Emergency management and treatment of the critically ill diabetic: Diabetes Ketoacidosis (DKA) and Hyperglycemic, Hyperosmolar Syndrome (HHS)!

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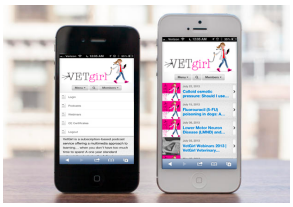

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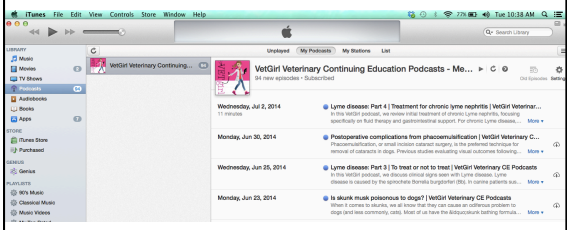

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Goals

- Definitions of DKA & HHS
- Pathophysiology
- Diagnosis
- Clinical signs
- Treatment
  - Fluid therapy
  - Electrolyte/acid-base
  - Osmolality
  - Insulin therapy

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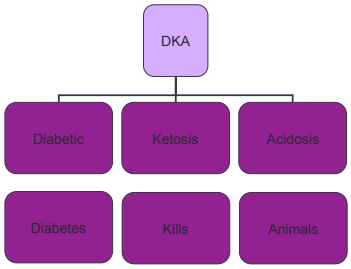
Definition of DKA



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Definitions

- DKA: Diabetic ketoacidosis
- HHS: Hyperglycemic hyperosmolar syndrome
  - Used to be called HONK (hyperosmolar non-ketotic)

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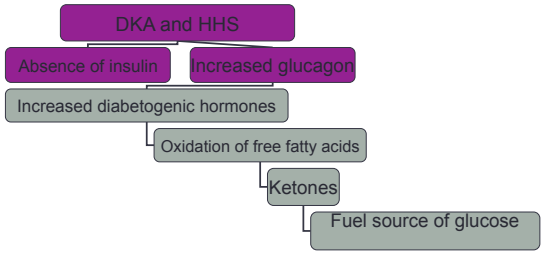

- MOA: glucosuria/ketonuria → osmotic diuresis → dehydration → hypovolemia → electrolyte abnormalities

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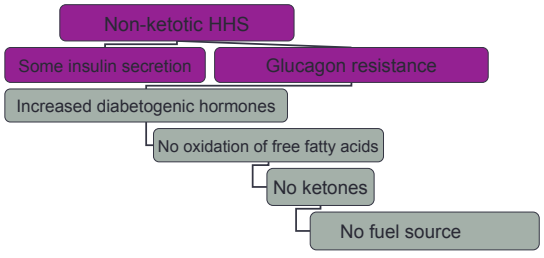

- MOA: Severe hyperglycemia and glucosuria → osmotic diuresis → dehydration → hypovolemia → hyperosmolarity → electrolyte abnormalities

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Diabetic Ketoacidosis: (DKA)

- Life-threatening:
  - Acidosis
  - Dehydration
- Concurrent disease
- Treatment:
  - Lots of fluids
  - Electrolytes
  - Eventually….insulin

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Hyperglycemic Hyperosmolar Syndrome: (HHS)

- Life-threatening:
  - Hyperosmolarity
  - Dehydration
- Not always ketotic (or low levels)
- More common in cats than dogs
- Severe hyperglycemia
  - >600 mg/dl (> 33 mmol/L)
- Hyperosmolarity
  - >350 mosm/kg
- Severe clinical dehydration
- Lack of ketones
- Lactic acidosis
- CNS depression

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The question remains…

- Can well-controlled diabetic patients become ketoacidotic?
- Lack of insulin
- Fasting / Dehydration
- Stress hormones
  - Glucagon
  - Cortisol
  - Epinephrine
  - Growth hormone
- Stress hormones → worsen hyperglycemia

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Causes for DM → DKA
Concurrent diseases:

- Cushings
- Hepatic lipidosis
- Cholangiohepatitis
- Pancreatitis
- CRF
- Prostatitis
- Skin disease
- UTI
- Neoplasia
- Pneumonia
- CHF

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DM → DKA
CANINE Concurrent diseases:

71% of dogs!!!

