Indications for Hospitalization of Patients With Hyperkalemia

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**Background:** Although the methods for the appropriate management of patients with hyperkalemia are well established, no criteria for hospital admission of patients with this common electrolyte disorder have been promulgated.

**Objectives:** To examine the current practices regarding hospitalization of patients with hyperkalemia and to consider appropriate criteria for admission.

**Patients and Methods:** We evaluated a consecutive series of patients hospitalized for hyperkalemia and excluded patients who developed hyperkalemia after admission. For comparison, we selected a series of patients with a similar degree of hyperkalemia who were treated as outpatients. Hyperkalemia was classified as minimal, moderate, or severe. The causes of hyperkalemia were identified, and the therapeutic maneuvers used were ascertained. Although the study did not have the power to determine the relative safety of the 2 therapeutic approaches, we compared the outcomes of the 2 groups of patients.

**Results:** The inpatient group consisted of 11 patients who were admitted for the treatment of hyperkalemia, and we identified 12 patients who received outpatient therapy for hyperkalemia. The patients in the 2 treatment groups were similar with respect to age and the values of serum urea nitrogen, creatinine, and potassium prior to the identification of hyperkalemia. The mean ± SD potassium concentrations at baseline were 5.4±0.7 mmol/L in the inpatients and 5.5±0.5 mmol/L in the outpatients. The mean ± SD potassium concentration in the inpatients was 6.7±0.8 mmol/L at the time of hospital admission, compared with 6.7±0.5 mmol/L in the outpatients at the time that hyperkalemia occurred. Similar proportions of both groups (6 of 11 inpatients and 7 of 12 outpatients) had moderate or severe hyperkalemia.

**Conclusions:** Patients admitted to the hospital were clinically indistinguishable from patients treated as outpatients. The justification for the decision to admit patients to the hospital or to treat them as outpatients was often not evident. We suggest criteria for hospitalization, which include severe hyperkalemia (≥8.0 mmol/L, with changes other than peaked T waves on the electrocardiogram), acute worsening of renal function, and supervening medical problems.

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**HYPERKALEMIA** is a common laboratory abnormality complicating between 1.1% and 10.0% of all hospital admissions.¹⁻⁴ Hyperkalemia occurs in as many as 11% of patients using angiotensin-converting enzyme inhibitors (ACEIs),¹⁻⁶ which are commonly used in the management of hypertension, congestive heart failure, diabetic nephropathy,⁷ and chronic renal insufficiency. Although severe hyperkalemia is generally considered a life-threatening event,⁸ there is little agreement on what constitutes mild, moderate, or severe hyperkalemia. While Levinsky⁹ defined “minimal” hyperkalemia as a serum potassium concentration less than 6.5 mmol/L accompanied by only minor electrocardiographic changes, others¹⁰ have described a potassium concentration greater than 6.0 mmol/L as severe hyperkalemia. Moreover, few definitions take into account that the toxic effects of a given potassium concentration depend on the baseline value and rate of increase in potassium concentration as well as the acid-base status and serum calcium concentration.¹¹⁻¹⁴

Additional complexity arises from the numerous modalities available for the treatment of hyperkalemia. These include, but are not limited to, cessation of foods and drugs capable of worsening hyperkalemia; infusion of calcium gluconate, sodium bicarbonate, and insulin; administration of oral potassium-binding resins; and hemodialysis.¹²,¹³,¹⁶

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PATIENTS AND METHODS

IDENTIFICATION OF PATIENTS

The study was approved by the human studies committee of the local institutional review board. A computerized search of International Classification of Diseases, Ninth Revision (ICD-9), codes for all discharges from the New York Department of Veterans Affairs Medical Center, New York, between October 1, 1995, and April 15, 1997, was performed. Of 10961 admissions, 59 were coded for hyperkalemia. The summaries, laboratory records, and, if necessary, additional records obtained at hospital discharge of these 59 cases were reviewed. Subjects were considered to have been admitted for the treatment of hyperkalemia only if the discharge summary indicated that hyperkalemia was a reason for admission. Diagnoses of hyperkalemia made at discharge were confirmed by review of the admitting physician’s note if available. Patients with normokalemia on admission were excluded regardless of subsequent elevations in potassium concentration. We did not consider admissions in which the reason for admission was unrelated to potassium concentration but in which hyperkalemia was found in the results of the laboratory tests performed at admission. Conversely, the presence of acute medical problems in addition to hyperkalemia was not a cause for exclusion as long as it was apparent that the hyperkalemia was the primary indication for admission. As the primary purpose of this study was to investigate the severity of elevation of potassium concentration in patients admitted for the treatment of hyperkalemia, neither upper nor lower exclusion criteria for potassium concentration obtained at admission were applied. Of the 59 cases coded for hyperkalemia, 10 met these criteria. An additional case meeting these criteria was identified from a review of elevated potassium concentrations (see below).

