CHRONIC HYPOKALEMIA: WHAT’S IN A NAME?

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Potassium Homeostasis

External potassium balance is determined by rate of K intake (100 meq/day) and rate of urinary (90 meq/day) and fecal excretion (10 meq/day).

Internal potassium balance depends on distribution of K between muscle, bone, liver, and red blood cells (RBC) and the extracellular fluid (ECF).

Modified from Stanton and Giebisch
POTASSIUM HOMEOSTASIS

• Healthy persons are in potassium balance, which means that the daily intake of potassium is equal to the amount excreted.

• In children, normal daily potassium requirements vary by age. However, they are estimated at approximately 2 mEq per 100 kcal of energy requirement throughout most of childhood (Linshaw, 1987).

• An adult’s dietary intake varies from approximately 50 to 150 mEq per day. Potassium is present in sufficient quantities in most fruits, vegetables, meat, and fish.
CHRONIC HYPOKALEMIA: WHAT’S IN A NAME?

- Hypokalemia is generally defined as a serum potassium level of less than 3.5 mEq/L (3.5 mmol/L).
- Moderate hypokalemia is a serum level of 2.5-3 mEq/L.
- Severe hypokalemia is a level of less than 2.5 mEq/L.
- Hypokalemia is a potentially life-threatening imbalance that may be iatrogenically induced.
Physiologic Roles of K

- Major ion determining resting membrane electrical potential, which in turn, limits and opposes K efflux.
- Major osmotically active cation in ICF and participates in cell volume regulation (exits with Cl when cells swell).
- Changes in K concentrations (particularly in ECF) have marked effects on cell excitability (heart, brain, nerve, muscle).
- Critical for enzyme activities and for cell division and growth.
- Intracellular K participates in acid base regulation through exchange for extracellular H and by influencing the rate of renal ammonium production.
CLINICAL MANIFESTATIONS OF HYPOKALEMIA

- Hypokalemia can cause a variety of clinical manifestations because of alterations in the excitability of neuromuscular tissues.

- A decrease in ECF K concentration leads to hyperpolarization of the cell membrane, causing the cell to become less sensitive to exciting stimuli.
CLINICAL MANIFESTATIONS OF HYPOKALEMIA

- Hypokalemia can cause a variety of clinical manifestations because of alterations in the excitability of neuromuscular tissues.

- A decrease in ECF K concentration leads to hyperpolarization of the cell membrane, causing the cell to become less sensitive to exciting stimuli.

- Clinically, this effect accounts for the association of hypokalemia and muscle weakness which can be severe enough to cause paralysis, as in patients with hypokalemic dRTA.

- Myopathy also may occur, which in its most severe form can lead to frank rhabdomyolysis and kidney failure.
CLINICAL MANIFESTATIONS OF HYPOKALEMIA

Gastrointestinal tract
- Altered gastrointestinal motility (nausea, vomiting, constipation, paralytic ileus)
- Worsening of hepatic encephalopathy

Genitourinary tract
- Hypotonic bladder

Respiratory system
- Respiratory acidosis secondary to respiratory muscle weakness

Endocrine system
- Insulin resistance and impairment in insulin release
Effect of hypokalemia on myocardial cells

- Increased excitability
- Prolonged exaltation phase
- Short absolute refractory period
- Reduced conductivity
- Increased autorhythmicity

The above changes make it easy to produce arrhythmia (increased heart rate, ectopic beats from Purkinje fiber and ventricular muscle).
Broad and flat T wave appears because the potassium permeability in hypokalemia is reduced, the rate of repolarization is reduced. The phase 3 is prolonged.

Prolonged QRS complex is caused by reduced conductivity.

The typical electrocardiogram change is ST depression, T-wave flattening, and an increase in amplitude of the U wave.
Hypokalemia effect on acid-base balance

Hypokalemia leads to metabolic alkalosis.

When ECF [K+] ECF decreases, the K from the ICF moves out of the cells, at the same time, H⁺ moves into the cells to maintain electroneutrality.

Then the [H⁺] in ECF will be reduced, leading to metabolic alkalosis.

