

2026 AAHA Diabetes Management Guidelines for Dogs

Andrew Bugbee, DVM, DACVIM (Small Animal Internal Medicine),[†]
 Renee Rucinsky, DVM, DABVP (Feline),[†] Elizabeth Alvarez, DVM, DABVP (Canine and Feline),
 Audrey Cook, BVM&S, FRCVS, DACVIM (Small Animal Internal Medicine), DECVIM-CA, DABVP (Feline),
 Patty Lathan, VMD, MS, DACVIM (Small Animal Internal Medicine), Cassie Panning, BS, CVT, VTS (Nutrition)

ABSTRACT

A substantial number of dogs develop diabetes mellitus (DM), a serious endocrine disorder that can lead to other physiologic complications. Although DM management can be complex, successful control that alleviates the patient's clinical signs and avoids hypoglycemia is achievable in most dogs. The *2026 AAHA Diabetes Management Guidelines for Dogs* retain clinically relevant information from the *2018 AAHA Diabetes Management Guidelines* and present new findings and expert opinions. Subjects such as pathophysiology, diagnosis, various insulin options for dogs, how to monitor dogs receiving insulin, diet and physical activity recommendations, advantages and disadvantages of at-home glucose monitoring devices, diagnosing and treating diabetic ketoacidosis, and client education are all discussed. The guidelines also cover how to identify patients who are at risk of developing DM and how to differentiate DM from transient or mild hyperglycemia. (*J Am Anim Hosp Assoc* 2026; 63:■■■-■■■. DOI 10.5326/JAAHA-MS-7583)

AFFILIATIONS

Texas A&M University, College Station, Texas (A.B.); Mid Atlantic Cat Hospital, Mid Atlantic Feline Thyroid Center, Queenstown, Maryland (R.R.); University of Wisconsin – Madison, Madison, Wisconsin (E.A.); Texas A&M University, College Station, Texas (A.C.); Louisiana State University, Baton Rouge, Louisiana (P.L.); Minneapolis, Minnesota (C.P.)

CONTRIBUTING REVIEWERS

Thomas Schermerhorn, VMD, DACVIM (Small Animal Internal Medicine), Kansas State University, Manhattan, Kansas

Ellen Behrend, VMD, PhD, DACVIM (Small Animal Internal Medicine), Auburn University, Auburn, Alabama

Correspondence: guidelines@aaha.org

[†] Andrew Bugbee and Renee Rucinsky are cochairs of the AAHA Diabetes Management Guidelines task force.

These guidelines were prepared by a task force of experts convened by the American Animal Hospital Association. This document is intended as a guideline, not an AAHA Standard of Care. These guidelines and recommendations should not be construed as dictating an exclusive protocol, course of treatment, or procedure. Variations in practice may be warranted based on individual patient needs, resources, and limitations unique to each practice setting. Evidence-guided support for specific recommendations has been cited whenever possible and appropriate. Other recommendations are based on practical clinical experience and a

consensus of expert opinion. More research is needed to further substantiate some recommendations. As each case is different, veterinary teams must base their decisions on the best available scientific evidence in conjunction with their knowledge and experience. All task force members contributed to the development of the guidelines. Although task force members attempted to reach an expert consensus, individual members of the task force are not responsible for the final guidelines or specific aspects of the guidelines.

Conflict of interest statement: Andrew Bugbee has received speaking, consultation, and honorarium fees from Boehringer Ingelheim, Dechra, Elanco, Purina, Royal Canin and Zomedica, and research support from ScoutBio/CEVA. Renee Rucinsky has received speaking, consultation, and honorarium fees from Boehringer Ingelheim and Elanco. Audrey Cook has received speaking and consultation fees from Boehringer Ingelheim and Elanco, research support from Dechra, and is an Advisory Board member for VetRec. Patty Lathan has received honoraria and consultation fees from Boehringer Ingelheim, Dechra, IDEXX, Hill's Pet Nutrition, and ScoutBio, and research support from Dechra and IDEXX.

AAHA gratefully acknowledges our task force facilitator, Mia Cary, DVM (she/her), of Cary Consulting, and Theresa Entriiken, DVM, developmental editor.

The AAHA Diabetes Management Guidelines are generously supported by Adapet Medical, Boehringer Ingelheim, Dechra, and Merck Animal Health.