- Cushings
- Pancreatitis
- Neoplasia
- UTI
- Hypothyroid
- Pneumonia

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DM → DKA
FELINE Concurrent diseases:

93% of cats!!!

- Hepatic lipidosis
- Cholangiohepatitis
- CRF
- UTI / Infection
- Neoplasia
- Pancreatitis

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Causes: Diabetes Mellitus (DM)

Dog	Cat
• Genetics	• Islet amyloidosis
• Immune insulitis	• Obesity
• Pancreatitis	• Depo injections
• Obesity	• Infection
• Infection	• Concurrent Illness
• Concurrent illness	• Drugs (ovaban)
• Drugs (glucocorticoids)	• Pancreatitis
• Islet amyloidosis	• Genetics
	• Immune insulitis

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Clinical presentation

- Middle-aged to older
- Obesity (weight loss?)
- Lethargy
- Dehydration
- Muscle wasted
- Weakness
- PU/PD/PP (historical)
- Weight loss
- Polyphagia → Anorexia
- Vomiting
- Ketotic/acetone breath

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Bonder: 10 yo MC Siamese

- PC: lethargy, anorexia, pu/pd X 3 days
- PE:
  - 10% dehydrated
  - 5 kg
  - 3rd eyelids elevated
  - HR 180, RR 30, T 98.2°F (36.8° C)
  - Slightly icteric
  - Doughy abdomen
  - Sweet "acetone" breath

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Diagnostic workup of DKAs

Marshfield

Big 4 + serum ketones

- Big 4 (while drawing CBC/CHEM)
  - PCV/TS/BG/Azo
- Urine or serum ketone dipstick

Initial diagnostics

- Big 4 (draw CBC/CHEM at same time)
  - PCV/TS *(55%/9.8 g/dL (98 g/L), icteric serum)*
  - Blood glucose (BG) *(680 mg/dL; 37.7 mmol/L)*
  - Azostick (BUN) *(50-80 mg/dL; 35-57 mmol/L))*
  - Venous blood gas (VBG)
- Within 2-3 minutes, identifies:
  - Hemoconcentration
  - Diabetes
    - Serum ketones!
  - Renal failure vs. pre-renal
  - Metabolic acidosis + elyte abnormalities

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"Ideal" diagnostic workup of DKAs

- CBC
- Chemistry
- Urinalysis
- Urine culture + MIC
- Radiographs
  - Chest
  - Abdomen
- Abd. ultrasound
- Lipase/Amylase (dog)
- Coagulation panel
- Venous blood gas
  - Electrolytes!
- Doppler

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Typical DKA clinicopathologic findings

- Hemoconcentration
  - Dehydration
- Anemia
  - Anemia of chronicity
  - GI blood loss
- Leukocytosis
  - Left shift
- Increased LES
  - Hypoxia
  - Pancreatitis
  - Underlying disease
- Azotemia
  - Pre-renal
  - Renal

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Typical DKA clinicopathologic findings

- Hyperglycemia
- Urinalysis/Culture
  - Glucosuria
  - Ketonuria
  - Bacteruria
  - Specific gravity
- Metabolic acidosis
  - Ketone bodies
  - Lactic acid
  - Uremic acids
  - *Electrolytes*!

