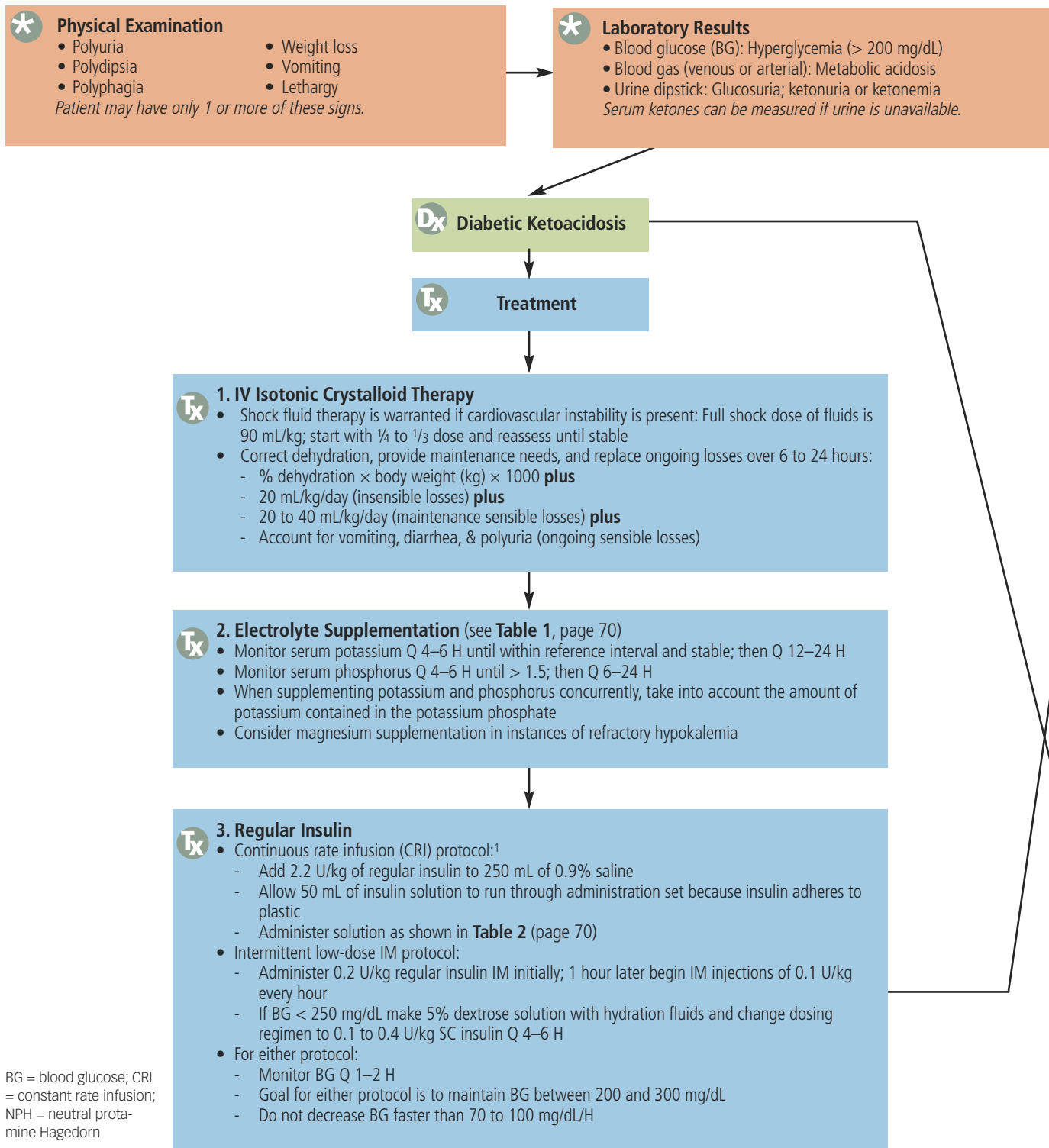


# Canine Diabetic Ketoacidosis

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**Monitoring**

- Physical examination: Respiratory rate/effort, heart rate, pulse quality
- Hydration: Central venous pressure, weight, skin turgor, mucous membrane quality
- Electrolytes: Potassium, phosphorus, +/- magnesium
- Ketones: Serum, urine
- Blood glucose
- Blood gas and acid/base status
- Appetite/emesis
- As needed: Packed cell volume/total solids, serum biochemical profile, blood pressure

- Hydrated?
- Acidemia and electrolyte abnormalities resolved?
- Underlying condition resolving?

