



Letters to the Editor

Heparinised saline solutions

Sir,

I read with interest the article "A comparison of heparinised saline irrigation solutions in a model of microvascular thrombosis" by Cox *et al.* (1992).

The authors studied the effect of different concentrations of heparinised normal saline on the patency rate of a laboratory model of arterial microvascular thrombosis in Lewis rats at 20 minutes and 24 hours. They conclude that 100 U/ml is the ideal concentration of heparinised saline irrigation because it significantly improved patency without producing any systemic effects. They also conclude that the use of higher concentrations (250 and 500 U/ml) do not result in a decrease in patency rates, contrary to the results reported by Zinberg *et al.* (1989).

Zinberg *et al.* (1989) examined the effect of different concentrations of heparinised Ringer's lactate solution on the patency rate at 2 hours of 4 different types of microvascular anastomoses in Sprague-Dawley rats. The use of heparinised Ringer's lactate solution resulted in a statistically significant improvement in patency rate only in the end-to-end venous anastomosis group. A concentration of 20 U/ml produced the optimum patency rate (85%), whereas the use of 100 U/ml resulted in a statistically significant worsening in the patency rate (55%).

It is interesting to speculate on possible reasons for a reduction in the antithrombotic effects of heparin at higher concentrations. Zinberg *et al.* postulated that the associated excessive and prolonged bleeding seen with the use of the higher heparin-containing solutions may have resulted in vessel spasm or possible external compression by haematoma. A further possible explanation, however, is provided by the work of Saba *et al.* (1979), who showed that heparin neutralises the inhibitory effect of prostacyclin on platelet aggregation. This effect was demonstrable in normal human volunteers at concentrations as low as 2.5 U/ml, and may

also account for reports of heparin being associated with thrombotic episodes and thrombocytopenia.

Finally, although heparin has been shown to improve the patency rates of injured vessels in thrombosis models (Cox *et al.*, 1992), it is more difficult to demonstrate its ability to maintain patency in uncomplicated repairs, particularly on the arterial side (Zinberg *et al.*, 1989). When tissue factor (thromboplastin/factor IV) is present, as it would be in any thrombosis model employing tissue damage, the activation of coagulation via the interaction of tissue factor and factor VII (*i.e.* the extrinsic pathway) can be prevented by heparin. It may be that thrombosis of otherwise *uncomplicated* repairs – especially on the arterial side – is via a heparin – independent mechanism *i.e.* platelet adhesion and activation. As the experimental thrombosis models simulate the clinical trauma situation, it seems logical to continue to use heparin in this high risk group of patients, although the most effective dose and route of administration has not yet been determined satisfactorily.

Yours faithfully,

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