For comparative purposes, we identified a consecutive series of outpatients treated for hyperkalemia. We performed a computerized search of all potassium concentrations of the laboratory “panic value” of 6.1 mmol/L or higher. (The panic value was chosen by the laboratory director and constituted the laboratory’s only criterion for contacting the primary care physician.) Because we expected a large number of samples to meet this criterion, the search was restricted to samples drawn between January 1, 1997, and March 31, 1997—a period that overlaps with the final 3 months of the inpatient study. A total of 142 cases with abnormal potassium concentrations were identified during these months. To identify true cases of outpatient hyperkalemia, hemolyzed specimens and specimens drawn from inpatient units were excluded. Specimens obtained monthly before hemodialysis from patients in the hemodialysis unit were also excluded, as these samples would rarely lead to any therapeutic plan other than to proceed with hemodialysis. Patients undergoing dialysis were not excluded from the inpatient group if hyperkalemia was the indication for admission. The remaining samples were cross-referenced with computerized notes taken at discharge and laboratory data to exclude patients who were admitted for treatment. A total of 12 cases met these criteria. The emergency department did not have standing criteria for admission for hyperkalemia.

ABSTRACTION OF CLINICAL DATA

For the inpatients, charts, computerized records of laboratory data, and computerized pharmacy records were reviewed. The data obtained included age, sex, baseline medical conditions and medical history, and medications used at admission and discharge. The electrocardiograms obtained at admission were reviewed for the presence of peaked T waves, P-wave flattening, and widening of the QRS complex. The following laboratory values were obtained at admission: electrolytes, serum urea nitrogen, and creatinine. We also recorded the values of potassium, urea nitrogen, and creatinine obtained at least 1 week prior to admission (baseline values), the potassium concentration, and the potassium concentration obtained nearest to 4 weeks after the hyperkalemic episode. Treatments administered for the purpose of decreasing the potassium concentration were also recorded.

The severity of hyperkalemia was evaluated according to the criteria of Levinsky.9 Minimal hyperkalemia was defined as a potassium concentration higher than the laboratory’s upper limit of normal (5.2 mmol/L) and less than 6.5 mmol/L, with only minor electrocardiographic changes; moderate hyperkalemia, a potassium concentration between 6.5 and 8.0 mmol/L, with electrocardiogram changes limited to peaking of T waves; and severe hyperkalemia, a potassium concentration higher than 8.0 mmol/L. An elevated potassium concentration of any level accompanied by widened QRS complexes, atrioventricular block, or ventricular dysrhythmia. We considered hospital admission unnecessary when hyperkalemia was minimal and there were no other conditions necessitating admission.

For the outpatients, computerized records were reviewed. Age, sex, highest potassium concentration treated without hospitalization, and the concomitant values of urea nitrogen, creatinine, and bicarbonate were recorded. The previous (baseline) values from samples drawn at least 1 week earlier were also documented. We also recorded the potassium concentration obtained nearest to 4 weeks after the episode of hyperkalemia.

STATISTICAL ANALYSIS

The potassium concentration obtained at admission (inpatients) or the highest potassium concentration treated without admission (outpatients) was expressed as mean ± SD. Data were analyzed for statistical significance using a 2-tailed t test. The null hypothesis was rejected at a .05 level of significance.
were to determine the severity of hyperkalemia in a con-
secutive series of patients admitted for the treatment of hy-
perkalemia and to compare these inpatients to a similar con-
secutive series of outpatients. We then could develop 
reasonable criteria for admission for this condition. The 
study was not designed to test the relative safety of outpa-
tient and inpatient management of hyperkalemia, but the 
2 groups are illustrative of the important issues in decid-
ing on hospitalization.