*Once the patient is hydrated, the route of regular insulin administration can be switched from IM to SC, if desired. There is no need to switch to SC administration if CRI protocol is chosen initially.*

Yes

No

**Investigation** Ketones present?

Yes

**Treatment**

- Continue IV fluids and electrolyte supplementation
- Continue regular insulin administration, either CRI or IM (can administer via SC route if patient is hydrated)

No (or trace)

**Investigation** Eating consistently?

No

**Treatment** Treat Any Concurrent Conditions

- Pancreatitis
- Urinary tract infection
- Renal failure
- Cholangiohepatitis
- Pyometra
- Skin disease
- Heart disease
- Neoplasia

**Treatment** Switch to SC Long-Acting Insulin

- Neutral protamine Hagedorn (NPH) insulin (0.25–0.5 U/kg SC Q 12 H initially); consider starting at higher dose if patient previously diagnosed with diabetes mellitus and known to require higher doses of insulin
- Lente (not currently commercially available)

No

Yes

**Investigation** Hyperadrenocorticism (see **Hyperadrenocorticism: Why Wait to Test**, page 70)

**Treatment** Continue Management for Uncomplicated Diabetic

**Investigation** Underlying cause of insulin resistance identified?

**Further Investigation**

It is essential to identify the underlying cause of the increase in diabetogenic hormones (catecholamines, glucagon, glucocorticoids, growth hormone, and estrogen) that lead to the ketogenic crisis:

- Physical examination
- Complete blood count
- Serum biochemical profile
- Urine culture
- Canine pancreatic lipase immunoreactivity (cPLI)
- Abdominal radiographs
- Abdominal ultrasound
- Thoracic radiographs

**Dx** Diagnosis

**Investigation**

**Tx** Treatment

**\*** Result

This algorithm can be downloaded and printed for use in your clinic at [cliniciansbrief.com](http://cliniciansbrief.com).

**▶ Table 1. Electrolyte Supplementation**

Serum Potassium Concentration (mEq/L)	Potassium Chloride Dose
> 3.5 (maintenance)	0.05–0.1 mEq/kg/H
3–3.5	0.1–0.2 mEq/kg/H
2.5–3	0.2–0.3 mEq/kg/H
2–2.5	0.3–0.4 mEq/kg/H
< 2	0.4–0.5 mEq/kg/H
Serum Phosphorus Concentration (mg/dL)	Potassium Phosphorus Dose
2–2.5	0.03 mmol/kg/H
1.5–2	0.06 mmol/kg/H
1–1.5	0.09 mmol/kg/H
< 1	0.12 mmol/kg/H

**▶ Table 2. CRI Infusion of Insulin Solution**

BG Concentration (mg/dL)	IV Hydration Fluids	Rate of Insulin Solution (mL/H)
> 250	0.9% saline	10
200–250	0.9% saline + 2.5% dextrose	7
150–200	0.9% saline + 2.5% dextrose	5
100–150	0.9% saline + 5% dextrose	5
< 100	0.9% saline + 5% dextrose	Discontinue

### Hyperadrenocorticism: Why Wait to Test?

Although hyperadrenocorticism is one of the most frequent causes of insulin resistance, it is not appropriate to test for it during a diabetic ketoacidosis crisis because false positives would be expected. Diagnostic testing for hyperadrenocorticism should not be performed until the patient has been systemically healthy for at least 2 weeks. Appropriate regulation of diabetes mellitus may be difficult to achieve prior to diagnosis of concurrent hyperadrenocorticism.

See Aids & Resources, back page, for references & suggested reading.

BG = blood glucose