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Bonder's blood results:

- CBC:
  - WBC: 17,400 X 10^3/uL
  - Neuts: 16,000 X 10^3/uL
  - Plts: Normal, clumped
  - Slightly icteric plasma
- Diagnosis:
  - Inflammatory leukocytosis
  - Underlying hepatic disease!
    - Hepatic lipidosis
    - Cholangiohepatitis
    - Pancreatitis

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Bonder's results

- CHEM: mg/dL / mmol/L
  - BUN: **61 /43**
  - Creat: **2.7 / 239**
  - Tbili: **1.8 / 30 µmol/L**
  - ALT: 210
  - AST: 175
  - Glucose: **656/ 36.4**
  - Bicarb: 7.0 mEq/L
  - Na+: **135** mEq/L
  - Cl-: 95 mEq/L
  - K^+: **2.7** mEq/L
  - P^+: 2.6 /0.84
- Diagnosis:
  - Azotemia
  - Icteric
  - Increased LES
  - Diabetes mellitus
  - Metabolic acidosis
  - Severe electrolyte abnormalities
  - Hyperosmolar
- Treatment:
  - IVF

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Bonder's results

- Urinalysis:
  - 1.025
  - pH 6.0
  - 3+ glucosuria
  - 2+ ketones (moderate)
  - Ruleouts:
    - Isosthenuria
      - Renal disease
      - Osmotic diuresis
    - Ketonuria
    - Glucosuria
- Cystocentesis

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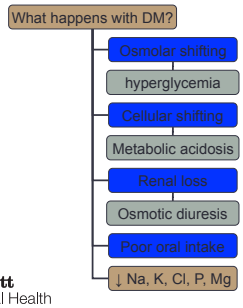
Electrolyte disturbances



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Potassium

- Clinical signs of hypokalemia:
  - Muscle weakness
  - Cervical ventroflexion (cats)
  - Cardiac arrhythmias
  - Respiratory muscle failure
- Supplement, supplement, supplement!

  And then consider.. over-supplementing!

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Potassium supplementation

Serum K+ (mEq/L)	K+/Liter fluids
> 3.5	20
3.0-3.5	30
2.5-3.0	40
2.0-2.5	60
< 2.0	80

****NOT recommended to exceed 0.5 mEq/kg/hr**

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Potassium supplementation

- 5 kg cat on 50 ml/hr
- 1 liter bag of LRS = 20 hours of IV fluids
- K^+ supplementation formula:
  - ≤ 0.5 mEq KCL/kg/hr
  - 0.5 mEq KCL X 5 kgs = 2.5 mEq KCl/hour
  - 20 hours X 2.5 mEq KCl/hour = 50 mEq KCL into the bag!

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Why over-supplement?

- Insulin drives potassium into cells

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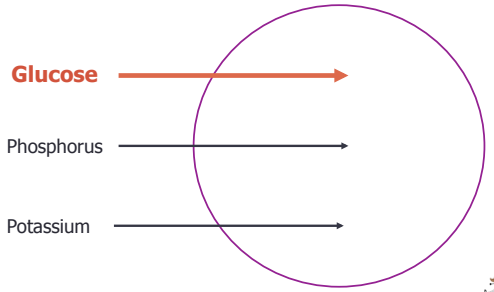
Insulin Therapy



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Phosphorous

- Clinical signs of hypophosphatemia:
  - Weakness, ataxia, seizures
  - Acute hemolytic anemia < 1.5 mg/dL (0.48 mmol/L)
- Same shifts as K^+ (acidosis, insulin, urinary losses)
- Supplement as Potassium Phosphate
  - 0.01-0.12 mmol/kg/hr CRI
- KCL + KPhos at 50:50

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Fix electrolytes first!

- Fix electrolytes first
- Worry about insulin administration *later!*

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Osmolality: Sodium control

- For each ↑ 62 mg/dL in $glucose_{mg/dL}$, ↓ 1 $Na^+_{mEq/L}$
- For each ↑ 3.4 mmol/L in $glucose_{mg/dL}$, ↓ 1 $Na^+_{mEq/L}$
- Fluid shifts into intravascular space due to hyperglycemia
- Dilutes sodium and potassium
- Is your patient hyponatremic or hyperglycemic?
  - Bonder: Na^+ 135 mEq/L BG: 656 mg/dL (36.4 mmol/L)

