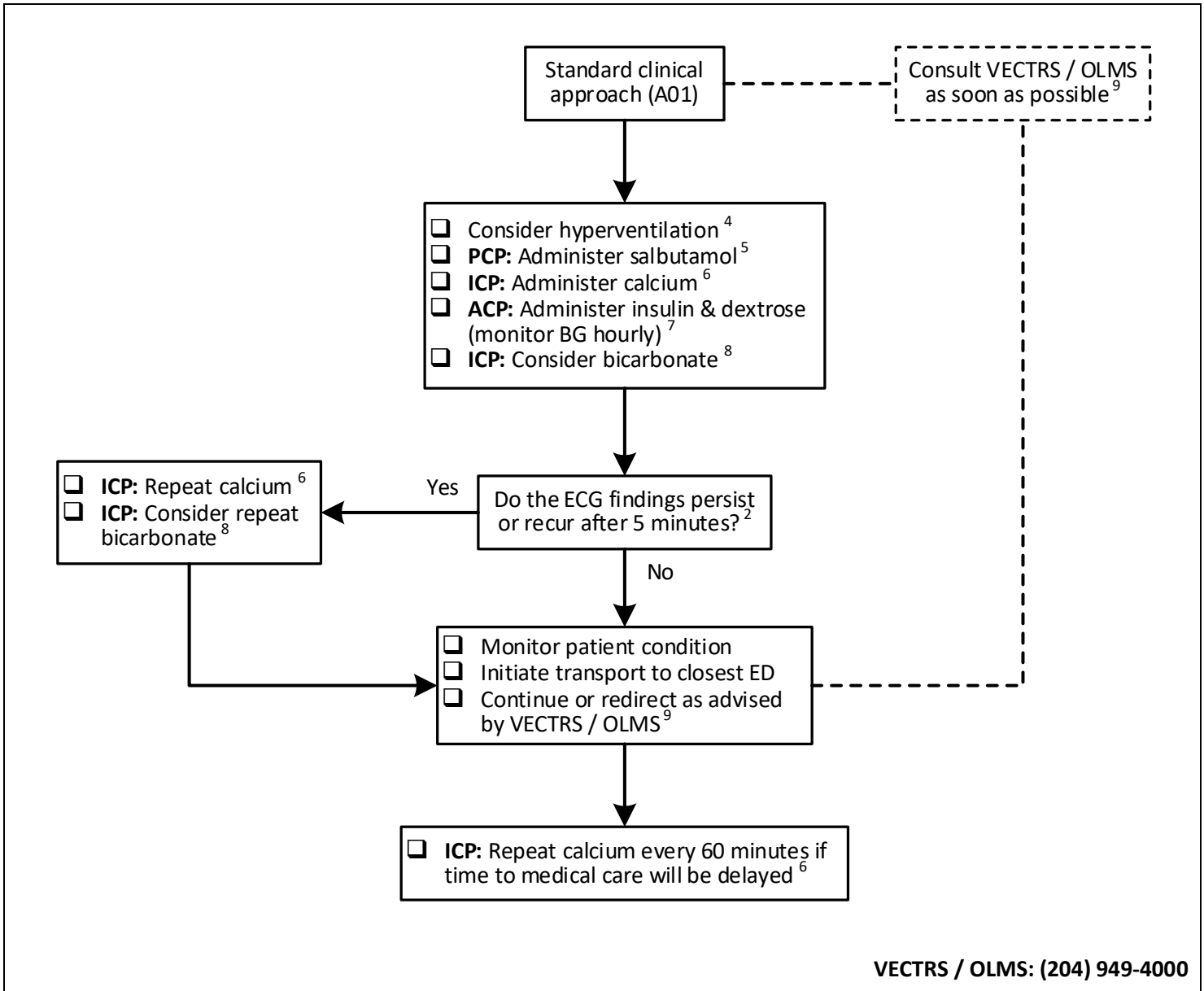
	E11 - HYPERKALEMIA (ALL AGES)		
	Version date: 2025-03-21	Effective date: 2025-04-30 (07:00)	
PCP= PCP - ACP	ICP = ICP & ACP	ACP = ACP only	None = EMR - ACP