(Depending on the primary disease)
Effect of hypokalemia on the kidney

- Pathologic study found swelling, proliferation, vacuolation in proximal tubular cells, renal tubular cells can not produce sufficient cAMP necessary for ADH to work, so the tubules lose its urine concentrating ability.

  - Increased urine volume
  - Decreased urine specific gravity
  - Polydipsia, polyuria
Effect of hypokalemia on glucose metabolism

Because insulin release is regulated partially by serum potassium, hypokalemia can lead to glucose intolerance.

For every 1-mEq/L decrease in serum potassium level, there is an approximate 10-mg/dL (0.56-mmol/L) increase in glucose level.

- Metabolic complications of hypokalemia include impaired urinary concentrating ability and decreased β-cell release of insulin, resulting in glucose intolerance.

- Administration of a carbohydrate load to malnourished patients can unmask total-body deficits of potassium, phosphate, and magnesium because of insulin-mediated shifts into the intracellular compartment.

Causes of Hypokalemia

• Inadequate potassium intake
• Increased potassium excretion
• A shift of potassium from the extracellular to the intracellular space.

• Clinical history, physical examination with particular emphasis on determination of volume status, and assessment of acid-base status will allow the cause of hypokalemia to be readily determined in most cases.
A 38-year-old woman is referred for refractory hypokalemia. Three months ago, she presented to her primary care physician reporting weakness, and a routine metabolic profile laboratory test showed the following values: sodium, 138 mEq/L (138 mmol/L); potassium, 2.5 mEq/L (2.5 mmol/L); chloride, 90 mEq/L (90 mmol/L); bicarbonate, 32 mEq/L (32 mmol/L); serum creatinine, 0.9 mg/dL (79.56 μmol/L); serum urea nitrogen, 20 mg/dL (7.14 mmol/L); and estimated GFR of 81 mL/min/1.73 m² (1.35 mL/s/1.73 m²), calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.
During the next 12 weeks, she was treated with oral potassium, 20 mEq/d; losartan, 50 mg/d; and spironolactone, 25 mg/d. Despite this therapy, serum potassium levels remained in the range of 2.5-3.0 mEq/L (2.5-3.0 mmol/L). On evaluation, blood pressure was 110/72 mm Hg and heart rate was 88 beats/min. The rest of the physical examination findings were unremarkable, with no skin discoloration or edema. Laboratory test results were unchanged from previous measurements.
Plasma renin activity was 6.5 ng/mL/h (1.8 ng/L/s; reference range, 0.5-4 ng/mL/h [0.1-1.1 ng/L/s])

Plasma aldosterone concentration was 35 ng/dL (0.97 nmol/L; reference range, 4-31 ng/dL [0.11-0.86 nmol/L])

Plasma magnesium level was 1.8 mEq/L (0.9 mmol/L; reference range, 1.3-2.1 mEq/L [0.65-1.05 mmol/L]).

Urine electrolytes showed the following values: sodium, 83 mEq/L (83 mmol/L); potassium, 40 mEq/L (40 mmol/L); and chloride, <10 mEq/L (<10 mmol/L).
Assessment of urinary potassium excretion allows one to determine whether hypokalemia is due to renal or extrarenal causes.

Renal potassium handling can be assessed using a 24-hour urine collection or spot urine sample determining potassium-creatinine ratio.

Twenty-four–hour urinary potassium excretion < 20 mEq or a spot urine potassium-creatinine ratio < 1 suggests an extrarenal cause of hypokalemia.
APPROACH TO HYPOKALEMIA

Decreased serum K

Decreased total body K

Urinary potassium <20 mEq/L
(Extrarenal losses)

Metabolic acidosis
GI losses: diarrhea, laxatives

Normal pH
Decreased intake/GI losses
Villous adenoma
Laxatives, geophagia

Metabolic alkalosis
Renal tubular acidosis
Ureterosigmoidostomy
Diabetic ketoacidosis

Metabolic acidosis
Villous adenoma

Variable pH
ATN recovery
Postobstructive diuresis
Drugs, e.g., platinum, aminoglycosides

Metabolic alkalosis

Urinary potassium >20 mEq/L
(Renal Losses)