ABBREVIATIONS

ALP, alkaline phosphatase; BG, blood glucose; BGC, blood glucose curve; BHB, beta-hydroxybutyrate; BP, blood pressure; CBC, complete blood count; CGM, continuous glucose monitor; DKA, diabetic ketoacidosis; DM, diabetes mellitus; ESS, euthyroid sick syndrome; FSL, FreeStyle Libre; HbA1C, hemoglobin A1C; HC, hypercortisolism; IM, intramuscular; IR, insulin resistance; NPH, neutral protamine Hagedorn; PD, polydipsia; PBGM, portable blood glucose monitor; PP, polyphagia; PU, polyuria; PZI, protamine zinc recombinant human insulin; SQ, subcutaneous; TT4, total thyroxine; U, units; UG, urine glucose

Guidelines Table of Contents

Pathophysiology and Management

- 1 Section 1: Overview of Diabetes Mellitus in Dogs
- 2 Section 2: Recognizing and Diagnosing DM in Dogs
- 3 Section 3: Diabetic Ketoacidosis in Dogs
- 4 Section 4: Insulin Treatment
- 5 Section 5: Monitoring Dogs Receiving Insulin
- 6 Section 6: Dietary Management in Diabetic Dogs
- 7 Section 7: Troubleshooting in Diabetic Dogs
- 8 Section 8: Client Education

General Monitoring Considerations

- 9 Section 9: Glucose Monitoring
- 10 Section 10: Other Methods for Monitoring Glycemic Status
- 11 Section 11: Hypoglycemia and Troubleshooting Algorithms

Tables

- 12 Table 4.1: Insulin Products for Dogs

Figures

- 13 Figure 3.1: Diabetic Ketoacidosis Protocol for Dogs
- 14 Figure 5.1: Monitoring and Adjusting Intermediate or Long-Acting Bolus Insulin Treatment in Dogs: NPH, Lente, or PZI
- 15 Figure 9.1: Monitoring Glucose in Diabetic Dogs
- 16 Figure 11.1: Managing Hypoglycemia in Dogs
- 17 Figure 11.2: Troubleshooting Diabetic Dogs Receiving High Doses of Insulin

Introduction

Diabetes mellitus (DM) can be successfully managed in dogs but requires an up-to-date understanding of diabetes pathophysiology and a thorough grasp of how to incorporate new treatments into therapeutic approaches. Staying informed about emerging treatment options is crucial. Diabetic canine patients need diligent clinical, observational, and diagnostic assessments and individualized treatment and monitoring plans. Rewarding diabetes management is intertwined with committed client education, communication, and compliance.

These guidelines retain clinically relevant information from the 2018 AAHA *Diabetes Management Guidelines*¹ and present new findings and expert opinion on diagnosing, treating, and monitoring DM in dogs, recognizing at-risk patients, and identifying patients with hyperglycemia who do not require diabetic treatment. The refreshed reorganization of these guidelines separates the feline and canine content into two distinct publications to provide veterinarians with an easier reference to managing the disease in each species.

The guidelines cover traditional and new insulin formulations for dogs and when and how to use each. Easy-to-use at-home monitoring tools such as continuous glucose monitors and glucometers and methods to help detect abnormalities in urine are highlighted. The guidelines also feature new or updated algorithms, tables, and boxes for quick reference on a variety of topics including managing hypoglycemia, addressing diabetic ketoacidosis, and troubleshooting suboptimal responses to therapy. Dietary management is also discussed, and key points in client education on treatment and monitoring, including the financial and time commitments and lifestyle changes, are emphasized.

Diabetes Mellitus Pathophysiology and Management in Dogs

Section 1: Overview of Diabetes Mellitus in Dogs

Top 3 Takeaways

1. Dogs develop a form of DM characterized by an absolute insulin deficiency from permanent beta cell loss.
2. Conditions that can contribute to insulin resistance in dogs (but alone do not typically lead to DM) include obesity, hypercortisolism, hypertriglyceridemia, hypothyroidism, dental disease, systemic infection, pancreatitis, pregnancy/insulin-resistant effects of diestrus, or medications.
3. Prognosis for dogs with DM is favorable with appropriate treatment and monitoring.

DM is associated with protracted hyperglycemia due to beta-cell loss and/or diminished insulin sensitivity in tissues. Although this is likely simplistic, dogs with DM are traditionally regarded as suffering from absolute insulin deficiency because beta-cell loss tends to be progressive and is often believed to involve a component of immune-mediated destruction, vacuolar degeneration, or damage secondary to repeated bouts of chronic pancreatitis.²

DM is most commonly a disease of middle-aged to older dogs. A genetic susceptibility is a suspected risk factor, with certain breeds of dogs (Australian terriers, beagles, Samoyeds, keeshonden) being more susceptible.³ The exact pathogenesis of DM in dogs is unknown but is believed to be complex and multifactorial. Conditions that can contribute to insulin resistance in dogs (but alone do not typically lead to DM) include obesity, hypercortisolism, hypertriglyceridemia, hypothyroidism, dental disease, systemic infection, pancreatitis, pregnancy/insulin-resistant effects of diestrus, or medications (e.g., steroids and progestins).¹

Prognosis for dogs with DM is favorable with proper treatment and monitoring. Most dogs remain controlled after proper insulin therapy has been established. Most dogs tolerate short periods of moderate hyperglycemia (BG >180 mg/dL) as long as the dog spends most of the day below this range and clinical signs are controlled. This mild elevation in dogs does not seem to lead to retinopathy, strokes, and other complications as it can in humans after decades of hyperglycemia.

