K⁺: Hi and Lo ECG in hyper- and hypokalaemia Management in children and special considerations

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Royal Hospital for Children Glasgow, UK
Honorary Senior Clinical Lecturer, University of Glasgow
### Intracellular and extracellular fluid electrolyte content

<table>
<thead>
<tr>
<th>Electrolytes</th>
<th>Intracellular Levels</th>
<th>Extracellular Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>K⁺</strong></td>
<td>145 mMol</td>
<td>4 mMol</td>
</tr>
<tr>
<td><strong>Na⁺</strong></td>
<td>5-15 mMol</td>
<td>135-145 mMol</td>
</tr>
<tr>
<td><strong>Cl⁻</strong></td>
<td>5-15 mMol</td>
<td>103 mMol</td>
</tr>
<tr>
<td><strong>Ca⁺</strong></td>
<td>0.0002 mMol</td>
<td>2 mMol</td>
</tr>
<tr>
<td><strong>Mg²⁺</strong></td>
<td>25 mMol</td>
<td>1-2 mMol</td>
</tr>
<tr>
<td><strong>Protein</strong></td>
<td>16 mMol</td>
<td>2 mMol</td>
</tr>
<tr>
<td><strong>H⁺</strong></td>
<td>10⁻⁷.² mMol</td>
<td>10⁻⁷.⁴ mMol</td>
</tr>
<tr>
<td><strong>HCO₃⁻</strong></td>
<td>8-12 mMol</td>
<td>24-30 mMol</td>
</tr>
</tbody>
</table>
Na – main phase 0 depolarising current

K – main repolarising current (but also phase 4)
Ion channels in atrial, ventricular and specialised conduction tissue

SA-nodal phase 4 depolarization
Sinoatrial node
Atrial myocyte
AV node
Endocardial cell
Purkinje fiber

Epicardial cell
Midmyocardial cell

Surface ECG

ATRIAL

0 mV

VENTRICLE

200 ms

Ion currents
Channel protein
SCNA families
CACNA families
Kv4.3 + Kv1.4 + KCNP2
Kv1.5
not expressed

(H)ERG + MIRP1
KvLQT1 + MinK
Kir 2.1 - 2.4
GIRK4
not expressed
Kir6.2 + SUR2A

l_{Na}
l_{Ca}
l_{K1}
l_{K2}
l_{Kur}
l_{Kr}
l_{Kr1}
l_{Kmab}
l_{KATP}
l_{KATP}
“Funny” K+ and Ca++ currents are responsible for automaticity
Ion channels may consist of several subunits and form macromolecular complexes ("channelosomes")

Excitation-contraction coupling


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Excitation-contraction coupling

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<table>
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<tr>
<th>Age Range</th>
<th>Normal serum potassium levels (mEq/L or mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature infant</td>
<td>4.5 to 7.2</td>
</tr>
<tr>
<td>Newborn</td>
<td>3.7 to 5.2</td>
</tr>
<tr>
<td>Child</td>
<td>3.5 – 5.8</td>
</tr>
<tr>
<td>Adult</td>
<td>3.5 – 5.5</td>
</tr>
<tr>
<td>Child &gt;1 year old</td>
<td>3.6 to 5.2*</td>
</tr>
</tbody>
</table>

*Mayo Medical Laboratories: “Reference values have not been established for patients that are less than 12 months of age”

Causes of Hyperkalaemia

**Causes of Increased Serum K+**

- **The HYPERKALEMIA “Machine”**
  - Medications – ACE inhibitors, NSAIDS
  - Acidosis – Metabolic and respiratory
  - Cellular destruction – Burns, traumatic injury
  - Hypoaldosteronism, hemolysis
  - Intake – Excessive
  - Nephrons, renal failure
  - Excretion – Impaired

**HYPERKALEMIA** (Serum K+ > 5.5)

- **PRODUCTION ISSUE** (Too much K+ being put into the serum)
  - Cell Lysis
  - Shift of Intracellular K+ into Serum
- **CLEARANCE ISSUE** (Too little K+ being taken out of the serum)
  - Increased K+ Intake
  - Impaired Renal Excretion of K+ (Most common cause/contributing factor)
Elevated serum K+ effects

