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### Simplified Vasoactive Drug Infusion

To the Editor:—Recent letters to ANESTHESIOLOGY by Webb, <sup>1</sup> Kondo, <sup>2</sup> and Tanaka<sup>3</sup> 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  $\mu$ g/min of drug. Obviously, multiples or fractions of 15 drops/min will deliver equal multiples or fractions of x  $\mu$ g/min. For example, if 10 mg of phenylephrine diluted in 250 ml of solution is infused at 15 ml/h, then 10  $\mu$ g/min of drug will be infused. Likewise, 60 ml/h will deliver 40  $\mu$ g/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  $\mu$ g/min is easily calculated.

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### REFERENCES

- Webb TD: Intravenous infusing: Making life easy. ANESTHE-SIOLOGY 59:482, 1983
- Kondo K: Vasoactive drug infusion: Making life easier. ANES-THESIOLOGY 60:617, 1984
- Tanaka K: A simple nomogram for determining drug infusion rates. ANESTHESIOLOGY 62:99, 1985

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# The Relationship between Sympathoadrenal Activity and Extrarenal Potassium Regulation

To the Editor:—The demonstration by McCammon and Stoelting that  $\beta$ -blockade causes an exaggerated increase in serum potassium following succinylcholine<sup>1</sup>

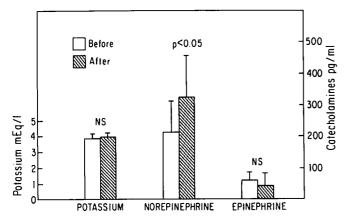


FIG. 1. Serum potassium and catecholamines before and after median sternotomy.

raises an important question. What is the clinical significance of intraoperative extrarenal potassium regulation?

There is evidence that enhanced  $\alpha$ -adrenergic receptor activity increases serum potassium and that this effect is reversed by  $\alpha$ -adrenergic receptor antagonism.<sup>2</sup> Furthermore,  $\beta$ -adrenergic blockade inhibits extrarenal cellular uptake of potassium.<sup>3</sup> 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  $\alpha$ -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  $\beta$ -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  $\mu$ g/kg iv fentanyl,

and intubation was facilitated with 0.15 mg/kg of pancuronium bromide. Ventilation with 100% oxygen was controlled to maintain normocapnea. Figure 1 shows that, in spite of an increase in norepinephrine following sternotomy, there was no change in serum potassium. Current evidence may have predicted an increase in serum potassium, especially in the presence of  $\beta$ -blockade. Measurement of extracellular serum potassium, however, is an insensitive index of continuous transmembrane ionic flux. Therefore, in spite of our findings, we suggest that intraoperative extrarenal potassium regulation can be influenced by adrenergic mechanisms and that this is a potential cause of cardiovascular morbidity.

It is well established that catecholamine levels often are increased intraoperatively. Changing sympathoadrenal activity, modified by the presence of receptor blockade, may occur because of mismatching between the depth of anesthesia and the degree of surgical stress or during the administration of vasoactive adrenergic agents. Arrhythmogenesis may ensue as a result of subsequent potassium effects on phase iv depolarization of cardiac pacemaker and conduction cells.

In summary, the intraoperative relationship between sympathoadrenal activity and extrarenal potassium regulation merits further investigation. R. WILLIAM MCINTYRE, M.D. Assistant Professor

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### REFERENCES

- McCammon RL, Stoelting RK: Exaggerated increase in serum potassium following succinylcholine in dogs with beta blockade. ANESTHESIOLOGY 61:723-725, 1984
- Williams ME, Rosa RM, Silva P, Brown RS, Epstein FH: Impairment of extrarenal potassium disposal by α-adrenergic stimulation. N Engl J Med 311:145–149, 1984
- Rosa RM, Silva P, Young JB, Landsberg L, Brown RS, Rowe JW, Epstein FH: Adrenergic modulation of extrarenal potassium disposal. N Engl J Med 302:431–434, 1980
- Halter JB, Pflug AE, Porte D: Mechanism of plasma catecholamine increases during surgical stress in man. J Clin Endocrinol Metab 45:936–944, 1977

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## Anesthesia Ventilators Should Have Adjustable High-pressure Alarms

To the Editor:—We wish to call attention to a serious shortcoming of the Drager Model AV-M® anesthesia ventilator with the integral Model DPM-S pressure alarm (North American Drager, P.O. Box 121, Telford, Pennsylvania 18969). We discovered this while investigating a critical incident in which the high-pressure alarm did not sound when an endotracheal tube became occluded by kinking.

In experiments on a properly functioning ventilator and a lung simulator, we found that there are certain typical settings of the ventilator at which even total occlusion of the endotracheal tube will not activate the alarm, nor will there be any noticeable change in the rhythmic sounds from the ventilator to alert the anesthesiologist to the problem. This occurs because, for a given inspiratory flow setting on the machine, the delivered flow rate will vary with the respiratory system impedance. Thus, when outflow is obstructed, the bellows may not completely empty, and the airway pressure may not increase to the alarm threshold of  $63.5~{\rm cm}{\rm H}_2{\rm O}$   $\pm 10\%$ . Because the machine is time-cycled, the inflation

time remains fixed, and the ventilator's sound does not change.

There are too many parameters involved to permit us to state exactly the settings under which the alarm can fail. However, the following factors, in various combinations, tend to favor alarm failure: high respiratory rate, low inspiratory/expiratory ratio, low tidal volume, high tubing compliance, low inspiratory flow rate, and low rate of fresh gas inflow from the anesthesia machine.

The alarm would not fail to trip if the pressure limit could be adjusted to a value slightly greater than the peak inflation pressure of any given patient. Adjustable high-pressure alarms are now commonly build into intensive care ventilators. Nevertheless, only two of the 11 stand-alone ventilation alarms in a comparative review\* had this capability, and the Drager Model DPM-S was the best-rated model because of its other features.

<sup>\*</sup> Health Devices 10:204-220, 1981.