Section 2: Recognizing and Diagnosing DM in Dogs

Top 3 Takeaways

1. Although insulin resistance (IR) is not suspected to play a major role in the development of DM in dogs, diabetic dogs often develop conditions that lead to IR and complicate regulation.
2. Documenting glucosuria and concurrent hyperglycemia establishes a DM diagnosis in dogs.
3. Absolute insulin deficiency predictably results in ketosis within several days, so most dogs have a relatively short period of clinical signs before presentation.

As DM progresses without treatment, dogs typically present with persistent hyperglycemia and glucosuria. The average canine renal threshold for glucose is 160–200 mg/dL, so expect polyuria (PU) and polydipsia (PD) when the blood glucose (BG) exceeds this range. Insulin deficiency also induces polyphagia (PP) with weight loss. Although clinical signs may not be obvious to clients as dogs slowly progress during the early stages of DM development, most overtly insulin-deficient dogs exhibit significant clinical signs for a relatively short period before presentation. Some patients, especially if chronically untreated, may show systemic signs of illness due to diabetic ketosis/ketoacidosis (DKA), such as lethargy, weakness, anorexia, vomiting, and depression. If ketosis is present, the pet's breath may have a sweet odor.

Obtain a complete history, including information about current medications, particularly use of glucocorticoids or progestins, which may impact insulin sensitivity. In intact females, identify the time of the last estrus. Obtain current dietary management and body weight histories. Establish the dog's ideal body weight and typical daily calorie intake to help inform feeding strategies moving forward (see Section 6, Dietary Management).

Physical examination findings can be relatively normal early in the course of DM. Increased fat mobilization leads to hepatomegaly and increased catabolism. Some dogs present with dehydration, poor hair coat, decreased muscle condition, and/or diabetic cataracts. Dogs presenting with DM and concurrent disorders may exhibit a variety of other signs (e.g., abdominal pain with pancreatitis) that are not directly caused by DM.

Diagnosing DM in Dogs

Documenting glucosuria and concurrent hyperglycemia in a dog exhibiting characteristic clinical signs quickly establishes a DM diagnosis. In clinically stable patients (with an appropriate history) who are well hydrated and eating, the following information is sufficient to diagnose DM and initiate treatment:

- **Blood glucose:** As defined by Project ALIVE, a fasted or nonfasted BG $\geq$ 200 mg/dL and evidence of glucosuria and classic clinical signs

Causes of Transient Versus Sustained Insulin Resistance in Dogs

It is important to differentiate patients with clinical DM from those with transient causes of insulin resistance or those with conditions causing sustained insulin resistance increasing their risk of developing DM.

Insulin deficiency

- Diabetes mellitus

Transient insulin resistance

- Postprandial: Mild, physiologic (BG normalizes within a few hours)
- "Stress:" Mild in canines, lasts 4–6 hr
- Pregnancy* (diestrus—see Diestrus Diabetes below)
- Iatrogenic (certain drugs)
- Acute pancreatitis
- Sepsis

Sustained insulin resistance

- Hypercortisolism
- Acromegaly (mammary tumor associated)
- Hyperglucagonemia
- Pheochromocytoma

of hyperglycemia (with no other plausible cause) or a hyperglycemic crisis.⁴

- **Urine glucose/urine ketones:** Urinalysis in dogs with DM reveals glucosuria. Ketonuria may also be noted. Although patients with DKA are usually ketonuric, ketonuria is not synonymous with DKA (See Section 3, Diabetic Ketoacidosis in Dogs).

*Diestrus Diabetes

Every heat cycle in a dog (irrespective of whether pregnancy occurs) induces an approximately 9 wk diestrus (luteal) phase, accompanied by high levels of progesterone. This drives the production of growth hormone by mammary tissue, which causes profound insulin resistance. Depending on genetics and other factors, this can result in overt DM in nonpregnant (diestrus diabetes) or pregnant (gestational diabetes) dogs. Clinical signs are often noted 30–50 days following estrus and are identical to those routinely seen in dogs with DM.^{5,6} Although the underlying pathogenesis reflects an insulin-resistant state, all affected dogs should be treated with insulin. Patients should also be screened for concurrent pyometra, particularly if ketonuric or systemically unwell.

Prompt ovariectomy results in a rapid decline in growth hormone and may allow some dogs to undergo diabetic remission. This often occurs within a few weeks, so patients must be monitored closely to prevent inadvertent hypoglycemia while receiving insulin.

Gestational DM has also been described in dogs. In some individuals, DM resolves within days of whelping but may be permanent.⁷ These dogs should also be ovariectomized to prevent dysregulation of DM with subsequent heat cycles.