• Suppression of SA node automaticity causing bradycardia
• Slowing down conduction down atrio-ventricular node and His-Purkinje system causing conduction blocks
• Serum potassium level of > 9.0 mEq/L causes cardiac arrest due to
  • Asystole
  • Ventricular fibrillation
  • PEA with bizarre wide complex rhythms

Serum K+ level may not correlate with the ECG changes. Patients with relatively normal ECGs may experience sudden hyperkalaemic cardiac arrest
<table>
<thead>
<tr>
<th>Serum Potassium</th>
<th>Typical ECG Appearance</th>
<th>Possible ECG Abnormalities</th>
</tr>
</thead>
</table>
| Mild (5.5-6.5 mEq/L) | [Graph of ECG] | Peaked T waves  
Prolonged PR segment |
| Moderate (6.5-8.0 mEq/L) | [Graph of ECG] | Loss of P wave  
Prolonged QRS complex  
ST-segment elevation  
Ectopic beats and escape rhythms |
| Severe (> 8.0 mEq/L) | [Graph of ECG] | Progressive widening of QRS complex  
Sine wave  
Ventricular fibrillation  
Asystole  
Axis deviations  
Bundle branch blocks  
Fascicular blocks |
Mild hyperkalaemia

- Serum potassium > 5.5 mEq/L is associated with repolarization abnormalities:
  - Peaked T waves (usually the earliest sign of hyperkalaemia)
Moderate hyperkalaemia

- Serum potassium > 6.5 mEq/L is associated with progressive paralysis of the atria:
  - P wave widens and flattens
  - PR segment lengthens
  - P waves eventually disappear
Severe hyperkalaemia

- Serum potassium > 7.0 mEq/L is associated with conduction abnormalities and bradycardia:
  - Prolonged QRS interval with bizarre QRS morphology
  - High-grade A-V block with slow junctional and ventricular escape rhythms
  - Any kind of conduction block (bundle branch blocks, fascicular blocks)
  - Sinus bradycardia or slow AF
  - Development of a sine wave appearance (a pre-terminal rhythm)
Severe hyperkalaemia: Sine wave
# Treatment - Principles

<table>
<thead>
<tr>
<th>Effect</th>
<th>Agent</th>
<th>Dose</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Membrane Stabilization</td>
<td>Calcium Gluconate (10%)</td>
<td>10mL IV over 10 min</td>
<td>Immediate</td>
<td>30 – 60 minutes</td>
</tr>
<tr>
<td></td>
<td>Hypertonic (3%) Normal Saline</td>
<td>50mL IV push</td>
<td>Immediate</td>
<td>Unknown</td>
</tr>
<tr>
<td>Shifters</td>
<td>Insulin (Short Acting)</td>
<td>10 units IV push with 25 – 40 g dextrose (50% solution)</td>
<td>20 minute</td>
<td>4 – 6 hours</td>
</tr>
<tr>
<td></td>
<td>Albuterol</td>
<td>10 – 20 mg in 4 mL of Normal Saline nebulized over 10 minutes</td>
<td>30 minute</td>
<td>2 hours</td>
</tr>
<tr>
<td>Excreters</td>
<td>Furosemide</td>
<td>40 – 80 mg IV x1</td>
<td>15 minute</td>
<td>2 -3 hours</td>
</tr>
<tr>
<td></td>
<td>Sodium Bicarbonate</td>
<td>150mmol/L IV at variable rate</td>
<td>Hours</td>
<td>Duration of Infusion</td>
</tr>
<tr>
<td></td>
<td>Sodium Polystyrene Sulfonate</td>
<td>15 – 30 g in 15 – 30 mL (70% sorbitol orally)</td>
<td>&gt; 2 hours</td>
<td>4 – 6 hours</td>
</tr>
<tr>
<td>Definitive</td>
<td>Hemodialysis</td>
<td>-----</td>
<td>Immediate</td>
<td>3 hours</td>
</tr>
</tbody>
</table>
1. **Stabilise the myocardial cell membrane**

- Calcium does not lower the serum K+ level but stabilises cellular membrane
- Use Calcium if wide QRS or sine wave pattern, or in hyperkalaemic cardiac arrest
- Complications: bradycardia, hypotension, peripheral vasodilation.
- Avoid calcium in digoxin toxicity, use magnesium as alternative
  - **Calcium Chloride** – more potent than Ca Gluconate, irritates veins – thrombophlebitis if given peripherally
  - **Calcium Gluconate** – less potent, less irritating to veins
2. **Shift potassium into the cell**

