Dilemmas in Potassium Therapy

To be provocative and to draw attention to the problem rather than resolve it, I suggest semiferently that a debate might be staged with the title: Resolved: That more lives have been lost than saved by potassium therapy in the past year in American hospitals. That potassium depletion is a frequent and sometimes serious problem in contemporary medical practice cannot be denied, but my implication is that repetitive or preventive measures, if they be unduly enthusiastic, may evoke more serious problems. Guidelines for dosage of the potassium often seem drawn from a small data base with occasionally an exclusive focus on serum K levels, possibly obtained hours previously.

The clinician’s approach to the patient with suspected potassium depletion, maybe with a concomitant arrhythmia, may provide him with a paradigm of the difficulty in properly weighing the inputs to his decision-making process, i.e., from (1) the theory based on cellular biology; (2) the results of physiologic experiments on tissue and the whole heart; and (3) prior clinical experience with potassium administration, together with anecdotal data of his teachers and colleagues.

In a small monograph entitled “Potassium Therapy: A Seminar” edited by Soffer,1 many of the problems of potassium kinetics are informally discussed. Many facts and opinions are clearly presented. The not-occasional dilemma is highlighted by one truism, namely, “potassium depletion and hypokalemia are not synonymous.” To the beginner, this little book might well be confusing, but to the experienced but often puzzled clinician, this candid presentation of potassium problems by a group of experts is refreshing.

While potassium depletion can be generally inferred from a low serum level, and both atrial and serious ventricular arrhythmias may be facilitated, the clinical state of a patient with such a problem is generally less precarious than when hyperkalemia is threatening or present. The emergence of digitalis-related arrhythmia with hypokalemia has been well described and standard references are those of Lown and Levine2 and of Fisch and Surawicz.3

There has been a long interest in potassium effects and paradoxes. One of the first paradoxes noted was that huge doses of KCl (e.g., 24–48 g daily) could be tolerated orally by one experimental subject, while for others, particularly in those with mild renal insufficiency, small doses (e.g., 5 g orally) could have toxic effects.4

An apparent paradox is the sinus arrest produced when a perfusate that has been free of potassium is brought up to normal level in an experimental preparation. This “paradoxical phenomenon of Zwaardemaker and Libbrecht” has been reinvestigated by Surawicz and Gettes5 who point out the electrophysiologic effect of a temporary increase in the velocity of repolarization (a short Q-T interval).

In a recent review, Zipes and Fisch6 stated that “potassium is an excellent antiarrhythmic agent. The drug can be administered either orally or intravenously, the latter route being the preferable one. Contraindications to its use are hyperkalemia, impaired renal function, and the presence of A-V block greater than the simple prolongation of the P-R interval.” The validity of the conclusions is unquestioned, but the authors may not have properly emphasized safeguards, specifically the dosage and rate of administration. When one administers potassium, one thinks of (1) the availability of, and the receptivity by, the cellular depots, and (2) the rate of loss by kidney and bowel.

To these determinants of serum K levels, the status of acid-base balance and of organ perfusion (cardiac output) are of prime importance. The old dependence of physicians on the wisdom of the body is frequently challenged now, particularly since the body under stress may show seemingly inappropriate responses. However, at times, the physician’s response may be also inappropriate. One surgical resident told me that he likes to see the serum potassium levels a little above 5 mEq in his postoperative patients as there appears to be less arrhythmia. Such a viewpoint obviously might be labelled tunnel vision; viz, if renal function or

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pulmonary ventilation should change suddenly, a continuing order, for instance, of a does of “5 or 10 mEq/hour” could portend trouble.

When pH shifts are related to pCO2 manipulation, the serum K shows a reasonably predictable change, with the level falling at about 0.2 mEq/liter per each 0.1 rise in pH. A dramatic illustration of the importance of the acid-base state modulating K toxicity is that reported by Keating et al.; when a potassium infusion was given with NH4Cl in the nephrectomized dog, death was invariable; whereas when the same amount of potassium was infused with isotonic NaHCO3, cardiac arrest was not observed.

A somewhat absurd projection of the importance of the rate of administration is illustrated by the report that massive doses of potassium penicillin can be infused rapidly enough into an animal to produce K arrest.9 There is evidence also that a fatal “dose” of K can be endogeneously produced, through destruction of tumor tissue, specifically in chemotherapy of lymphoma.10

Potassium loading has been used in attempts to differentiate T-wave abnormalities in the ECG as either functional or organic. The discriminatory power of this procedure relative to the danger has never made the method attractive to me. In a recent report by Thomsen et al.,11 potassium was given intravenously in doses as high as 32 mEq, but the period of the injection was not mentioned. The authors’ statement that the test “should not be used as a clinical testing procedure” deserves to be remembered.

A recent editorial in Lancet,12 entitled “Glucose and the Heart,” states that the glucose-insulin-potassium solution (GIK) “is a safe and simple addition to cardiac therapy.” Relevant to this editorial is only the question of safety. A slow injection is inherent in the method described by Sodi-Pallares et al.,13 and to be noted is the progressive decrease in the KCl added per liter of solution infused (from 60+ to 0 mEq) as the serum K is found to be elevated (from < 4– to 5.6 mEq).

The optimal intake of potassium has not been clearly established for a normal individual or for a patient on medication. It may be a routine for some practitioners to give a potassium supplement to anyone taking a diuretic, and as an approach, it is reasonable. Such practice is fostered by the extensive competitive advertising of potassium preparations. Parenthetically, I could not predict the number of instances of hyperkalemia which have been abetted by concomitant use of a spiroactone or triamterene. Probably it is uncommon; of interest is the apparent propensity of elderly diabetics to develop hyperkalemia on triamterene. In a wise approach to the whole patient, the physician will remember that anorexia, gastric distress, diminished food intake, and loose bowel movements can defeat his hope of optimal treatment. In my hospital, the cost to the patient of a daily proprietary K supplement of 60 mEq would vary from 2.50 to 8.50 dollars a month. In Minnesota, the cost of buying fruit to supply the same amount of K would vary seasonably, but would be about three times greater than the cost of the drug.

The threat of potassium depletion seems ubiquitous; it appears, for example, in astronauts on the lunar missions,14 and rarely in patients on frequent chronic dialysis for renal insufficiency.15

From this outline, I hope, may have emerged the foundations, wherein may rest my theoretic debate: “Resolved: That more lives may have been lost than saved by potassium . . .” To the standard bearer for the affirmative may flock those who are impressed with the dangers related to high doses, inadequate monitoring, and unstable states; to the negative, those conscious of the seeming ubiquity of potassium-depleted states, and in them, the propensity for arrhythmias, and a decreased tolerance for digitalis.

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References


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