Urinary chloride <20 mEq/L
Diuretics (after drug effect has dissipated)
Vomiting
Posthypercapnea

Urinary chloride >20 mEq/L

Hypertension

Normal or decreased BP
Diuretics, severe K depletion
Barter's or Gitelman's syndromes
Mg depletion

Elevated aldosterone
Primary aldosteronism

Secondary aldosteronism
Increased cortisol
Cushing's syndrome

Normal aldosterone
Normal cortisol
Liddle's syndrome
AME
TTKG IN HYPERKALEMIA

TTKG  =  \frac{\text{Urine K}}{\text{(Urine osmolality)}} \div \frac{\text{(Plasma osmolality)}}{\text{Plasma K}}

TTKG in normal subjects on a regular diet is 8 to 9, and rises to above 11 with a potassium load.

During hyperkalemia, the TTKG should be greater than 7 - lower values suggest hypoaldosteronism.

During hypokalemia, the TTKG should be < 3 – greater values suggest renal potassium wasting.

In patients with hypo and hyper K, the degree of renal K excretion in the distal nephron can be estimated by calculating the TTKG

For this formula to be accurate, urine osmolality must exceed plasma osmolality and urine sodium should be greater than 25 mmol/L.
Renal excretion of potassium continuously

<table>
<thead>
<tr>
<th>Amount of $K^+$ Excretion</th>
<th>Extreme Dietary Restriction of Potassium Alone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Potentially can lead to hypokalemia over time.</td>
</tr>
<tr>
<td>38~150 mmol/day</td>
<td>Although the kidney can elaborate urine virtually free of sodium in response to dietary sodium restriction, it can decrease urinary potassium excretion to only approximately 15 mEq/d in response to a potassium-free diet.</td>
</tr>
<tr>
<td>No K intake</td>
<td></td>
</tr>
<tr>
<td>1~3 day</td>
<td>50 mmol/day</td>
</tr>
<tr>
<td>4~7 day</td>
<td>20 mmol/day</td>
</tr>
<tr>
<td>10 day</td>
<td>5~10 mmol/day</td>
</tr>
</tbody>
</table>
Box 1. Causes of Hypokalemia Due to Internal Cellular Shift

- Alkalosis (effect is trivial)
- Insulin administration
- $\beta_2$-Adrenergic stimulation
  - Stress-induced epinephrine release
  - Drugs: theophylline intoxication, ritodrine, terbutaline, albuterol, clenbuterol
- Anabolism
  - Treatment of pernicious anemia
  - Rapidly growing leukemias and lymphomas
- Hypokalemic periodic paralysis
  - Acquired in association with hyperthyroid state
  - Familial
- Drugs/toxins/herbs
  - Barium intoxication
  - Chloroquine intoxication
  - Cesium salts (reported in herbal preparations marketed as antitumor agent)
Inadequate potassium intake (<40 meq/L)
- Eating disorders
- Alcoholism

Urinary loss
- Diuretics: loop and thiazide
- Antimicrobials: amphotericin B, cisplatin, aminoglycoside, piperacillin, ticarcillin
- Osmotic diuresis (diabetic ketoacidosis, mannitol)
- Hypomagnesemia
- Cushing syndrome
- Primary mineralocorticoid excess
- Bartter syndrome or Gitelman syndrome

Renal Regulation of Potassium

Regulatory influences

Secretion
K intake
ALDO, ADH
Flow
Alkalosis

Reabsorption
K loss

65%
R
DCT

PCT

R
25%
TAL

R
CCT

S
MCD

R

Principal cell

Intercalated cell


AJP - Renal Physiology

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Under normal circumstances, delivery of sodium (Na+) to the distal nephron is associated inversely with serum aldosterone levels. For this reason, urinary potassium (K+) excretion is kept independent of changes in extracellular fluid volume. Hypokalemia due to renal potassium wasting can be explained by pathophysiologic changes that lead to coupling of increased distal sodium delivery and aldosterone or aldosterone-like effects. When evaluating a patient who has hypokalemia caused by urinary potassium wasting, one must determine whether the primary disorder is an increase in mineralocorticoid activity or in distal sodium delivery.
Assess Renal K⁺ Handling