- **Insulin & Glucose**
  - Reduces K+ by 1mmol/L and ECG changes within the first hour

- **Sodium Bicarbonate**
  - Effective at driving K+ into cells if the patient is acidotic
  - Onset of effect in 30-60 min, continues for several hours

- **Salbutamol**
  - Beta2 agonist therapy lower K+ via IV or nebulizer route.
  - Salbutamol can lower K+ level by 1mmol/L in about 30 minutes, and maintain it for up to 2 hours.
  - Very effective in fluid overloaded renal patients
3. **Stimulate excretion of K+**

- **Calcium Resonium**
  - Large insoluble molecule, binds K+ in large bowel and facilitates excretion with faeces. Effects take 2-3 hours

- **Frusemide**
  - Potassium-wasting loop diuretic. Requires functioning kidneys

- **Normal Saline**
  - Used to stimulate diuresis hence urinary K+ loss by volume expansion, increasing renal perfusion and urinary output. Cautious use in patients with renal & heart failure

- **Dialysis**
  - Gold standard for removing potassium from the body. Provides immediate and reliable removal.
  - Can lower potassium by 1mmol/L in first hour and another 1mmol/L over the next 2 hours.
Authors conclusion: in view of the limited information from small studies of uncertain quality no firm recommendations for clinical practice can be made. Until further evidence is ascertained from larger well designed and executed randomised controlled trials, it appears that the combination of insulin and glucose is preferred over treatment with rectal cation-resin intervention for hyperkalaemia in preterm infants.
Lab confirmed serum Potassium
Potassium > 6.5 mmol/L

- ECG for arrhythmia
- Correct acidosis with sodium bicarbonate
- 10% Calcium chloride 0.5 mmol/kg if ECG changes
- Stop all potassium containing infusions

Check level in 4 hrs still rising

Preterm:
- Insulin (0.05-2u/kg/hr) and 20% dextrose 2.5-5ml/kg/hr in non-oliguric
- Salbutamol bolus in renal failure 4-5mcg/kg
- Check level in 4 hrs
- If rising start Salbutamol infusion 0.1mcg/kg/min

Term:
- Calcium resonium 0.3 – 1ml/kg PR
- Salbutamol bolus in renal failure 4-5mcg/kg
- Check levels in 4 hours
- If rising start Salbutamol infusion 0.1mcg/kg/min
- Insulin (0.05-2u/kg/hr) and 20% dextrose 2.5-
Hypokalaemia

• Hypokalaemia definition: K+ level < 3.5 mmol/L

• Moderate hypokalaemia: serum K+ level of < 3.0 mmol/L
  • Symptoms may include feeling tired, leg cramps, palpitations, weakness, delirium, depression, and constipation

• Severe hypokalaemia: serum K+ level < 2.5 mmol/L
  • Risk of an abnormal heart rhythm: tachycardia/bradycardia, cardiac arrest
Hypokalaemia

Leads to hyperpolarization in the myocytes, resting membrane potential lowers to less than -100mV

Normal action potential becomes harder to trigger

hERG/I_{Kr} potassium current diminishes, resulting in ventricular repolarisation prolongation which in turn promotes ventricular arrhythmias by potentiating afterdepolarisations
Causes of hypokalaemia

- Diarrhoea
- Medication e.g. furosemide and steroids
- Dialysis
- Diabetes insipidus
- Hyperaldosteronism
- Hypomagnesemia
- Poor dietary intake
- Familial: Bartter, Gitelman
ECG changes

• ECG changes appear when K+ drop below 2.7 mmmol/L:

  • Increased amplitude and width of the P wave
  • Prolongation of the PR interval
  • T wave flattening and inversion
  • ST depression
  • Prominent U waves (best seen in the precordial leads)
  • *Apparent* long QT interval due to fusion of the T and U waves (= long QU interval)
Arrhythmias in hypokalaemia

