Ascorbic acid status in upper gastrointestinal haemorrhage

B. J. G. STRAUSS, M. L. WAHLQVIST, M. G. KORMAN, G. N. BRODIE and P. S. HUNT
Gastroenterology and Medical Oncology Units, Prince Henry's Hospital, Melbourne, Australia; Department of Human Nutrition, Deakin University, Geelong, Australia (BJGS, MLW).

Background

A 1969 study, at Prince Henry's Hospital, Melbourne[1], showed that in acute upper gastrointestinal haemorrhage (haematemesis & melaena, H&M), more patients were likely to have pre-admission ascorbic acid (AA) intakes less than 50 mg/d than a matched group of other hospital patients.

Aims

We decided to assess AA intake as well as plasma and platelet AA levels in a similar group of patients in order to assess whether AA status is a determinant of H&M.

Methods

Pre-treatment blood samples from 23 controls and 64 patients presenting with H&M at Prince Henry's Hospital, Melbourne during the period 1980–81 were separated into plasma and platelet components, the AA content of which was assayed within 48 hours. A dietary history concerning intakes over the previous month was obtained from the patients within 72 hours of admission. Patients were grouped according to the endoscopic site of primary bleeding, and the presence or absence of shock on admission.

Results

AA intakes

There were no significant differences between the pre-admission intakes of controls and those presenting with bleeding duodenal ulcer (DU), gastric ulcer (GU), and Mallory-Weiss tear (M-W), or between those presenting with or without shock (Table 1). In all groups studied, about half the patients had an AA intake reduced in relation to energy intake with respect to the US RDA per MJ (Recommended Nutrient Density, RND).

Circulating AA levels

There were no significant differences in plasma AA and platelet AA between any of the groups studied. A significant correlation existed between plasma and platelet AA levels in all patients with H&M, especially the DU group and the non-shocked group. There was no correlation between AA intake and plasma or platelet AA levels in any of the groups studied (Table 2).

Conclusions

Although overt AA deficiency is associated with mucocutaneous haemorrha-

Table 1. Pre-admission AA intakes. (Mean ± s.e.m.): No significant differences

	Controls	*DU*	*GU*	*MW*	*Shock*	*Non-shock*
n	*(23)*	*(19)*	*(8)*	*(7)*	*(16)*	*(48)*
AA intake (mg/d)	82 ± 18	67 ± 11	82 ± 19	91 ± 20	74 ± 24	73 ± 9
Plasma AA (μmol/l)	54.10 ± 9.95	59.08 ± 5.95	69.88 ± 9.50	45.54 ± 13.83	57.04 ± 9.85	56.20 ± 4.53
Platelet AA	201.19 ± 29.03	220.36 ± 18.76	229.33 ± 59.79	155.15 ± 30.17	181.63 ± 24.89	233.98 ± 20.28

Table 2. Circulating AA levels

Plasma AA vs Platelet AA	0.46*	0.68***	0.12	0.60	0.442	0.59***
AA intake vs Plasma AA	−0.05	0.29	0.52	−0.43	0.30	0.21
AA intake vs Platelet AA	0.30	0.36	−0.50	−0.14	−0.20	0.16

*Spearman's rank correlation rS: $P < 0.05$, ***$P < 0.001$.

ge, it did not appear to be a risk factor in H&M at the time of this study. The increasing AA intake in the general Australian population from 60 to 80 mg/d between 1968-69 and 1980-81[2] may have altered this risk over the time interval of the two studies. Both plasma and platelet AA levels did not reflect AA intakes in these patients.

References
1 Hansky, J. & Allmand, F. (1969): Gastrointestinal bleeding: the role of vitamin C. *Aust. Ann. Med.* **18**, 248-250.
2 Aust. Bureau Statistics (1983): Apparent consumption of foodstuffs and nutrients 1981-82 Catalogue No. 4306.0. Canberra: Commonwealth Govt. Printer.

Neuro-toxic interaction between alcohol and thiamin deficiency in mice

S. C. PHILLIPS *Department of Anatomy, Monash University, Clayton, Victoria, 3168, Australia.*

The combined effects of ethanol consumption and thiamin deficiency on mammalian brain biochemistry[1] and cerebral vasculature[2] have been previously investigated in studies incorporating 'ethanol only' and 'thiamin-deficient only' control experimental groups, in addition to a group subjected to the combined treatment. It has not yet been demonstrated whether the mechanism of ethanol's neurotoxicity (if any) operates independently of the mechanism of brain damage in thiamin-deficiency, or whether the two processes can interact.

Two groups, each of six adult male mice, were fed either a thiamin-deficient diet for ten weeks, and thereafter treated with ethanol by making them inhale vapourized cane spirit for 10 weeks to yield blood alcohol levels in the range 0.1 to 0.3 per cent, or given both treatments simultaneously. The brains of these mice were then searched for degeneration using light microscopy of Nanta-stained[3] frozen sections, and electron-microscopy. No degenerating nerve cells were observed in any animal in the cerebral cortex, hippocampus, cerebellum, olfactory bulbs, midbrain or hindbrain, although expanded endoplasmic recticulum was seen in cells of the cerebral cortex. In light microscopy sections axon terminal degeneration was seen in the olfactory bulbs, deep cerebellar nuclei and ventral aspect of the brain stem in all mice given the combined treatment. No cerebellar degeneration was found and only a little degeneration was present in the olfactory bulbs of mice given the two treatments at different times. Two animals in this group showed sparse brain stem degeneration. A third group of animals received only the thiamin-deficient diet, and the brains of these mice had degenerative signs restricted to the olfactory bulbs and ventral hindbrain, but this result was found in only two of six animals. Animals which received ethanol treatment only possessed no sign of axon terminal degeneration detectable with either light or electron microscopy.

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