Evaluating Newly Diagnosed Diabetic Dogs

A comprehensive diagnostic evaluation provides useful baseline information and allows for identification of any metabolic derangements or comorbidities. Recommended tests are listed below in order of clinical and diagnostic importance for dogs newly diagnosed with DM:

- **Chemistry panel:** Expect hyperglycemia (BG >200 mg/dL). Increased fat mobilization can lead to hypercholesterolemia and hypertriglyceridemia. Expect mild to moderate increases in alkaline phosphatase (ALP) and possibly alanine aminotransferase (ALT). Azotemia suggests dehydration or concurrent renal disease. Serum electrolytes are usually within normal limits in stable new diabetic dogs, but dogs with DKA may have sodium, potassium, and acid base status derangements.
- **Urinalysis:** Expect glucosuria. Urine specific gravity is often <1.030. Protein, ketones, bacteria, and/or casts may also be identified.
- **Quantitative urine culture:** An appropriate test in patients with signs of urinary tract infection or an active urine sediment. Bacteriuria without clinical or laboratory abnormalities may represent subclinical bacteriuria. Treatment may not be warranted unless urinary signs develop or there is concern for infection-related insulin resistance. For more information on antimicrobial stewardship, see the *AAFP/AAHA Antimicrobial Stewardship Guidelines* at aaha.org.

- **Quantitative pancreas-specific lipase immunoreactivity (PLI):** Not required unless clinical signs of pancreatitis are present. Pancreatitis is a common comorbidity and, if clinical signs are present, may warrant supportive care, appetite support, pain control, and/or consideration in planning nutrition recommendations.⁸
- **CBC:** Stable new diabetic dogs may have a stress leukogram. Dogs with DKA or pancreatitis may have an inflammatory leukogram.
- **Total T4 (TT4):** The task force does not recommend routine measurement of TT4 in newly diagnosed diabetic dogs unless this condition is suggested by physical examination or other findings. As euthyroid sick syndrome is common in dogs with untreated DM, a low TT4 should be further investigated by measuring free T4 by equilibrium dialysis and thyroid-stimulating hormone.
- For further information on diagnosing hypothyroidism, see the *AAHA Selected Endocrinopathies of Dogs and Cats Guidelines* at aaha.org.

Initial Evaluation of the Diabetic Dog

- Perform a physical examination and gather a thorough history including diet, treats, and current medications.
- Evaluate for risk factors or comorbidities that may interfere with the patient's response to treatment if consistent clinical signs or abnormalities on history or physical examination are present. Conditions may include obesity, hypercortisolism, hypertriglyceridemia, hypothyroidism, dental disease, systemic infection, pancreatitis, pregnancy/insulin-resistant effects of the diestrus phase, or certain medications (e.g., steroids or progestins).
- Spaying or neutering dogs following diagnosis with DM may help mitigate hormonal influence on insulin sensitivity or treatment response.

The task force referenced Project ALIVE for standardized DM terminology and to define criteria used for DM diagnosis. For more information on Project ALIVE, see esve.org/alive/search.aspx.

Section 3: Diabetic Ketoacidosis in Dogs

Top 3 Takeaways

1. Diagnosis of DKA relies on identifying compatible clinical signs in sick diabetic patients and confirming ketosis and acidosis.
2. Ketones can be measured with a point-of-care ketone meter or standard urine test strips, although ketone meters measuring blood beta-hydroxybutyrate (BHB) more accurately identify earlier stages of DKA.
3. Because of the affordability and ease of point-of-care ketone meters, the task force recommends having a handheld ketone meter available in the clinic.

DKA: Treatment for Severely Compromised Patients

Refer to a 24 hr facility for patients presenting with any of the following: weak/laterally recumbent/obtunded, severely dehydrated, uncontrolled DM and systemic illness (PU/PD, weight loss, vomiting, diarrhea, anorexia), hypovolemic/hypotensive, severely acidotic, significant electrolyte abnormalities, hyperosmolar, and/or CNS signs.

DKA: Primary Care Treatment for Less Critical Patients

Consider referral if your clinic's capabilities do not allow for optimal nursing care and hospitalization.

Start supportive care if transfer to referral center is not immediately available.

- ◆ Administer IV fluids to correct dehydration over 6–12 hr (12–24 hr for patients prone to fluid overload) and meet maintenance fluid requirements. Balanced crystalloids (e.g., lactated Ringer's solution, Plasmalyte, or Normosol R) are ideal.^{a,b}
- ◆ Monitor BG with a PBGM or CGM. If using CGM, confirm accuracy of initial BG readings via comparison to PBGM results.
- ◆ Add potassium chloride (KCl) to fluids, and/or potassium phosphate (KPhos) as needed.^{a,b,c}
- ◆ Once on fluids and the potassium is >3.0–3.5 mEq/L, it is safe to administer 0.1 U/kg regular (R) insulin IM. Try to initiate insulin within 4–6 hr of presentation.
- ◆ Measure BG every 1–2 hr and give 0.1 U/kg R insulin IM until BG is <250 mg/dL.
- ◆ Then add dextrose to the fluids to make a 2.5–5% solution and maintain the BG around 200–250 mg/dL while continuing insulin administration.
- ◆ Monitor BHB every 8–12 hr until the patient is stable.^d
- ◆ Manage concurrent disease, including infections (e.g., urinary tract infection) and pancreatitis.
- ◆ Nursing care! Warmth, food, and love.
- ◆ When BG is <250 mg/dL and BHB has improved or resolved, if the patient is not consistently eating or drinking, regular insulin injections can be continued every 1–2 hr IM or transitioned to 0.2–0.4 U/kg SQ (if the dog is rehydrated) every 4–6 hr.
- ◆ When BG <250 mg/dL, BHB has improved or resolved, euhydration is achieved, and the dog is eating consistently, transition to SQ intermediate or basal-acting insulin.
- ◆ Glycemic regulation before discharge is not a goal of DKA management and should not be expected.

