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Simplified Vasoactive Drug Infusion

To the Editor:—Recent letters to ANESTHESIOLOGY by Webb,[1] Kondo,[2] and Tanaka[3] have recommended systems for calculating the drug dosage delivered via infusion pumps. These systems require either preparing specific dilutions for each drug and using a nomogram or using a conversion formula with fixed dilutions.

We employ a system that does not require any special dilutions and that can be easily used without the aid of a nomogram or a conversion formula. Most infusion pumps are calibrated in ml/h or drops/min. When a "minidrip" infusion set is used (where 60 drops = 1.0 ml) then drops/min = ml/h. Furthermore, if x mg of drug is diluted in 250 ml of solution, then an infusion pump with a "minidrip" set at 15 drops/min or 15 ml/h will deliver x μg/min of drug. Obviously, multiples or fractions of 15 drops/min will deliver equal multiples or fractions of x μg/min. For example, if 10 mg of phenylephrine diluted in 250 ml of solution is infused at 15 ml/h, then 10 μg/min of drug will be infused. Likewise, 60 ml/h will deliver 40 μg/min.

This system allows us to rapidly prepare our drip solutions by simply adding an "amp" of the desired drug to the 250 ml solution of 5% dextrose in water or normal saline. As long as the solution is carefully labeled with the total milligram amount of drug and the infusion rate of the "minidrip" in drops/min or ml/h is known, then the infusion rate of drug in μg/min is easily calculated.

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The Relationship between Sympathoadrenal Activity and Extrarenal Potassium Regulation

To the Editor:—The demonstration by McCammon and Stoelting that β-blockade causes an exaggerated increase in serum potassium following succinylcholine[1] raises an important question. What is the clinical significance of intraoperative extrarenal potassium regulation?

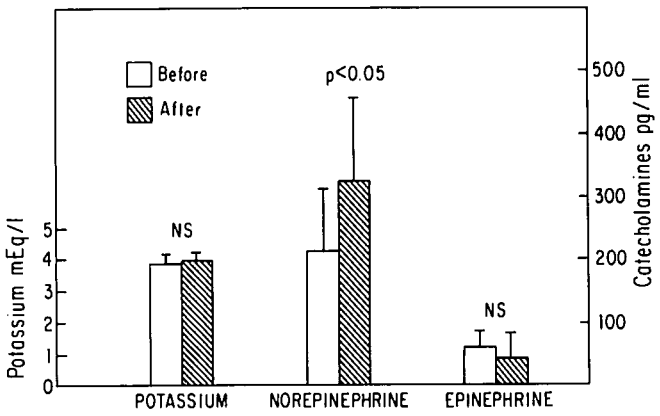

FIG. 1. Serum potassium and catecholamines before and after median sternotomy.

There is evidence that enhanced α-adrenergic receptor activity increases serum potassium and that this effect is reversed by α-adrenergic receptor antagonism.[2] Furthermore, β-adrenergic blockade inhibits extrarenal cellular uptake of potassium.[3] These findings are from data obtained during potassium infusion in normal human subjects. Knowledge of the circulating level of catecholamines during MacCammon's study would have been of interest. Inhibition of α-adrenergic tone caused by the central effect of diazepam may explain the observed attenuation of postsuccinylcholine hyperkalemia.

We measured serum potassium, norepinephrine, and epinephrine before and after median sternotomy in five β-blocked patients undergoing coronary artery revascularization. None of the patients was receiving diuretics or other potassium-regulating drugs preoperatively. The patients were anesthetized with 40 μg/kg iv fentanyl,