RESULTS

BASELINE CHARACTERISTICS OF THE PATIENTS

Of 10961 admissions during the 18-month study pe-
riod, 11 patients (0.1%) were admitted for the treat-
ment of hyperkalemia. During the last 3 months of the 
study period, 12 patients received outpatient therapy for 
hyperkalemia. The patients in the 2 treatment groups 
were similar with respect to age and the values of urea 
nitrogen, creatinine, and potassium at baseline, as shown in 
Table 1. The baseline values showed hyperkalemia of 
similar magnitude in both groups. All patients in both 
groups were men.

In the inpatient group, a number of risk factors for 
hyperkalemia were found. As defined by a baseline cre-
atinine level of 132.6 µmol/L (1.5 mg/dL) or higher, all 
11 inpatients had chronic renal insufficiency, with 2 
undergoing long-term hemodialysis. Five patients 
(45%) had adult-onset diabetes mellitus. One patient 
(9%) had human immunodeficiency virus. Of the 11 
patients, 8 (73%) were taking at least 1 drug with the 
potential to cause hyperkalemia. Of these, 4 (36%) were 
using 1 agent that promotes hyperkalemia, 1 (9%) was 
using 2, and 3 (27%) were using 3. Some patients were 
taking more than 1 medication. Of the 11 patients, 
the most common hyperkalemia-promoting drugs 
used were ACEIs (5 [45%]), nonsteroidal anti-
inflammatory drugs (NSAIDs) (4 [36%]), and trimetho-
prim (3 [27%]). Some patients were taking more than 1 
medication.

The inpatients received medical attention for a va-
riety of reasons. Of 11 patients, 5 (45%) were contacted 
at their place of residence and asked to report to the emer-
gency department as a result of outpatient laboratory val-
ues demonstrating an elevated potassium concentra-
tion. An additional 3 patients (27%) had no complaints 
but were admitted from the clinic after routine labora-
tory results revealed an elevated potassium concentra-
tion. Only 3 patients (27%) had complaints in outpa-
tient clinics that prompted laboratory investigation, and 
the discovery of hyperkalemia was unrelated to the chief 
complaints. The baseline values of potassium and cre-
atinine and use of relevant medications of individual pa-
tients admitted for hyperkalemia are summarized in 
Table 2.

Table 1. Baseline Characteristics of the Patients*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Inpatients (n = 11)</th>
<th>Outpatients (n = 12)</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>70 ± 8.3</td>
<td>71 ± 9.2</td>
<td></td>
</tr>
<tr>
<td>Urea nitrogen, mmol/L (mg/dL)</td>
<td>14.5 ± 6.8</td>
<td>18.4 ± 6.8</td>
<td></td>
</tr>
<tr>
<td>Creatinine, µmol/L (mg/dL)</td>
<td>(40.6 ± 19.0)</td>
<td>(51.5 ± 19.0)</td>
<td></td>
</tr>
<tr>
<td>Potassium, mmol/L</td>
<td>221.0 ± 88.4</td>
<td>256.4 ± 150.3</td>
<td></td>
</tr>
<tr>
<td>(mg/dL)</td>
<td>(2.5 ± 1.0)</td>
<td>(2.9 ± 1.7)</td>
<td></td>
</tr>
<tr>
<td>(mg/dL)</td>
<td>5.4 ± 0.7</td>
<td>5.5 ± 0.5</td>
<td></td>
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</table>

*Baseline serum values represent the last value obtained at least 1 week 
before the hyperkalemic episodes. Baseline values were not available for 1 
patient in each group. None of the values in the 2 groups were statistically 
different.

As shown in Table 3, there were no significant differ-
ces between groups in acid-base status, electrolyte lev-
els, or renal function at the time that therapy for hyper-
kalemia was initiated. Remarkably, the mean potassium 
centralations of the 2 groups were the same. Treat-
ment was started on the basis of a mean ± SD potassium 
centration of 6.7±0.8 mmol/L in the inpatients, 
compared with a mean ± SD potassium concentration of 
6.7±0.5 mmol/L in the outpatients. The index potas-
sium concentration was from 5.5 to 8.4 mmol/L in the 
inpatients vs 6.2 to 8.0 mmol/L in the outpatients. The 
average change in potassium from baseline was similar 
as well. Indices of renal function were also comparable 
in the 2 groups. In both groups, the increases in the val-
ues of urea nitrogen and creatinine at the time of the 
hyperkalemic episode compared with baseline values 
were not statistically significant. As shown in Table 3, 
the decline in renal function was not significantly differ-
ent in the 2 groups. The serum bicarbonate levels were 
slightly low; however, whether this was due to meta-
abolic acidosis or respiratory alkalosis is not known since 
arterial blood gas values were not uniformly obtained. If 
metabolic acidosis was present, its contribution to 
hyperkalemia, if any, was small since the bicarbonate 
centralations in the 2 groups were similar. Laboratory 
values for the individual inpatients are summarized in 
Table 4.