BG, blood glucose; BHB, beta-hydroxybutyrate; CGM, continuous glucose monitor; CNS, central nervous system; DKA, diabetic ketoacidosis; DM, diabetes mellitus; KCl, potassium chloride; KPhos, potassium phosphate; IM, intramuscular; PBGM, portable blood glucose monitor; PD, polydipsia; PU, polyuria; R, regular insulin; SQ, subcutaneous; U, unit

a. Gal A, Odunayo A. Diabetes ketoacidosis and hyperosmolar hyperglycemic syndrome in companion animals. *Vet Clin North Am Small Anim Pract* 2023;53(3):531–50.

b. Panciera D. Fluid therapy in endocrine and metabolic disorders. In: DiBartola S, ed. *Fluid, Electrolyte, and Acid-Base Disorders in Small Animal Practice*. 4th ed. Philadelphia: WB Saunders; 2012:500–13.

c. Boag A. Diabetic ketoacidosis and hyperglycemic hyperosmolar syndrome. In: Mooney CT, Peterson ME, Shiel RE, eds. *BSAVA Manual of Canine and Feline Endocrinology*. 5th ed. Gloucester: British Small Animal Veterinary Association; 2023:243–51.

d. Stojanovic V, Ihle S. Role of beta-hydroxybutyric acid in diabetic ketoacidosis: a review. *Can Vet J* 2011;52(4):426–30.

FIGURE 3.1

Diabetic Ketoacidosis Protocol for Dogs

Diabetic ketoacidosis is a life-threatening complication of DM. Although DKA is more commonly seen in naive diabetic patients, it may occur in treated diabetics. Concurrent diseases (e.g., pancreatitis, urinary tract or other infections, hypercortisolism, and renal disease) often precipitate DKA. Insulin deficiency leads to reduced glucose transport into cells and increased release of free fatty acids, resulting in increased hepatic ketone production, acidosis, electrolyte imbalances, and dehydration (Figure 3.1).

A continuous glucose monitor (CGM) (e.g., FreeStyle Libre) records the interstitial glucose concentration continuously over multiple days. See Section 9, Glucose Monitoring for more information.

Diagnosis of DKA relies on identifying compatible clinical signs in diabetic patients, who are often sick, and confirming ketosis and acidosis. Dogs with diabetic ketoacidosis may show very elevated BG concentrations, azotemia, decreased total CO₂ or bicarbonate secondary to metabolic acidosis, osmotic diuresis, dehydration, and, in the case of profound hyperosmolality, coma. Urinalysis will reveal the presence of glucose. It may also show the presence of protein, ketones, bacteria, and/or casts.

How to Formulate Dextrose-Containing Fluids^a

Amount of 50% Dextrose Solution Added to 1L Bag of Isotonic Crystalloids*	Final Dextrose Concentration
25 ml	1.25%
50 ml	2.5%
100 ml	5%
150 ml	7.5%

*Remove an equivalent volume of the isotonic crystalloid fluid from the bag before adding 50% dextrose solution.

a. Modified from Table 14, *AAHA Fluid Therapy Guidelines*. Available at aaha.org/resources/2024-aaha-fluid-therapy-guidelines-for-dogs-and-cats/.

If acid/base status measurement is not available, treat sick, ketotic diabetic patients as DKA patients (Figure 3.1). Measure ketones with a point-of-care handheld ketone meter (these measure beta-hydroxybutyrate [BHB, see Box, Measuring Blood Beta-Hydroxybutyrate]) or with standard urine test strips (these measure acetoacetate). Both may be helpful, but BHB more accurately

Appropriate initial fluid therapy alone will provide significant glucose, electrolyte, and acid-base correction in the absence of insulin. However, only insulin eliminates ketosis so attempts should be made to start insulin within 4–6 hr of hospital admission in most cases.

Measuring Blood Beta-Hydroxybutyrate

BHB is the earliest and most predominant ketone body produced during diabetic ketoacidosis in dogs. Point-of-care whole blood ketone meters are available to measure BHB, and a commercial model has been validated in dogs.^a

The currently validated model, Precision Xtra (Abbott), has been discontinued but strips will continue to be available for an unknown period of time. Veterinary practices must plan to transition to new devices and watch for further validation studies in other models.

a. Henderson DW, Schlesinger DP. Use of a point-of-care beta-hydroxybutyrate sensor for detection of ketonemia in dogs. *Can Vet J* 2010;51(9):1000–2.

identifies earlier stages of ketosis.⁹ Clinical laboratories also measure BHB if a point-of-care ketone meter is not an option; however, rapid identification of ketone elevation is important. Because of the affordability and ease of point-of-care ketone meters, the task force recommends having a handheld ketone meter available in the clinic.