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Osmolality: Sodium control

- Is your patient hyponatremic or hyperglycemic?
  - Bonder: Na^+ 135 mEq/L BG: 656 mg/dL (36.4 mmol/L)
- 656 mg/dL/62 = 10.6 mg/dL
- Bonder's Na^+ 135 + 10.6 = Corrected sodium of really 145 mEq/L

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Osmolality: Sodium control

- 2 (Na^+ + K^+) + BUN/2.8 + Glucose/18
- 2 X Na^+

Normal: 280-310 mOsm/kg

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Osmolality: Sodium control

Compare the two formulas:

- 2(Na + K) + BUN/2.8 + Glucose/18
- 2*(Na)

VS.

- Bonder:
  - Na^+: 135 mEq/L K^+: 2.7 mEq/L
  - BUN: 61 mg/dL (43 mmol/L) BG: 656 mg/dL
  - Full formula: 275 + 22 + 36 = 333 mOsm/kg
  - 2 (Na) = 270 mOsm/kg → *not very accurate!*

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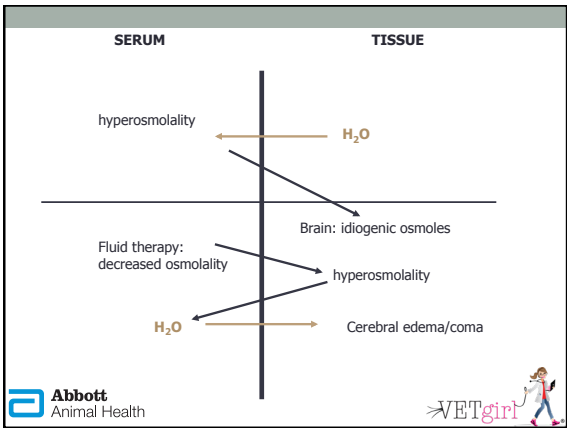

What other tests do you want to do in DKA patients?

- Chest radiographs
  - Aspiration pneumonia
  - Volume overload
  - Metastasis

What other tests do you want to do in DKA patients?

- Abdominal radiographs
  - Pancreatitis
  - Neoplasia
- AUS
  - Neoplasia
  - Pancreatitis
  - Underlying disease
    - abscess

What other tests do you want to do in DKA patients?

- Lipase/amylase/c/fTLI/PLI
  - Pancreatitis
  - R/O renal insufficiency
- Coagulation
  - DIC
  - Thrombocytopenia
  - Prolonged clotting

Bonder's blood results

- Coag: WNL
- FELV/FIV negative
- CXR: WNL
- AXR:
  - small kidneys
  - mild decrease in cranial abdominal detail
  - Ruleouts:
    - Pancreatitis
    - Renal disease
- AUS:
  - Mild pancreatitis

TREATMENT FOR DKA

So now that we diagnosed DKA, what do we do?

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Stop trying to fix that blood glucose first!

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#1: Fluid therapy

perfusion, perfusion, perfusion!

- **Volume resuscitation/perfusion**
  - Treat for hypovolemia
  - Replace the dehydration
  - Maintenance + ongoing losses
- **Correction of electrolyte disturbances**
- **Dilution of blood glucose**

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Fluid choice

perfusion, perfusion, perfusion!

- **LRS**
  - Needs hepatic conversion for bicarbonate generation
  - Buffered
- **0.9% NaCl**
  - Acidifying
  - Na^+ too high?
- **Plasmalyte-148**
  - Acetate/gluconate → bicarbonate buffer
  - Contains magnesium

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Fluid choice

Solution	Ringers	LRS	Plasmalyte 56	Plasmalyte R	Plasmalyte A; Norm R	0.9% NaCl
Na^+	147	130	40	140	140	154
K^+	4	4	13	10	5	0
Ca^{++}	4	3	0	5	0	0
Mg^+	0	0	3	3	3	0
Cl^-	156	109	40	103	98	154
Gluconate	0	0	0	0	23	0
Lactate	0	28	0	8	0	0
Acetate	0	0	16	47	27	0
Osmolarity	310	270	111	312	294	310