• With worsening hypokalaemia:
  • Frequent supraventricular and ventricular ectopics
  • Supraventricular tachyarrhythmias: AF, atrial flutter, atrial tachycardia
  • Potential to develop life-threatening ventricular arrhythmias, e.g. VT, VF and Torsades de Pointes
Hypokalaemia

ST depression.
T wave inversion.
Prominent U waves.
“U” waves in Hypokalemia
Hypokalaemia
Treatment of Hypokalaemia

- Reduction of losses
- Normalising potassium and magnesium (normalising K+ levels is difficult if Mg++ is also low)
- Evaluation for potential toxicities and determination of the cause

Correction

- Patients with a potassium level of 2.5-3.5 mEq/L may need only oral potassium replacement
- Role of potassium-sparing diuretics
- If the potassium level is less than 2.5 mEq/L, intravenous (IV) potassium via central line
- Check Mg++ in any patient with arrhythmia, top up K+ to 4.0-4.5 mmol/l and Mg++ to > 1.0 mmol/l (standard practice in most CCUs and ICUs)
Scottish Paediatric Cardiac Service

Special considerations
**Digoxin and potassium**

**Mechanism of action of digoxin**

- Increases the force of cardiac contraction, causing the cardiac output.
- Improved circulation leads to reduced sympathetic activity, which then reduces peripheral resistance with reduction in heart rate.
- Slows down conduction velocity through the AV node, which accounts for its use in atrial fibrillation.
Digoxin use in paediatrics

- In paediatrics, digoxin is currently used as an adjunct in myocardial disease accompanied by inappropriate sinus tachycardia (DCM, myocarditis) and to treat non-WPW SVT.

- Digoxin levels and serum electrolyte monitoring is required during digitalisation, consult BNF and pharmacist.

- In acute phase of myocarditis, low starting dose (1/2 to ¾ maintainance) is required.

- Check interaction with other antiarrhythmic drugs if polypharmacy – Digoxin dose may need to be adjusted to avoid toxicity.

Inverse “Tick” sign
**Digoxin toxicity**

- In Digoxin poisoning, arrhythmias are due to
  - Increased automaticity (increased intracellular Ca++) – slow PVCs, AV block, in adults slow regularised AF, severe cases - VT
  - Decreased AV conduction (vagal effects on AV node) – AV block

- “Slow and sick” – Digoxin toxicity

- Low K+, low Mg++, high Na++, high Ca++ serum levels and acid-base disturbance exacerbate Digitalis toxicity – correct!

- For severe toxicity of overdose, Digoxin-specific antibody fragments can be given IV – contact Pharmacy
# LQT and potassium

