuse might be detected or the police informed, despite reassurances to the contrary. We compiled an index of the severity of injuries in our patients and related this to the need for admission to hospital by using a

logistic regression model because a suitable scale for categorising injuries in victims of assault was not available.

The categories of the index were as follows: category I=one haematoma or one laceration; II= multiple haematomas or lacerations, or both; III=one fracture; IV=one fracture and haematomas or lacerations, or both; and V=more than one fracture.

Of the 126 patients in category I, eight (6%) were admitted to hospital, compared with 19 (9%) of the 213 in category II, eight (28%) of the 29 in category III, 25 (35%) of the 71 in category IV, and 23 (75%) of the 31 in category V. The index significantly improved the fit compared with the model that assumed a constant chance of admission to hospital ($p<0.01$) and also facilitated investigation of the link between the degree of intoxication and the number of injuries sustained. The index was therefore preferable to admission to hospital as an indicator of severity of injury. Interestingly, recent intake of alcohol alone was also a reasonably accurate indicator of the need for admission ($p<0.05$), though neither this nor the injury severity scale was used to assess patients in this department.

The table shows the number of patients in each category of injury and recent alcohol consumption. Patients who reported abstinence tended to have more

Severity of injury and consumption of alcohol. Figures are numbers of patients

Recent consumption alcohol (units*)	Severity of injury (category†)					Total
	I	II	III	IV	V	
0	54	54	14	20	5	147
1-10	49	96	9	28	17	199
>10	23	63	6	23	9	124
Total	126	213	29	71	31	470

*One unit=½ pint of beer or lager, one measure of spirits, one glass of wine.
†I=One haematoma or laceration; II= multiple haematomas or lacerations, or both; III=one fracture; IV=one fracture and haematomas or lacerations, or both; V= multiple fractures.
 χ^2 (Yates's correction)=25.5, df=10, $p<0.05$.

minor (category I) and fewer severe (category V) injuries whereas drinkers who had consumed more than 10 units of alcohol tended to have more severe (category IV) injuries. Only 23 (18%) of those with category I injury had consumed more than 10 units of alcohol, compared with nine (29%) of those with category V injury. In addition, multiple injuries (categories II, IV, and V) were more likely and single injuries (categories I and III) less likely in drinkers than non-drinkers. Of the patients with single injuries, 68 (44%) were abstainers, but only 79 (25%) of those who sustained multiple injuries had abstained.

Comment

This link between consumption of alcohol by a victim of assault and more serious injury and multiple wounds may arise because aggression and loss of judgment induced by alcohol in the victims prolongs violence or simply because intoxicated victims are less able to avoid blows during an assault. Our data showed that abstinence made severe or multiple injuries in assault less likely and suggested that binge drinking, when more than 10 units were consumed, conferred greater risk of severe injury.

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