Section 4: Insulin Treatment

Top 3 Takeaways

1. All diabetic dogs are insulin deficient and require exogenous insulin therapy.
2. Calculate the insulin dose based on the dog's estimated ideal body weight and round down to the nearest whole unit (U).
3. Diabetes management continues to evolve, necessitating that veterinarians stay up to date on new therapeutic options and changes to treatment or monitoring recommendations.

Intermediate (Bolus) Insulin Treatment for Dogs

Treating DM in dogs always requires exogenous insulin therapy. Start insulin on the day of diagnosis. Either U-40 porcine insulin zinc suspension (Vetsulin) or U-100 neutral protamine Hagedorn (NPH) are recommended by task force members as a good first-choice

option for diabetic dogs, using a starting dose of 0.25–0.5 U/kg every 12 hr (Table 4.1). Most dogs are well controlled on insulin at an average dose of 0.5 U/kg every 12 hr (range of 0.2–1.0 U/kg every 12 hr). It is typically recommended to calculate the insulin dose based on the dog's estimated ideal body weight and to round down to the nearest whole U. Use judicious initial dosing if concurrently changing the diet, as the nutrient profile of the new diet or changes in the patient's food intake may affect their response to insulin.

Always give a bolus insulin after the dog consumes a full meal. If less than 50% of the meal is eaten, one-half of their normal insulin dose should be administered, and the dog should not be offered any additional food or treats until the next scheduled mealtime. Although consistent timing of a dog's feeding and insulin dosing schedule is important, clients should be allowed flexibility within a 2-hr window on each side of the normal dosing/feeding time with occasional missed or off-schedule doses considered acceptable.

TABLE 4.1
Insulin Products for Dogs

Insulin Product	Product Description	Brand Name (Manufacturer)	Veterinary FDA Approval Status	Peak Action (Nadir) and Duration of Effect	Starting Dose	Concentration
Intermediate-Acting Insulin						
Lente	Porcine insulin zinc suspension	Vetsulin (Merck Animal Health)	Approved	Nadir 1–10 hr. ^a Duration 10–24 hr. ^a	0.25–0.5 U/kg <i>q</i> 12 hr. Manufacturer recommended starting dose: 0.5 U/kg <i>q</i> 24 hr.	U-40
					Comments: <ul style="list-style-type: none">Commonly used in dogs; injection pens (in either 0.5 U or 1 U increments) are available.Shaking insulin bottle is required until the suspension appears homogeneously white and frothy.	
Glargine U-100	Recombinant DNA origin human insulin	Lantus (Sanofi) Basaglar (Lilly) Semglee (Viatris & Biocon Biologics) Glargine yfgn (Viatris & Biocon Biologics) Rezvoglar (Lilly)	Not approved	Nadir 6–10 hr. ^b Duration 12–20 hr. ^b	0.3 U/kg <i>q</i> 12 hr. ^c 0.25–0.5 U/kg <i>q</i> 12 hr. ^b	U-100
					Comments: Potential option in dogs.	
Neutral protamine Hagedorn (NPH)	Recombinant DNA origin human insulin	Novolin N (Novo Nordisk) Humulin N (Lilly)	Not approved	Nadir 0.5–8.5 hr. ^d Duration 4–10 hr. ^d	0.25–0.5 U/kg <i>q</i> 12 hr. ^d	U-100
Long-Acting Insulin						
Protamine zinc insulin (PZI)	Recombinant DNA origin human insulin	ProZinc (Boehringer Ingelheim)	Approved	Nadir 8–12 hr. ^d	0.25–0.5 U/kg <i>q</i> 12 hr. ^d Manufacturer recommended dose: 0.5–1 U/kg <i>q</i> 24 hr. ^e	U-40

(Continued on next page)

TABLE 4.1, CONTINUED
Insulin Products for Dogs

Insulin Product	Product Description	Brand Name (Manufacturer)	Veterinary FDA Approval Status	Peak Action (Nadir) and Duration of Effect	Starting Dose	Concentration
PZI, cont.	Comments: <ul style="list-style-type: none">Once-daily dosing in dogs is FDA approved. Can have a prolonged duration of action in dogs, which requires consideration of starting only once-daily administration or closely monitoring initial insulin response with a CGM to minimize the risk of clinical hypoglycemia and/or rebound hyperglycemia.^fSome clinicians recommend a starting dose for new diabetics of 0.25 U/kg <i>q</i> 12 hr, with 0.5 U/kg <i>q</i> 12 hr reserved for challenging diabetics.					
Basal Insulin						
Degludec	Recombinant DNA origin human insulin	Tresiba (Novo Nordisk)	Not approved	Peakless	0.5 U/kg <i>q</i> 24 hr ^g	U-100, U-200
						U-200 requires administration using the manufacturer provided pre-filled insulin pen, and is best used in large breed dogs due to the pen's dosing increments of 2 units.
Comments: <ul style="list-style-type: none">Can be considered as a first option insulin in dogs.Does not need to be given in association with a meal.Administration <i>q</i> 12 hr may be needed in some dogs and may reduce inter-day glycemic variability.^hUnbranded generic available from manufacturer.						
Glargine U-300	Recombinant DNA origin human insulin	Toujeo (Sanofi)	Not approved	Peakless	0.5 U/kg <i>q</i> 24 hr ⁱ	Requires administration using the manufacturer provided pre-filled insulin pen in increments of 1 unit.
Comments: <ul style="list-style-type: none">Can be considered as a first option insulin in dogs.Does not need to be given in association with a meal.Administration <i>q</i> 12 hr may be needed in some dogs and may reduce inter-day glycemic variability.^hUnbranded generic available from manufacturer.						