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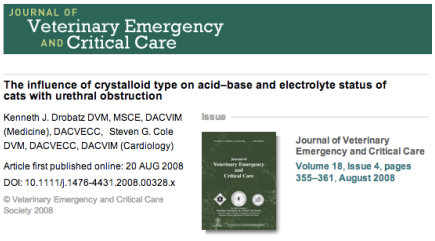
Isotonic Crystalloids



JOURNAL OF Veterinary Emergency AND Critical Care

The influence of crystalloid type on acid–base and electrolyte status of cats with urethral obstruction

Kenneth J. Drobatz DVM, MSCE, DACVIM (Medicine), DACVECC, Steven G. Cole DVM, DACVECC, DACVIM (Cardiology)

Article first published online: 20 AUG 2008

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Issue

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Isotonic Crystalloids

JOURNAL OF Veterinary Emergency AND Critical Care

Original Study

Effects of rapid intravenous 100% L-isomer lactated Ringer's administration on plasma lactate concentrations in healthy dogs

Søren R. Boysen DVM, DACVECC[1,2,*] and Patricia Dorval DVM, DACVECC[1]

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IV fluid effect on blood glucose

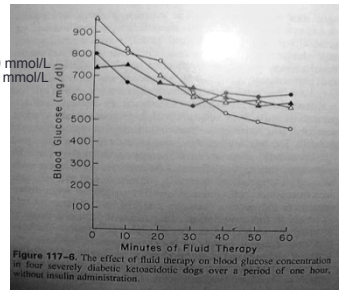

Figure 117-6. The effect of fluid therapy on blood glucose concentration in four severely diabetic ketoacidotic dogs over a period of one hour, without insulin administration.

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Fluid therapy: Calculations

Shock: ___ ml bolus to effect

+ Dehydration: ___ ml/hr
+ Maintenance: ___ ml/hr
+ Ongoing Losses: ___ ml/hr

=Initial Fluid Rate ___ ml/hr

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Assessment: Dehydration

CLINICAL SIGNS	ESTIMATE OF DEHYDRATION
NORMAL	<5%
DRY MUCOUS MEMBRANES	5%
REDUCED SKIN TURGOR	6-8%
INCREASED HEART RATE	8-10%
WEAK PULSES	10-12%
COLLAPSE, SHOCK	12-15%

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Bonder: How much fluid do we want to give him?

- Doppler on initial presentation: 70 mmHg
- Gave ¼ of a shock dose of crystalloid to improve blood flow
  - ¼ of 60 ml/kg = ¼ of 60 (5kg) = 75 mls
- 60-75 mls Norm-R over 30 minutes – 1 hour
- Reassess Doppler = 110 mmHg

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Bonder needs more!

- Maintenance + ongoing losses (pu/pd)
  - 5 kg X 60 ml/kg/24 hours = 300 mls
  - **13** ml/hour
- Replace dehydration over 24 hours
  - 5 kg X 10% dehydration X 1000 ml = 500 mls
  - **20** mls/hour
- On-going losses
  - **PU/PD**
  - Severe pu!

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Fluid resuscitation

- 13 mls/hour + 22 mls/hour = approximately:

40 ml/hr crystalloid X 12 hours + KCl + KPhos

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Fluid resuscitation

- Reassess! Recheck PCV/TS, VBG, elytes, BG
- Reassess based on:
  - Physical examination
  - PCV/TS (55%/98 g/L →40/70 → 35/60)
  - Azo/BUN (61 mg/dL/43 mmol/L → improvement)
  - Body weight (5 kgs at 10% dry)
    - 5 kgs X 0.1 X 1000 = 500 mls
    - 1 L = 1000 mls
    - After dehydration, should weigh 500 mls more = 0.5 kgs
    - Ideal weight: 5.5 kgs

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Fluid therapy

- By 48 hours: after hydration improved, ↓ to 30 ml/hr + KCl + KPhos

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Monitor that osmolality!