Decreased K⁺ excretion

- Cell shift
- Extrarenal loss

High

- Renin, Aldosterone

↑ Renin, ↑ Aldosterone

- Renal artery stenosis
- Renin secreting tumor

↓ Renin, ↑ Aldosterone

- Adrenal adenoma
- Bilateral cortical hyperplasia
- Glucocorticoid suppressible hyperaldosteronism

↓ Renin, ↓ Aldosterone

- Cushing syndrome
- 11β-hydroxylase deficiency
- 17α-hydroxylase deficiency
- Syndrome of apparent mineralocorticoid excess
- Liddle syndrome

Increased K⁺ excretion

Assess BP, EABV

Low-Normal

- Serum [HCO₃⁻]

Low

- Proximal RTA
- Distal RTA

High

- Urine Cl⁻

Low

- Non-reabsorbable anion
- Vomiting
- Carbenicillin, ticarcillin

High

- Loop diuretics
- Thiazide diuretics
- Mg⁺⁺ deficiency
- Bartter syndrome
- Gitelman syndrome
### PRIMARY INCREASE IN MINERALOCORTICOID ACTIVITY

- Primary increases in renin secretion, primary increases in aldosterone secretion, or increases in a nonaldosterone mineralocorticoid or increased mineralocorticoid-like effect.

- In all these conditions, extracellular fluid volume is expanded and hypertension typically is present.

- Workup of these patients is extremely important because these disorders represent the most common causes of curable hypertension.

### PRIMARY INCREASES IN DISTAL SODIUM DELIVERY

- Most frequently are caused by diuretics, which act proximal to the cortical collecting duct.

- Increased delivery also can be the result of nonreabsorbed anions, such as bicarbonate, as with active vomiting or proximal renal tubular acidosis.

- The inability to reabsorb these anions in the proximal tubule results in increased delivery of sodium to the distal nephron. Because these anions also escape reabsorption in the distal nephron, a more lumen-negative voltage develops and the driving force for potassium excretion into the tubular fluid is enhanced.
Assess Renal $K^+$ Handling

Decreased $K^+$ excretion
- Cell shift
- Extrarenal loss
  - High
    - Renin, Aldosterone
      - ↑ Renin, ↑ Aldosterone
        - Renal artery stenosis
        - Renin secreting tumor
      - ↓ Renin, ↑ Aldosterone
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        - Bilateral cortical hyperplasia
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        - Cushing syndrome
        - $11\beta$-hydroxylase deficiency
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        - Syndrome of apparent mineralocorticoid excess
        - Liddle syndrome

Increased $K^+$ excretion
- Assess BP, EABV
  - Low-Normal
    - Serum $[HCO_3^-]$
      - Low
        - Proximal RTA
        - Distal RTA
      - High
        - Urine $Cl^-$
          - Low
            - Non-reabsorbable anion
              - Vomiting
              - Carbenicillin, ticarcillin
          - High
            - Loop diuretics
            - Thiazide diuretics
            - $Mg^{++}$ deficiency
            - Bartter syndrome
            - Gitelman syndrome
HYPOKALEMIA IN PROXIMAL RTA

- In proximal renal tubular acidosis, the threshold for bicarbonate reabsorption is decreased, resulting in self-limited bicarbonaturia.

- The loss of sodium bicarbonate in urine leads to volume depletion, which in turn activates the renin-angiotensin-aldosterone system. The coupling of increased aldosterone levels with increased distal sodium delivery results in urinary potassium wasting.

- In the steady state, when virtually all filtered bicarbonate is reabsorbed in the proximal and distal nephron, urinary potassium wasting is minimal and the degree of hypokalemia tends to be mild.