<table>
<thead>
<tr>
<th>LQTS subtype</th>
<th>Culprit gene</th>
<th>Protein</th>
<th>Functional effect of mutation</th>
<th>Frequency of cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LQT1</td>
<td>KCNQ1</td>
<td>Alpha-subunit of ( I_{Ks} )</td>
<td>Loss-of-function, reduced ( I_{Ks} )</td>
<td>30–35</td>
</tr>
<tr>
<td>LQT2</td>
<td>KCNH2</td>
<td>Alpha-subunit of ( I_{Kr} )</td>
<td>Loss-of-function, reduced ( I_{Kr} )</td>
<td>25–30</td>
</tr>
<tr>
<td>LQT3</td>
<td>SCN5A</td>
<td>Alpha-subunit of ( I_{Na} )</td>
<td>Gain-of-function, increased late ( I_{Na} ) inward current</td>
<td>5–10</td>
</tr>
<tr>
<td>LQT4</td>
<td>ANK2</td>
<td>Ankyrin-B; links membrane proteins with underlying cytoskeleton</td>
<td>Loss-of-function, disrupts multiple ion channels</td>
<td>&lt;1</td>
</tr>
<tr>
<td>LQT5</td>
<td>KCNE1</td>
<td>Beta-subunit of ( I_{Ks} )</td>
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</tr>
<tr>
<td>LQT6</td>
<td>KCNE2</td>
<td>Beta-subunit of ( I_{Kr} )</td>
<td>Loss-of-function, reduced ( I_{Kr} )</td>
<td>&lt;1</td>
</tr>
<tr>
<td>LQT7</td>
<td>KCNJ1</td>
<td>Alpha-subunit of ( I_{Nax} )</td>
<td>Loss-of-function, reduced ( I_{Nax} )</td>
<td>&lt;1</td>
</tr>
<tr>
<td>LQT8</td>
<td>CACNA1C</td>
<td>Alpha-subunit of ( I_{CaL} )</td>
<td>Gain-of-function, increased ( I_{CaL} )</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT9</td>
<td>CAV3</td>
<td>Caveolin-3; a scaffolding protein in caveolae</td>
<td>Increased late ( I_{Na} ) inward current</td>
<td>&lt;1</td>
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<tr>
<td>LQT10</td>
<td>SCN4B2</td>
<td>Beta 4-subunit of ( I_{Na} )</td>
<td>Gain-of-function, increased late ( I_{Na} ) inward current</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT11</td>
<td>AKAP9</td>
<td>A kinase-anchor protein-9; sympathetic ( I_{Ks} ) activation</td>
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<tr>
<td>LQT12</td>
<td>SNTA1</td>
<td>Alpha-syntrophin; regulation of ( I_{Na} )</td>
<td>Increased late ( I_{Na} ) inward current</td>
<td>Rare</td>
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<tr>
<td>LQT13</td>
<td>KCNJ5</td>
<td>Kir 3.4</td>
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<td>Rare</td>
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<tr>
<td>LQT14</td>
<td>CALM1</td>
<td>Calmodulin-1</td>
<td>Altered calcium signaling</td>
<td>&lt;1</td>
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<tr>
<td>LQT15</td>
<td>CALM2</td>
<td>Calmodulin-2</td>
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Spears DA, Gollob MH
Genetics of inherited primary arrhythmia disorders
The Application of Clinical Genetics, 2015
12 yo male, LQT2 (KCNH2 mutation) syncope - fall – head injury - vomiting

K 3.9
12 yo male, LQT2 (KCNH2 mutation) syncope - fall – head injury - vomiting

K 4.0
Mg 0.97
12 yo male, LQT2 (KCNH2 mutation)
syncope - fall – head injury - vomiting

K 4.6 Betablocker
# LQT and Potassium

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Spears DA, Gollob MH

Genetics of inherited primary arrhythmia disorders
The Application of Clinical Genetics, 2015
Periodic paralysis and potassium: Andersen-Tawil Syndrome

- Rare autosomal dominant disorder: periodic paralysis, LQT, ventricular arrhythmias, dysmorphic features
- Variable clinical manifestations, genetically heterogeneous disease: full triad of features (VA, PP, dysmorphic features) in 58-78%, 32-81% 2 of 3 organ involvement, 6-20% - non-penetrance
- Over 20 different mutations in KCNJ2 (encodes alpha-subunit of the inward rectifier K+ channel Kir2.1)
- Hypo-, normo- and hyperkalaemia can trigger arrhythmias and attacks of periodic paralysis in different patients

Micrognatia, hypertelorism, low set ears, clinodactily of 5th fingers
ATS: Mechanism of ventricular arrhythmias


- Demonstrated potential mechanisms underlying arrhythmias in patients with ATS due to reduction of inward rectifier K+ channel Kir2.1 conductance
  - Development of early after depolarisations (EADs) associated with action potential duration prolongation
  - Induction of spontaneous action potentials (AP) (abnormal automaticity) due to depolarization of resting membrane potential
- **Lo K+** facilitated and **Hi K+** suppressed EADs and spontaneous APs
- Adrenergic stimulation enhanced delayed afterdepolarisations (DADs) and could facilitate EADs and spontaneous APs especially in the setting of hypokalaemia
ATS: typical ECG changes

- Mild QTc interval prolongation
- Marked prolongation of the QUc interval
- Prominent U waves
15 year old, familial congenital myotonia, rare polymorphic PVCs, KCNJ2 mutation carrier

- Prolongation of T-wave downslope
- Wide T-U junction
- High amplitude, broad U waves

Zhang et al

- Arrhythmias: polymorphic PVCs, polymorphic VT typically non-sustained and bidirectional. Degeneration into lethal ventricular arrhythmias is uncommon

Mild QTc interval prolongation
Marked prolongation of the QUc interval
Prominent U waves
15 year old, familial congenital myotonia, rare polymorphic PVCs, KCNJ2 mutation carrier
Thank you