- Change it slowly!

- Correct sodium, water, and glucose derangements over 8-12 hours (24 hours in cats!)
- Pick Na^+ fluid closest to animal's Na^+!
- Wait to begin insulin therapy

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Assess hydration carefully!

- Diabetes:
  - Sp. gravity 1.015-1.018
  - Urine volume
- Drinking water in the cage

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INSULIN THERAPY

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#2: Regular insulin therapy

- PROS:
  - Fixes the blood glucose "#" right away
  - Provides cellular substrate by driving glucose intracellularly
  - Helps resolve the metabolic acidosis by providing substrate

Don't start insulin for several hours!

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#2: Regular insulin therapy

- CONS:
  - Drops the blood glucose "#" too quickly, changing the osmolality too quickly
  - Drops the electrolytes too quickly → shifts K^+ and P^+ intracellularly worsening clinical signs

Don't start insulin for several hours!

+

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Insulin therapy

- Regular insulin until nonketotic
- CRI vs. intermittent IM protocols
- Supplementation of glucose
- CAUTION: ↓ K^+, P^+, blood glucose, osmolality

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CRI insulin protocol

Insulin CRI(Regular Insulin) Cat- 1.1u/kg in 240ml 0.9% NaCl Dog- 2.2 u/kg in 240ml 0.9% NaCl	
Blood Glucose (mg/dL)	**Insulin CRI (ml/hr)**
>350	10
250-349	7
150-249	5 + 2.5% dextrose
100-149	3+ 5% dextrose
< 100	0

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CRI insulin protocol

- Insulin is used to lower blood glucose and to halt ketone production.
- Insulin administration should continue until ketosis has resolved, even if this means that glucose supplementation must be given to maintain euglycemia.

Insulin CRI(Regular Insulin) Cat- 1.1u/kg in 240ml 0.9% NaCl Dog- 2.2 u/kg in 240ml 0.9% NaCl	
Blood Glucose (mg/dL)	**Insulin CRI (ml/hr)**
>350	10
250-349	7
150-249	5 + 2.5% dextrose
100-149	3+ 5% dextrose
< 100	0

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CRI insulin protocol

2.2 U/kg regular insulin!!

Blood Glucose (mg/dL/mmol/L)	Fluids	Insulin CRI (ml/hr)
250 mg/dL 13.9 mmol/L	0.9% NaCl	10
200-250 mg/dL 11-13.9 mmol/L	NaCl + 2.5% dextrose	7
150-200 mg/dL 8.3-11 mmol/L	NaCl + 2.5% dextrose	5
100-150 mg/dL 5.5-8.3 mmol/L	NaCl + 5% dextrose	5
< 100 mg/dL < 5.5 mmol/L	NaCl + 5% dextrose	OFF

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Intramuscular (IM) protocol

- Start with 0.2 U/kg, adjust based on glucose measurements
- Check blood glucose q 4 hours
- Insulin q 4 hours
- Slow correction
- Use small amt blood (esp cats), place central line

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IM insulin protocol

If BG is:	Give:
> 250 mg/dL >13.9 mmol/L	__ units regular insulin IM
150-250 mg/dL 8.3-13.9 mmol/L	__ U insulin IM + 2.5% dextrose to IVF
100-150 mg/dL 5.6 – 8.3 mmol/L	__ U insulin IM + 5% dextrose to IVF
< 100 mg/dL <5.6 mmol/L	No insulin; 5% dextrose in IVF

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BG CONCEN-TRATION (mg/dl)	SOLUTION TO FULFILL FLUID REQUIREMENTS	RATE OF ADMIN OF INSULIN SOLUTIONS
>250	0.9% NaCl	10
200-250	0.45% NaCl + 2.5% Dextrose	7
150-200	0.45% NaCl + 2.5% Dextrose	5
100-150	0.45% NaCl + 5% Dextrose	5
<100	0.45% NaCl + 5% Dextrose	Stop Insulin Infusion

