The above arguments are based on a change in total blood binding of the drug, that is binding to plasma and erythrocyte components. However, if displacement occurs only in the plasma and not on or in the erythrocytes, binding sites in the latter might take up some of the excess free drug (Tucker et al., 1970; Hahn et al., 1973) and any change in total drug concentrations would be buffered by this effect. This would have the effect of reducing the overall rate of drug elimination by the liver.

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REFERENCES


SIR.—We would like to thank Drs. Tucker and Mather for their interest in our paper. We still think that we used concentrations of displacing drugs that can occur during their clinical use. Our references are the following: for diphenylhydantoin, Jensen and Grynderup (1966); quinidine, Goodman and Gilman (1970); pethidine, Reddin (1967); and desipramine, Borga and colleagues (1969). Incidentally, the volume of the buffer compartment was not significantly different from that of the plasma compartment at the end of dialysis. The difference between these references and those cited by Tucker and Mather may be the result of different methods of analysis. The cause of the decreased plasma binding of bupivacaine in patients who received pethidine as a pre-medication may be the pethidine itself. However, if we accept Tucker’s and Mather’s figures of pethidine concentrations, one may speculate about other causes. For example, the stress of impending surgery may increase the circulating free fatty acids which may displace bupivacaine from its binding sites (Spector and Santos, 1973). The rest of Tucker’s and Mather’s letter explains in detail our statement that “Apart from accidental intravascular injection or gross overdosage a decrease in binding would release only a small absolute amount of drug.”

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REFERENCES


POSTOPERATIVE FLUID AND ELECTROLYTE REQUIREMENTS

SIR.—The paper by the Dallas group (Jenkins, Giesecke and Johnson, 1975) concerning perioperative fluid and electrolyte balance emphasizes the great difference between American and British practice in this field. There is scarcely a paragraph in their paper with which I agree. However, perhaps I might be allowed to limit my comments to three points.

First, the description of their regimens is interesting by virtue of its ingenious and illogical complexity. However, it is irrelevant to British practice as one of the solutions used, 5% dextrose in a balanced salt solution, is not commonly available in this country. The amounts of fluid used, although thankfully less than those recommended by the same group 10 years ago, are still far in excess of that considered safe by British anaesthetists and surgeons. It must be remembered that a rapid infusion of only 1–2 litre normal saline to fit, healthy adults who are not subject to the profound fluid retention of surgery and anaesthesia show pulmonary changes which may lead to impairment of pulmonary gas exchange (Collins et al., 1973).

Second, the rationale for giving 5% dextrose in water to reduce “renal work” is appealing until it is realized that renal work, as assessed by oxygen consumption, is expended in reabsorbing rather than excreting solute. Eighty per cent of renal oxygen consumption is concerned with reabsorbing sodium. Thus, renal work can be reduced only by decreasing the amount of sodium presented to the proximal tubule, by reducing G.F.R. or by using agents which decrease tubular sodium reabsorption.

Third, I would dispute that urine output during surgery is a good guide to the adequacy of fluid replacement. It is my experience that anaesthesia and surgery are always associated with severe oliguria unless diuretic agents or a fluid load are given or unless renal perfusion is altered. Even with a fluid load of more than 10 ml/kg, only half the administered fluid is excreted (Fieber and Jones, 1967). Thus, a urine output of 50–100 ml/hr in the immediate perioperative period is suggestive of fluid overload rather than normal renal function.

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