Using Levinsky’s9 criteria, 5 of the 11 inpatients 
were admitted with minimal hyperkalemia, with a mean ± SD 
potassium concentration of 6.0±0.3 mmol/L at admi-
sion. Five patients were admitted for moderate hyper-
kalemia with a mean ± SD potassium concentration of 
7.0±0.4 mmol/L. A single patient with a potassium con-
centration of 8.4 mmol/L was admitted with severe hyperkalemia. Lack of electrocardiographic data lim-
ited classification of hyperkalemia in the inpatient group. 
Based on the potassium concentration alone, a similar 
proportion of the outpatients (7 of 12) had moderate or 
severe hyperkalemia compared with the inpatient group 
(6 of 11).

The subgroup of patients who were contacted at their 
homes because of hyperkalemia noted on routine labora-
tory determinations were called if they had a mean ± SD 
potassium concentration of 6.7±0.3 mmol/L. At admission, 
potassium concentration had spontaneously de-
creased by a mean of 0.1 mmol/L to a mean ± SD of 6.5±0.8 
mmol/L. At the time of admission, 2 patients had minimal 
hyperkalemia, and 3 had moderate hyperkalemia.
TREATMENTS

The type of therapy used in inpatients varied widely as reported elsewhere. All patients received sodium polystyrene sulfonate resin. Documentation that patients were provided with counseling regarding a low potassium diet was usually lacking. With all treatment modalities, failure to discontinue use of all hyperkalemia-promoting medications was common. While only 2 (25%) of 8 patients were treated without any adjustment in the use of potentially hyperkalemia-promoting medications, in 6 subjects (75%), the use of 1 or more such medications was continued throughout the episode. The use of NSAIDs was continued without adjustment in all 4 patients who took them at the time of admission. The use of ACEIs was discontinued in 4 (80%) of 5 patients during the hyperkalemic episode. In 3 (60%), the use of ACEIs was permanently discontinued, and in 1 (20%), it was reinstituted after discharge from the hospital.

OUTCOMES AND FOLLOW-UP

While intensive treatment often resulted in normokalemia (data not shown), subjects in both groups had hyperkalemia at follow-up 1 month later. The mean ± SD potassium concentration at follow-up was 5.3 ± 0.8 mmol/L in the inpatients and 5.4 ± 0.9 mmol/L in the outpatients.

There were no fatalities resulting directly from hyperkalemia in either group. However, one patient admitted for minimal hyperkalemia died of hospital-acquired bacteremia 16 days after admission. The patient admitted with severe hyperkalemia experienced cardiac arrest in the emergency department prior to the initiation of therapy but was discharged home without further incident. As shown in Table 4, this patient had the largest change in potassium concentration from baseline, the most severe acidemia, and the most severe azotemia of any subject in the study. Therapy for hyperkalemia resulted in hypokalemia in one inpatient. There were no other complications.

The mean ± SD length of stay for the inpatients was 6.9 ± 4.9 days, reflecting the presence of other medical problems identified at admission. Length of stay was as short as 3 days to as long as 17 days.

COMMENT

Although reviews of the pathophysiologic characteristics and management of hyperkalemia are available in abundance, specific thresholds for instituting various intensities of therapy are not always offered. We are unaware of any previous discussions of indications for hospital admission for hyperkalemia and undertook this review because we have often been asked for advice on this issue by internists, emergency department personnel, and house officers.