- In contrast, treatment of metabolic acidosis with bicarbonate improves the acidosis, but worsens the degree of hypokalemia.
## Distinctions between Bartter's and Gitelman's syndromes

<table>
<thead>
<tr>
<th></th>
<th>Bartter's syndrome</th>
<th>Gitelman's syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Localization of defect</strong></td>
<td>Ascending limb of Henle</td>
<td>Distal tubule</td>
</tr>
<tr>
<td><strong>Age of presentation</strong></td>
<td>Prenatal, during infancy, early childhood</td>
<td>Mostly late childhood or at adult age</td>
</tr>
<tr>
<td><strong>Biochemical differences</strong></td>
<td>Serum magnesium may be decreased</td>
<td>Serum magnesium decreased</td>
</tr>
<tr>
<td></td>
<td>Urinary excretion of calcium increased or normal</td>
<td>Urinary excretion of calcium reduced</td>
</tr>
<tr>
<td><strong>Molecular differences</strong></td>
<td>Na-K-2Cl cotransporter (NKCC2) or apical K channel (ROMK) or basolateral C1 channel (CICNKB) in thick ascending limb of Henle</td>
<td>Na-Cl cotransporter in the distal tubule</td>
</tr>
<tr>
<td><strong>Functional studies</strong></td>
<td>Concentrating capacity severely impaired</td>
<td>Concentrating capacity normal or slightly impaired</td>
</tr>
<tr>
<td></td>
<td>GFR may be normal, decreasing or declining</td>
<td>GFR is normal</td>
</tr>
</tbody>
</table>

Renal potassium excretion is increased by the following factors:

- Aldosterone
- High sodium delivery to the collecting duct (eg, diuretics)
- High urine flow (eg, osmotic diuresis)
- High serum potassium levels
- Delivery of negatively charged ions to the collecting duct (eg, bicarbonate)
A 38-year-old woman is referred for refractory hypokalemia. Three months ago, she presented to her primary care physician reporting weakness, and a routine metabolic profile laboratory test showed the following values: sodium, 138 mEq/L (138 mmol/L); potassium, 2.5 mEq/L (2.5 mmol/L); chloride, 90 mEq/L (90 mmol/L); bicarbonate, 32 mEq/L (32 mmol/L); serum creatinine, 0.9 mg/dL (79.56 μmol/L); serum urea nitrogen, 20 mg/dL (7.14 mmol/L); and estimated GFR of 81 mL/min/1.73 m² (1.35 mL/s/1.73 m²), calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.
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The treatment of hypokalemia has 4 facets:

- **Reduction of potassium losses**
- Replenishment of potassium stores
- Evaluation for potential toxicities
- Determination of the cause to prevent future episodes, if possible

- Discontinue diuretics/laxatives
- Use potassium-sparing diuretics if diuretic therapy is required (eg, severe heart failure)
- Treat diarrhea or vomiting
- Administer H2 blockers to patients receiving NGT suctioning
- Control hyperglycemia if glycosuria is present
The treatment of hypokalemia has 4 facets:
- Reduction of potassium losses
- Replenishment of potassium stores
- Evaluation for potential toxicities
- Determination of the cause to prevent future episodes, if possible

- For every 1 mEq/L decrease in serum K, the potassium deficit is approximately 200-400 mEq.
- Oral therapy
  - Generally safer
  - Degree of K+ depletion does not correlate well with the plasma K+
- KCl is usually the preparation of choice
**Box 2. Suggested Management for Hypokalemia**

- **Mild to moderate hypokalemia** (serum potassium concentration, 3.0-3.5 mEq/L)
  - Treat the underlying disorder if possible
  - Treat the hypokalemia with 60-80 mEq/d of potassium chloride orally in divided doses
  - Recheck serum potassium concentration after replacement therapy and adjust treatment accordingly

- **Severe hypokalemia** (serum potassium concentration < 3.0 mEq/L)
  - Preferred: potassium chloride, 40 mEq, orally every 3-4 hours for 3, recheck serum potassium concentration as needed, and continue replacement as necessary
  - If necessary: intravenous potassium chloride (10-20 mEq/h) in the setting of cardiac arrhythmias, digitalis toxicity, and recent or ongoing cardiac ischemia. This should be done with continuous cardiac monitoring. Recheck serum potassium concentration every 2-4 h to ensure that serum potassium concentration is > 3.5 mEq/L
GENERAL PRINCIPLES OF HYPOKALEMIA MANAGEMENT

Hypokalemia <3.5 mEq/L

Assess for life-threatening complications (arrhythmias, muscle weakness, respiratory failure)

YES

5-10 mEq KCl over 15-20 min. May repeat until symptoms are resolved.