BG, blood glucose; CGM, continuous glucose monitor; NPH, neutral protamine Hagedorn; PZI, protamine zinc insulin; U, unit.

- Graham PA, Nash AS, McKellar QA. Pharmacokinetics of porcine insulin zinc suspension in diabetic dogs. *J Small Anim Pract.* 1997;38(10):434–8.
- Fracassi F, Boretta FS, Sieber-Ruckstuhl NS, et al. Use of insulin glargine in dogs with diabetes mellitus. *Vet Rec* 2012;170(2):52.
- Hess RS, Drobatsz KJ. Glargine insulin for treatment of naturally occurring diabetes mellitus in dogs. *J Am Vet Med Assoc.* 2013;243(8):1154–61.
- Maggiore AD, Nelson RW, Dennis J, et al. Efficacy of protamine zinc recombinant human insulin for controlling hyperglycemia in dogs with diabetes mellitus. *J Vet Intern Med.* 2012;26(1):109–15.
- Ward CR, Christiansen K, Li J, et al. Field efficacy and safety of protamine zinc recombinant human insulin in 276 dogs with diabetes mellitus. *Domest Anim Endocrinol.* 2021;75:106575.
- Ward CR, Christiansen K, Li J, Bryson WL, Jerrentrup KA, Kroh C. Field efficacy and safety of protamine zinc recombinant human insulin in 276 dogs with diabetes mellitus. *Domest Anim Endocrinol.* 2021;75:106575.
- Mott J, Gal A, Tardo AM, et al. Insulin degludec 100 U/mL for treatment of spontaneous diabetes mellitus in dogs. *J Vet Intern Med.* 2025;39(1):e17303.
- Miller M, Pires J, Crakes K, Greathouse R, Quach N, Gilor C. Day-to-day variability of porcine lente, insulin glargine 300 U/mL and insulin degludec in diabetic dogs. *J Vet Intern Med.* 2021;35(5):2131–2139.
- Tardo AM, Fleeman LM, Fracassi F, Berg AS, Guarino AL, Gilor C. A dose titration protocol for once-daily insulin glargine 300 U/mL for the treatment of diabetes mellitus in dogs. *J Vet Intern Med.* 2024;38(4):2120–2128.

Peakless (Basal) Insulin Treatment for Dogs

Recently, “peakless” insulin formulations that are more likely to have a flat time-action profile like basal insulin secretion have become available. These products include insulin degludec and glargine U-300 (Table 4.1), which have a long duration of action that may permit once-daily administration and are associated with less day-to-day glycemic variability, allowing for more predictable insulin responses.^{10,11,12}

Insulin degludec may be considered a first-choice option. Insulin degludec forms a multihexamer depot after subcutaneous injection that is slowly metabolized to release insulin monomers into the circulation. This delayed absorption conveys most of degludec’s long duration of action, while protein binding to albumin in circulation provides a minor contribution. In a recent study, ~84% of dogs were able to be maintained on once-daily administration.¹¹ However, some task force members have anecdotally reported slightly lower success rates with maintaining once-daily administration.

Insulin glargine U-300 is structurally identical to glargine U-100 but is a more concentrated and less potent formulation. The U-300 concentration delivers a smaller injection volume into the subcutaneous tissues, and the reduced surface area of the droplet slows metabolism and absorption, thereby extending its duration of action compared with glargine U-100. In a recent study, 60% of dogs maintained control on once-daily administration.¹⁰

Basal insulins do not have to be timed with feeding and are less likely to induce hypoglycemia when meals are missed.^{10,11} Thus, they may be helpful in managing patients with inconsistent or unpredictable appetites and may relieve clients’ burden of following a strict daily schedule. Basal insulins can be effective in dogs with comorbidities, including hypercortisolism.¹⁰

Because either *q* 12 hr or *q* 24 hr administration may be needed, the task force recommends placing a CGM on patients when starting a basal insulin to allow for rapid determination of each dog’s insulin dose and administration frequency. Although uncommon, some dogs may experience significant postprandial hyperglycemia that overwhelms the insulin’s basal action and may necessitate feeding smaller more frequent meals over the day or adding a low dose (0.25 U/kg) of an intermediate-acting insulin, commonly NPH or lente, following only one of the two daily meals.