Because the intracellular to extracellular ratio of potassium is the major determinant of transmembrane potential, modest changes in potassium concentration can profoundly affect electrically excitable tissues. In skeletal muscle, profound hyperkalemia can lead to weak-
ness. However, the clinical myocardial effects are more important as hyperkalemia can lead to potentially serious dysrhythmias, including ventricular fibrillation and asystole. The grave consequences of these complications have led to recommendations that continuous electrocardiographic monitoring and treatment with all available therapeutic modalities are mandatory for any patient with potassium concentrations higher than 6.0 mmol/L. Hyperkalemia of this level is usually well tolerated, suggesting that such aggressive treatment is generally unwarranted. A potassium concentration of 6.0 mmol/L or higher may complicate 1.4% of all hospital admissions but is rarely life-threatening unless the potassium concentration is rapidly increasing. Moreover, potassium concentration is 6.0 mmol/L or higher in as many as 10% of prehemodialysis samples, and electrocardiographic changes are uncommon in patients with end-stage renal disease until the potassium concentration is 7.6 mmol/L or higher. Such data clearly indicate that admission of all patients with hyperkalemia to a monitored setting is both unnecessary and impractical.

Hyperkalemia can be well tolerated despite its effects on transmembrane potential, because membrane excitability is not determined solely by the difference between resting and threshold potentials. The toxic effects of hyperkalemia on the heart are enhanced by hypocalcemia, hyponatremia, and acidemia. In addition, membrane excitability may be more dependent on the rate of change in potassium concentration than on the concentration itself; initially, elevations increase membrane excitability, but with time persistent depolarization inactivates sodium channels, decreasing membrane excitability. As a result, rapid elevations in potassium concentration enhance the toxic effects, while slow elevations minimize those effects. This complex control of membrane excitability makes it impossible to predict the arrhythmogenic potential or expected morbidity and mortality from an isolated value of potassium. Similarly, one cannot recommend intensive treatment for all patients with a given elevated concentration of potassium.

Another factor that potentially increases the morbidity of hyperkalemia is the degree of renal insufficiency, since lower glomerular filtration rates lead to larger changes in potassium concentration with a given potassium load. Finally, the amount of potassium available for addition to the circulation is an important determinant of prognosis. Patients with elevated potassium levels that are derived from dietary intake can be counseled to avoid foods high in potassium content. At the other extreme, patients with necrotic bowel, hemolysis, or rhabdomyolysis may have hyperkalemia resistant even to hemodialysis.

The major conclusion from our data is that patients admitted to the hospital were clinically indistinguishable from patients treated as outpatients. Since the groups were small, we cannot exclude the possibility that larger samples would demonstrate statistically significant differences between the criteria for admitting or not admitting patients. However, even if our inpatient group were only a subgroup of a larger group with clearer indications for admission, they would constitute a group most noteworthy for being indistinguishable from the outpatient group. The 2 groups had similar potassium concentrations at baseline and similar degrees of renal insufficiency prior to the episode of hyperkalemia. At the time that hyperkalemia was identified, the potassium concentrations were also similar, and after discharge, the potassium concentrations were again indistinguishable. The justification for the decision to admit to the hospital or treat as an outpatient is not clearly evident in all but one of the hospitalized patients, the one with severe hyperkalemia. The physicians made these decisions based on what appear to be arbitrary criteria not discoverable from our review of the records. This finding reflects the lack of published criteria for hospitalization and for more intensive therapy in patients with hyperkalemia.

The therapies available for hyperkalemia are generally rapid in action and effective. Therapies intended to shift potassium from the extracellular to the intracellular space, such as nebulized β-agonists and insulin or calcium salts, which are given to stabilize depolarizing membranes, are readily administered in the emergency department. Definitive therapy requires removal of excess potassium from the body, using the bowel and kidneys to eliminate it. In otherwise stable outpatients, these therapies are effectively given in the emergency department or clinic. For patients undergoing long-term he-
Diabetic nephropathy has been proved, a minimal development of hyperkalemia. As the benefit of ACEIs despite the likelihood in each subject that other medications supervene. Such issues as tissue catabolism, decreased renal function, infection, or drug overdose are often adequate reason for admission, irrespective of the potassium concentration. Our study lacks the power to demonstrate whether the outcomes of the 2 groups differed and whether there was a survival advantage in the hospitalized patients. In our series of patients with hyperkalemia with a mean potassium concentration of 6.7 mmol/L, only 1 of 23 patients experienced a serious ventricular arrhythmia. That patient had the largest single change from baseline in potassium concentration accompanied by significant acidemia and azotemia. Unfortunately, no prospective data exist to demonstrate what degree of derangement in any of these variables would distinguish patients who would benefit from more short-term therapy or hospitalization.

