METHANOL INTOXICATION

SIR,—The identity of the metabolite that accounts for the acidosis associated with methanol intoxication is uncertain. One view is that methanol is oxidised to formaldehyde and then to formic acid, and that formic acid causes the acidosis. Smith et al.[6] have described a methanol-intoxicated patient whose blood pH was 6·78, serum sodium 136, potassium 5·5, chloride 101, and standard bicarbonate 3 mmol/l. The anion gap was 37·5 mmol/l, which is about 21·5 mmol/l above the average normal level. Their patient had a serum formate of 6·9 and lactate of 11·6 mmol/l, which together accounted for nearly all the excess anion gap. They suggested that pyruvate was reduced to lactate as the oxidation of methanol yielded protons. Their patient clearly had lactic as well as formic acidosis, but the question remains whether lactic acidosis is an inherent metabolic consequence of methanol intoxication, or whether in their patient (and perhaps others) the lactic acidosis was secondary to circulatory failure, which can occur during severe methanol poisoning. Smith et al.[6] did not state whether their patient had shock or imminent circulatory failure when the blood specimen was obtained.

McMartin et al.[7] reported elevated whole blood formate concentrations in two methanol-intoxicated patients, but the information in their report makes it difficult to judge whether the acidosis in those patients was due mainly to formic acid.

Studies in animals have given varying results, probably because methanol is metabolised differently in various species. In studies in monkeys by McMartin et al.,[8] whole blood formate concentrations were considerably increased but did not fully account for the decreases of plasma bicarbonate; lactate measurements were not reported. Clay et al.,[9] however, found that the raised blood formate concentrations in their methanol-intoxicated monkeys did almost completely account for the decreases of plasma bicarbonate; plasma lactate concentrations were only slightly increased.

The monkey experiments and clinical observations indicate that formic acidosis is probably a major component of the metabolic acidosis associated with methanol intoxication. It remains uncertain, however, whether lactic acidosis is also a consistent major component of the acidosis in humans and if so, whether such lactic acidosis arises in the way Smith et al. suggest or is due to shock. The question is important to answer because of Smith's hypothesis that the lactic acid is formed from reduction of pyruvate by protons derived from the oxidation of methanol. If this hypothesis is correct it might be unwise to treat methanol-intoxicated patients with ethanol because the metabolism of ethanol could also increase lactic acid formation, as Smith et al. pointed out.

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