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IM PROTOCOL

IF BG IS:	GIVE:
>250	____Units Regular Insulin IM
150-250	____Units Regular Insulin IM + Add 2.5% Dextrose to IVF
100-150	____Units Regular Insulin IM + Add 5% Dextrose to IVF
<100	No Insulin; 5% Dextrose to IVF

*CALL IF BG <80 OR >350

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S. Buob et al (ACVIM 2010) An Intermittent Insulin Protocol Improves Metabolic Acidosis Faster Than a CRI of Regular Insulin in Feline DKA

- Human study: children with DKA: SQ glargine + CRI of regular insulin → faster resolution of acidosis.
- Animal study: Evaluate SQ glargine + IM regular insulin in cats with DKA.
- N = 16 cats (8 CRI; 8 intermittent insulin) →
- 11/16 survived

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S. Buob et al (ACVIM 2010) An Intermittent Insulin Protocol Improves Metabolic Acidosis Faster Than a CRI of Regular Insulin in Feline DKA

- Intermittent: faster time to resolution of acidosis (16 hr vs. 38 hrs)
- No differences were detected between the two groups for:
  - hospitalization time
  - nadir of the hematocrit (p = 0.65)
  - resolution of ketonemia (p = 0.50)

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JOURNAL OF
Veterinary Emergency
AND Critical Care

Original Study

Use of lispro insulin for treatment of diabetic ketoacidosis in dogs

Kirk W. Sears DVM*, Kenneth J. Drobatz DVM, MSCE, DACVIM, DACVECC and Rebecka S. Hess DVM, DACVIM

Article first published online: 5 MAR 2012
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Issue

Journal of Veterinary Emergency and Critical Care
Volume 22, Issue 2, pages 211–218, April 2012

- To characterize the use of lispro insulin in dogs with diabetes ketoacidosis (DKA) and to compare the length of time required for resolution of hyperglycemia, ketosis, and acidosis, respectively, in dogs with DKA treated with lispro or with regular insulin.

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Use of lispro insulin for treatment of diabetic ketoacidosis in dogs

- N = 12
- The median time to biochemical resolution of DKA in dogs treated with lispro insulin was significantly shorter (26 h; range 26–50 h) than in dogs treated with regular insulin (61 h; range, 38–80 h, P = 0.02).
- No adverse effects were observed in association with IV lispro insulin administration
- Treatment of DKA in dogs with IV CRI lispro insulin is safe, and as effective as treatment with regular insulin.

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PATIENT MONITORING AND SUPPORTIVE CARE

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Therapeutics

- IV catheterization
  - Sampling line?
  - Central line?
    - TPN
    - CVP
    - Ease of blood draw

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IV access

- Double or triple lumen
  - Aseptic
  - TPN
  - CVP
  - Ease of blood draw
- Venocath (central line)
  - Hindleg
  - Jugular

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Therapeutics

- IV catheterization
- Fluid resuscitation
- Treat underlying infection

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Treat underlying infection

- Antibiotic therapy once UA/UC pulled!

- LOW-gun until culture +!
- Ampicillin 22 mg/kg IV q. 8
- Cefazolin 22 mg/kg IV q. 8
- Unasyn 25-30mg/kg IV q. 8

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Therapeutics

- IV catheterization

- Fluid resuscitation
- Treat underlying infection
- Slowly treat DKA
  - Regular insulin therapy

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Slowly treat the DKA: Bonder

- After 6-12 hours of IVF therapy, reassess for hydration
- 2 units IM regular insulin q. 4 hours if BG > 250-350 mg/dL (13.9-19.4 mmol/L)
- 1 unit IM regular insulin q. 4 hours if BG > 150-250 mg/dL (8.3 – 13.9 mmol/L)

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Patient monitoring

- Blood glucose every 2-4 hours
- Body weight, TPR
- Electrolytes q 8-12 hours
- PCV/TS/AZO
- Blood pressure
- Urine ketones

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Nutrition

- Great candidate for a FEEDING TUBE!
  - NE tube
  - Hydration (cardioprotective)
    - 90 ml/kg/day
  - Clinicare CRI's or Vivonex
  - Clinicare: 1 kcal/ml
  - Typical cat = 200 kcal/day
  - Start slow
    - Day 1: ¼ of RER
    - Day 2: ½ of RER
    - Day 3: Full RER

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Key points to remember!