INDICATIONS

- Known or suspected hyperkalemia in a patient who is dialysis-dependent or has known advanced stage chronic kidney disease with any one or more of the following:
 - Electrocardiographic signs of hyperkalemia (appendix A)
 - Symptoms consistent with hyperkalemia ³
 - Has missed at least one scheduled dialysis treatment in the absence of ECG findings or symptoms
- Overdose with potassium-containing medications

WARNINGS

- None

NOTES

1. End-stage kidney disease is defined by a glomerular filtration rate (GFR) that is insufficient to excrete the body's daily potassium load. Nephrologist classify this as stage G5 **chronic kidney disease** (CKD). Patients are usually being prepared for dialysis at this point and may be able to provide this in their history.
 However, patients with stage 4 (and occasionally stage 3) CKD may sustain an **acute kidney injury** (AKI) that abruptly reduces their GFR causing acute hyperkalemia.
2. Certain characteristic electrocardiographic (ECG) features evolve as the serum potassium level rises (appendix A). However, the absence of ECG changes does not exclude hyperkalemia.
 Rhythm abnormalities usually occur when the serum potassium reaches a level of approximately 7.0 mEq/l but can appear at lower levels if the rise is sudden. Patients can rapidly progress from an apparently normal ECG to cardiac arrest.
3. Symptoms of hyperkalemia involve cardiac or skeletal muscle. These include muscle weakness or paralysis, presyncope or syncope, and palpitations.
 However, symptoms of other conditions, such as nausea or abdominal pain, may indicate an acute condition causing an abrupt deterioration in renal function.
 Paramedics should have a low index of suspicion for the presence of hyperkalemia in patients who are dialysis-dependent or have advanced stage CKD. ECG monitoring should be employed in almost all of these patients, irrespective of the chief complaint.
4. Hypoventilation from any cause (e.g. fatigue, over sedation) will result in the movement of potassium out of cells raising the serum level. Hyperventilation can shift potassium back into cells temporarily lowering the extracellular level.
5. Beta-2 adrenergic agonists like salbutamol can reduce serum potassium levels by as much as 1 mEq/l and can be an effective temporizing measure until other therapies can be established.
6. Calcium is a direct antagonist to the effects of potassium on cell membranes, but it does not decrease the extracellular level so it should never be used without other potassium lowering strategies. Its effects are short lived. Repeat dosing may be required if the time to medical care will be delayed.

7. Insulin is very effective at shifting potassium into cells, thereby lowering the extracellular level by as much as 2 mEq/l. The half-life is prolonged in renal failure, with an antihyperkalemic action of 4 hours or more.

The blood glucose (BG) should be monitored hourly for six hours after insulin administration. Insulin can be administered alone in patients with concomitant hyperglycemia (BG greater than 15 mmol/l). Dextrose should never be administered alone as its effects on insulin release are variable.

8. Sodium bicarbonate has limited effect on serum potassium, but may be effective in the presence of metabolic acidosis. It may be indicated for the treatment of acidosis independent of the potassium level, but should never be used as monotherapy for hyperkalemia.

Bicarbonate by continuous infusion may be safer and more effective than bolus therapy but as its peak effect does not occur for at least 4 hours. It is rarely indicated unless the time to medical care is excessively prolonged.

Note that calcium and bicarbonate are incompatible. Flush the line well between agents if administering both.

9. For a patient who has missed dialysis, online medical support (OLMS) at the Virtual Emergency Care & Transport Resource Service (VECTRS) may advise transport directly to a facility where the patient can receive emergency dialysis.

