

EFFECT OF ALCOHOL ON PLASMA AND PLATELET ASCORBIC ACID IN  
GASTROINTESTINAL BLEEDING

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It has been suggested that patients presenting with upper gastrointestinal (G.I.) bleeding have lower intakes of ascorbic acid than controls (Hansky and Allman, 1969). A comprehensive study of nutritional status and upper G.I. bleeding has been developed at Prince Henry's Hospital. This paper documents preliminary data examining alcohol abuse and ascorbic acid deficiency. All patients presenting to the hospital with upper G.I. bleeding had blood collected on admission for an extensive nutritional profile including plasma and platelet ascorbic acid, measured by the dinitrophenylhydrazine reduction method. Alcohol abuse was defined as an average daily intake of more than 80g of ethanol.

	<u>Table 1.</u>	<u>Results</u>
	<u>No alcohol abuse</u> (n = 18)	<u>Alcohol abuse</u> (n = 11)
Plasma ascorbic acid μmol/L	71 ± 6	40 ± 8 (p<0.005)
Platelet ascorbic acid nmol/10 <sup>10</sup> platelets	220 ± 18	143 ± 26 (p<0.025)

This study demonstrates that with alcohol abuse, plasma and platelets are significantly deficient in ascorbic acid. Alcohol may compromise ascorbic acid status by displacing ascorbic acid-rich foods from the diet or interfering with absorption. Low ascorbic acid status has implications for the adequacy of haemostasis because of the role of ascorbic acid in collagen synthesis. Further, since the platelet is a concentrator of ascorbic acid it is possible that platelet function is ascorbic acid dependent.

HANSKY, J. and ALLMAN, (1969) F. Aust. Ann. Med. 18: 248.

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