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and to 9·5 (0·8) mmol/l at 120 minutes ($p<0·001$) after starting the infusion of arginine hydrochloride.

The observed increase in plasma urea is likely to be due to the cleavage of L-arginine to urea and ornithine catalysed in the liver by the enzyme L-arginase. This enzyme is present in the liver of all ureotelic organisms and, like most mammalian enzymes, only acts on the L-isomer. To test this hypothesis, we measured the changes in plasma urea concentrations occurring in eight anaesthetised and mechanically ventilated rabbits infused with either L-arginine or D-arginine at doses comparable with those used during an acidification test. After the infusion of L-arginine (0·05 mmol/kg/minute) in three animals, mean (SD) plasma urea rose from 6·4 (2·1) to 7·6 (2·1) mmol/l at 60 minutes ($p<0·01$) and to 8·9 (2·2) mmol/l at 120 minutes ($p<0·01$). By contrast, the infusion of the same dose of the D-isomer in three other rabbits did not significantly modify plasma urea (5·9 (0·2) before the infusion, 6·0 (0·1) at 60 minutes and 6·5 (0·1) mmol/l at 120 minutes). Plasma urea also remained stable in the two saline infused control rabbits.

Our clinical and experimental data thus suggest that cleavage of L-arginine to urea and ornithine by the liver L-arginase is probably responsible for the increase in plasma urea concentrations observed in children infused with arginine hydrochloride. The lack of endogenous D-arginase explains the failure of urea to increase after the administration of D-arginine. While this rise of plasma urea does not have any deleterious effects in children with normal renal function, it may likely lead to misinterpretation of urea data collected during an arginine infusion test.

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