At least 5 patients were admitted with minimal hyperkalemia. With a mean potassium concentration of only 6.0 mmol/L, minimal elevation from baseline potassium concentration, and no significant changes in the electrocardiogram, we found that only one of these patients—a patient with electrocardiographic and historical evidence suggestive of a recent myocardial infarction—objectively required admission. That indication was clearly for a reason distinct from the electrolyte level abnormality. Overall, the potassium concentration at initiation of therapy varied widely; some patients with a potassium concentration as low as 5.5 mmol/L were admitted to the hospital, and other patients with a potassium concentration as high as 8.0 mmol/L were successfully treated outside the hospital. The result was that the mean potassium concentration at initiation of therapy was identical in the outpatients and the inpatients.

The failure to recognize the importance of medications potentially affecting potassium homeostasis was common. Seventy-five percent of the patients continued to receive drugs that may promote hyperkalemia after the initiation of treatment. The use of NSAIDs was continued in every case, indicating that their role in hyperkalemia was not widely recognized. However, the use of ACEIs was frequently discontinued during the course of therapy. The use of ACEIs was permanently discontinued in the majority of patients with diabetes mellitus despite the likelihood in each subject that other medications or baseline renal insufficiency contributed to the development of hyperkalemia. As the benefit of ACEIs in mitigating the progression of chronic renal failure in diabetic nephropathy has been proved, a minimal degree of elevation in potassium concentration may be acceptable and preferable to permanent discontinuation of ACEI use.

The only fatality in the study was the result of hospital-acquired infection and might have been avoided with outpatient therapy. Moreover, the mean length of hospitalization in the inpatient group was 6.9 days. At an estimated cost of $1049 per bed per day at the Department of Veterans Affairs Medical Center, outpatient therapy saved about $6400 per patient, minus the minimal cost of outpatient follow-up. With the additional laboratory testing in the inpatient group and the costs of intravenous therapies, the actual savings with outpatient therapy may be substantially greater. These data indicate that considerations of efficacy, cost, and patient preference mandate outpatient treatment as the therapy of choice for routine hyperkalemia.

The data needed to develop useful criteria for hospitalization are not likely to be forthcoming. The relative efficacy of current therapy for hyperkalemia would cause measurable end points to be extremely uncommon, making a convincing randomized trial of therapies impractical. One study of 242 episodes of potassium concentration higher than 6.0 mmol/L found no serious arrhythmia or death in these hospitalized patients. Therefore, we are led to believe that hospital admission is not routinely indicated in the treatment of hyperkalemia.

To summarize, our results indicate that treatment for the same degree of hyperkalemia varies widely among physicians. Hospitalization is usually not necessary for the successful treatment of hyperkalemia. We believe that these findings reflect the diversity of expert opinion and lack of experimental evidence. In the absence of outcome data, we advocate outpatient treatment for patients with a potassium concentration of 6.5 mmol/L or lower, stable or slowly increasing potassium concentration, minimal changes in the electrocardiogram, and only mild acidemia. Patients with moderate hyperkalemia (concentrations between 6.5 and 8.0 mmol/L, with changes in the electrocardiogram limited to peaking of T waves) can be treated in the emergency department if oral sodium polystyrene sulfate resin can be administered, culpable drugs suspended, and supervening medical problems not identified. Acute deterioration in renal function is a significant indication for admission in its own right and may potentiate other factors that contribute to hyperkalemia. Discharge from the emergency department can be safely considered when the levels obtained from repeated potassium testing have decreased. Severe hyperkalemia (higher than 8.0 mmol/L, with more significant arrhythmia) would most safely be managed in a monitored, hospital setting. Where possible, treatment should start with dietary counseling, potassium-binding resins, and withholding medications that cause hyperkalemia. Oral administration of sodium citrate may be useful in patients with acidemia and furosemide may be useful in those with edema or hypertension, particularly in conjunction with sodium citrate administration. Hospital admission, nebulized albuterol, intravenous administration of calcium gluconate, insulin, and sodium bicarbonate should be reserved for patients with changes on the electrocardiogram, rapid increases in potassium concentration, or large changes from baseline potassium concentrations. Finally, the use of ACEIs might be carefully reinstated after the acute hyperkalemic episode unless use clearly causes intolerable increases in potassium concentration.
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