Resolve

Assess K+ deficit if any

Generally each ↓0.3 mEq in serum [K+] = ~100 mEq total body deficit.

Choose K+ preparation

Choose route & rate of administration

Can the patient take oral K+? Does the patient have a functioning bowel and a serum K+ >2.5 mEq/L?

YES

• 40-120 mEq/d KCl given orally in 2-4 divided doses.
• In certain situations, combined oral + IV may be needed.

NO

• 20-40 mEq/L IV KCl; infused at a rate of 10 mEq/h & monitor [K+] ~2-4 h.
• Higher concentration can be used carefully for patients with fluid overload (eg, 10 mEq/100 mL).
• Higher infusion rates (20 mEq/h) require cardiac monitoring & preferably central venous access.
• No more than 60 mEq should be given before serum [K+] is rechecked.

KCl: Most effective & preferred preparation.

Potassium phosphate: Effective with concomitant phosphate depletion. Eg, diabetic ketoacidosis.

Potassium bicarbonate, potassium acetate: Effective with concomitant metabolic acidosis.
The timing of a repeat serum potassium level depends on the severity of the initial value, the patient’s symptoms, and the form of potassium administered to the patient.

In a symptomatic patient who receives an intravenous dose of KCl, the dose should be repeated without measuring a serum value if the patient’s symptoms persist.

If the symptoms resolve, the serum potassium level can be obtained 1 hour after completion of an intravenous dose (Schaefer & Wolford, 2005).

In clinical situations in which an oral dose is administered based on a low serum value, in the absence of clinical symptoms, the serum level can be repeated the next day.
ACID-BASE AND ELECTROLYTE TEACHING CASE

A Physiologic-Based Approach to the Evaluation of a Patient With Hypokalemia

Biff F. Palmer, MD

Hypokalemia is a common electrolyte disorder. Transient causes of hypokalemia are due to cell shift, whereas sustained hypokalemia is caused by either inadequate intake or excessive potassium loss. Evaluation of the intake, distribution, and excretion of potassium should include the following: (1) a careful history, including use of drugs, medications, and the presence of vomiting or diarrhea; (2) physical examination, including orthostatic changes in blood pressure and heart rate; and (3) measurement of urine and plasma electrolytes. Urinary potassium wasting is caused by pathophysiologic conditions that couple increased distal sodium delivery with increased plasma aldosterone levels or aldosterone-like effects. If urinary potassium loss is caused by a primary increase in distal sodium delivery, a primary increase in distal delivery of sodium may be identified. The next step is to determine whether the primary increase in distal delivery of sodium is caused by increased aldosterone levels or aldosterone-like effects.

AJKD

Acid-Base and Electrolyte Teaching Case

A Physiologic-Based Approach to the Treatment of a Patient With Hypokalemia

Abdo Asmar, MD,1,2 Rajesh Mohandas, MD,1 and Charles S. Wingo, MD1,3

Hypokalemia is common and can be associated with serious adverse consequences, including paralysis, ileus, cardiac arrhythmias, and death. As a result, the body maintains serum potassium concentration within very narrow limits by tightly regulated feedback and feed-forward systems. Whereas the consequences of symptomatic hypokalemia and severe potassium depletion are well appreciated, chronic mild hypokalemia can accelerate the progression of chronic kidney disease, exacerbate systemic hypertension, and increase mortality. Persistent hypokalemia may reflect total-body potassium depletion or increased renal potassium clearance. In a patient with simple potassium depletion, potassium replacement therapy should correct serum potassium concentration, but may have little effect when renal potassium clearance is abnormally increased from potassium wasting. In such cases, the addition of potassium-sparing diuretics might be helpful. Serum potassium concentration is an inaccurate marker of total-body potassium deficit. Mild hypokalemia may be associated with significant total-body potassium deficits and conversely, total-body potassium stores can be normal despite serum hypokalemia.
The Pediatric Nephrology Society of the Philippines, Inc.

20th ANNUAL CONVENTION

Common Questions in Pediatric Nephrology: Realigning Recommendations with Reality

PRECONVENTION
November 22, 2015
CONVENTION PROPER
November 23-24, 2015

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