adjunct to replacement of blood volume in cases of acute hypovolaemic hypotension, though it has not been tested in animals. Meanwhile, much more accurate observations can be made of the haemodynamic disturbances that accompany acute blood loss in patients rather than volunteers.

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Alcohol and gout

Stopping alcohol may stop the gout

Alcohol and gout have long been associated in the medical and lay mind. Over 100 years ago Garrod thought that fermented liquors were the most powerful of all predisposing causes of gout[1] whereas more recently Khan showed that for victims of gout eating and drinking were two of life's greatest pleasures.[2] What might explain this association?

The metabolic disorder in gout results in too much urate in the blood and tissue fluids, and there are several possible ways that alcohol could contribute to this excess. The high energy content of alcohol predisposes towards obesity, and body weight and uric acid concentrations are related.[3] Drinking alcohol to excess produces moderate hypertriglyceridaemia, which is associated with hyperuricacidaemia and gout.[4] Alcohol may have a role in "washing down huge platters of meat"[5]—itself known to predispose towards gout.

Acute alcoholic intoxication may produce transient lactic acidaemia and ketosis, leading to inhibition of the renal tubular secretion of urate and to hyperuricacidaemia.[6] Long term oral and short term intravenous administration of alcohol to patients with gout showed that alcohol increases the synthesis of urate by increasing the turnover of adenine nucleotides.[7] Beer drinkers may have to contend with not only the hyperuricacidaemic effects of alcohol but also the high purine content of beer.[8] Taken together these studies suggest that the long term consumption of alcohol increases the synthesis of urate while acute intoxication makes things worse by reducing its excretion.

Some doctors and patients believe that certain alcoholic drinks—for example, red wine and port—are more conducive to gout than others—for example, white wine and whisky. If this is true then there is no satisfactory explanation for the differences.

These findings have clear implications for treating patients with hyperuricacidaemia and gout. Patients should be carefully assessed for any factors that are possibly contributing to their hyperuricacidaemia[9] and counselled to avoid them. Many patients who accept the advice will become normouricacidaemic and remain so. (Stopping overeating and overdrinking is likely to be beneficial itself quite apart from its effect on hyperuricacidaemia.) Drug treatment should be reserved for patients with gout who remain hyperuricacidaemic despite having corrected their eating and drinking habits or who, as is unfortunately often the case, disregard this advice.

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