LINKS

- A01 - Standard Clinical Approach
- M30 - Hyperkalemia Therapy

APPROVED BY



Medical Director - EMS

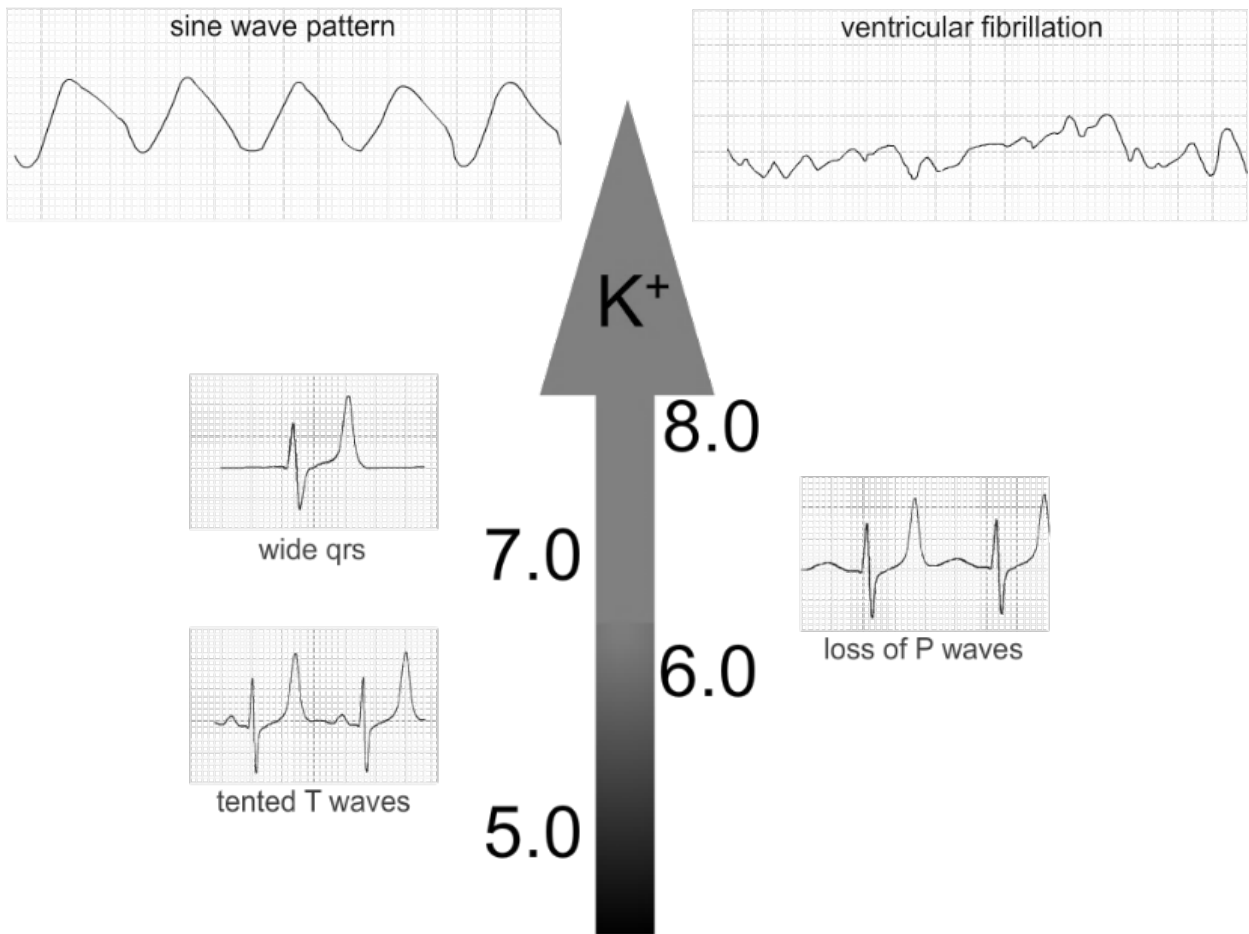


Associate Medical Director - EMS

VERSION CHANGES (refer to X05 for change tracking)

- Addition of ACP work scope
- Revised notes & flow chart
- Addition of insulin / dextrose at ACP level

APPENDIX A: ELECTROCARDIOGRAPHIC FEATURES OF HYPERKALEMIA ²



Serum potassium (mEq/l)	Usual ECG Features	Common Rhythm Abnormalities
5.5 - 6.5	<ul style="list-style-type: none"> • Peaked T waves (appendix B) 	<input type="checkbox"/> Bundle branch block <input type="checkbox"/> Sinus bradycardia / arrest <input type="checkbox"/> Idioventricular rhythms <input type="checkbox"/> Sine wave pattern <input type="checkbox"/> Ventricular tachycardia <input type="checkbox"/> Ventricular fibrillation <input type="checkbox"/> Asystole
6.5 - 7.5	<ul style="list-style-type: none"> • Loss of P waves 	
7.0 - 8.0	<ul style="list-style-type: none"> • Widening of QRS complex 	
> 8.0	<ul style="list-style-type: none"> • Sine wave 	

APPENDIX B: PEAKED T WAVES