- Don't start long-acting insulin until:
  - Eating and drinking
  - Improved attitude/hydration
  - Normotensive
  - No longer vomiting
  - Clinical signs improved

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HHS

Hyperglycemic, Hyperosmolar Syndrome

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Hyperglycemic Hyperosmolar Syndrome: (HHS)

- Life-threatening:
  - Hyperosmolarity
  - Dehydration
- Not always ketotic (or low levels)
- More common in cats than dogs
- Severe hyperglycemia
  - >600 mg/dl
- Hyperosmolarity
  - >350 mosm/kg
- Severe clinical dehydration
- Lack of ketones
- Lactic acidosis
- CNS depression

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Hyperglycemic Hyperosmolar Syndrome: (HHS)

- Severe hyperglycemia (>600 mg/dl/33.3 mmol/L)
- Hyperosmolarity (>350 mosm/kg)
- Severe clinical dehydration
- Lack of ketones (serum/urine)
- Lack of/mild acidosis
- CNS depression (lethargy)!

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Koenig et al: Hyperglycemic, hyperosmolar syndrome in feline diabetics: 17 cases (1995-2001)

- N = 17 cats
- Criteria:
  - hyperglycemic
  - non-ketotic
  - hyperosmolar syndrome
- HHS: calculated Osm > 350, effective Osm > 330, Glu > 600, urine ketone negative.

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Koenig et al: Hyperglycemic, hyperosmolar syndrome in feline diabetics: 17 cases (1995-2001)

- PC: long-standing DM receiving insulin for many months; older cats; presenting with: pu/pd, lethargy
- PE findings: profound dehydration, lactic acidosis, azotemia
- Serious concurrent disease in 15/17 cats (88%) → Neuro signs + respiratory signs
  - Renal failure
  - Respiratory complications
  - Congestive heart failure
  - Infection
  - Neoplasia
  - GI disease

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Koenig et al: Hyperglycemic, hyperosmolar syndrome in feline diabetics: 17 cases (1995-2001)

- Rarely found pancreatitis or hepatic disease with HHS
- 65% died or euthanized with 10 hours of presentation
- Long-term survival: 12%

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Hyperglycemic Hyperosmolar Syndrome: (HHS)

- Don't bolus fluids unless hypotensive!

- SLOW rehydration!
  - Plasmalyte 148 or LRS
  - CAREFUL with 0.45% NaCl or D5W
  - Water bowl!

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Hyperglycemic Hyperosmolar Syndrome: (HHS)

- CAUTIOUS correction of hyperglycemia
- Monitor mentation, electrolytes (sodium), blood glucose levels

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Hyperglycemic Hyperosmolar Syndrome: (HHS)

- Don't use Na Bicarbonate
  - Too hyperosmolar!
- Safest: oral water
  - NE tube with oral water?
  - Maintenance oral water

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CONCLUSIONS

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Pitfalls

- Never use insulin IM, IV unless regular insulin!
- No SC insulin if dehydrated!
- Central line for ease of patient

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Pitfalls

- When in doubt, slow correction of glucose!
- No BIG guns pending culture!
- Perfusion, perfusion, perfusion!
- Start on long-acting insulin once eating

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Conclusions:

- What happens chronically, treat chronically.
- Appropriate client communication with DKA, HONK, DM
  - Long-term management
- Not all DKA require 24 hour care
- Don't reach for insulin too early!

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- NAVC 2015
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Questions?

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