The Beer Drinker's Kidney

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The recent report of acute renal failure following excessive beer drinking [1] recalls the events of the late 1960s when several episodes of severe cardiac failure occurred soon after the imbibing of large quantities of beer made more appealing by the addition of cobalt chloride to form an attractive 'head'. These incidents, transpiring in two widely separated geographic areas, Omaha, Nebr. and Quebec City, Canada, have been documented [2]. After careful inquiry, cobalt chloride was identified as the culprit [3].

A large animal model of cobalt (chloride) cardiomyopathy, using the dog, was subsequently created [4]. In the course of this investigation, it was observed that the renal collecting tubules from the paired organs of 7 of the 9 animals studied exhibited, in 3–5 days, marked epithelial swelling, necrosis, and luminal obstruction [5]. Though the urinary output remained unimpaired, the renal tubular changes may, notwithstanding, have been a contributing factor to the extreme levels of clinical myocardial failure, muscle degeneration, and necrosis that ensued after 7 days of the cobalt regimen.

Similar renal alterations have not been described heretofore in cases of human cobalt cardiomyopathy. The data presented by Ghose et al. [1] suggest the presence, in the beer consumed by the reported subjects, of a noxious agent other than the alcohol itself. Perhaps, here as in North America, cobalt has been incorporated into the beer during its manufacture.

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