Insulin Handling

When insulin is refrigerated and handled carefully, the task force is comfortable with clients continuing to use insulin beyond the

manufacturer-recommended in-use duration (28–56 days for most insulin types). Preservative-containing insulin vials in use for 6 mo were not shown to be at increased risk of bacterial contamination.¹³ If the insulin ever appears flocculent, discolored, or exhibits consistency changes, it should be replaced immediately. If there are any questions about insulin efficacy or a dog’s DM clinical signs recur when insulin use is outside of the product’s package insert recommendations, the task force recommends replacing the insulin.

Use beyond the manufacturer’s labeled in-use period should be approached cautiously, discussed with the client, documented in the medical record, and avoided when there is any concern for loss of glycemic control, contamination, discoloration, flocculation, or change in insulin consistency.

Section 5: Monitoring Dogs Receiving Insulin

Top 3 Takeaways

1. Interpret all DM monitoring modalities by considering the presence or absence of clinical signs. DM is considered well controlled if the patient does not exhibit PU, PD, or PP and body weight is stable or increasing.
2. Spot-checking BG is unreliable; obtain a blood glucose curve (BGC) either with the patient at home or in the clinic, or place a CGM.
3. Monitoring BG or interstitial glucose (via CGM) is the only way to identify hypoglycemia. If hypoglycemia is documented in an insulin-treated patient, consider reducing the insulin dose. If low values are reported on CGM, it is ideal to confirm the BG concentration using a veterinary-calibrated portable blood glucose monitor (PBGM) if the patient is not showing clinical signs.

The overarching goals of monitoring diabetic dogs are to achieve control of DM clinical signs (PU/PD/PP), maintain an ideal body weight, and avoid complications like hypoglycemia. The goal is not tight BG control, but to maximize the amount of time the BG is below the renal threshold for glucosuria while avoiding hypoglycemia. No monitoring modalities are perfect, and each have strengths and weaknesses. Although normalizing clinical signs and achieving ideal body weight are paramount to other monitoring indicators, when there is evidence of uncontrolled DM, attempt to monitor the patient’s insulin response with a BGC or CGM to best guide therapeutic decision making (Figure 5.1). Without adequate data, the underlying cause of dysregulation cannot be identified in an uncontrolled patient. Incorrectly altering the insulin dose could be detrimental to the dog, such as risking hypoglycemia by increasing an insulin dose when a dog’s DM signs are due to a short duration of insulin action.

Monitoring and Adjusting Intermediate or Long-Acting Bolus Insulin Treatment in Dogs: NPH, Lente, or PZI

Monitoring on the Initial Day of Treatment

- ◆ Initiate insulin therapy.
- ◆ Consider measuring fructosamine.
- ◆ If there is clinical concern for possible hypoglycemia or a need to more rapidly determine an appropriate dose, consider placing a CGM as insulin is started to allow dose titration every 3–7 days until glycemic control is attained.
- ◆ If glucose is <100 mg/dL at any time during initial BG monitoring or on CGM following the first insulin dose, consider:
 - Decreasing the insulin dose by 25–50% in dogs where the BG was obtained using a veterinary-calibrated PBGM.
 - Closely monitoring for signs of hypoglycemia if using a CGM, or confirming low readings with a PBGM.
 - A patient's BG response after starting an intermediate insulin can be highly variable initially, often requiring at least 3–5 days before a stable or consistent response is appreciated.
- ◆ The insulin dose should not be increased within the first 24 hr of starting treatment no matter how high BG may be.

Monitoring Until Control Is Attained

- ◆ Perform a BGC every 5–14 days until an acceptable dose is found or use a CGM to titrate the dose every 3–7 days.
- ◆ Review the client log, which should include any changes to DM clinical signs.
- ◆ Perform a physical examination, including body weight, at least every 2 wk until the appropriate insulin dose is determined.
- ◆ A fructosamine measurement can also be considered at 2 wk intervals.

Ongoing Monitoring After Control Is Attained

- ◆ Well-controlled diabetics should be evaluated every 3–6 months.
- ◆ Review the client log, which should include any changes to DM clinical signs.
- ◆ Perform a physical examination, including body weight.
- ◆ A BGC can be done or a CGM placed at each recheck; however, these assessments are best used to evaluate dogs with clinical signs suggesting loss of glycemic control or hypoglycemia.
- ◆ Semiannually, perform full systemic diagnostics including chemistry panel, CBC, urinalysis, triglycerides, and BP. A fructosamine measurement can be considered. Urine culture is only recommended if there are clinical signs of a urinary tract infection.
- ◆ Any time an insulin dose is changed, perform a BGC in 5–14 days or monitor with CGM.
- ◆ Using “spot checks” or isolated BG values by themselves is not recommended as a sole reason to increase an insulin dose but can sometimes be used to decrease the dose (if hypoglycemia is verified).

(Continued on next page)

FIGURE 5.1

Monitoring and Adjusting Intermediate or Long-Acting Bolus Insulin Treatment in Dogs: NPH, Lente, or PZI

Insulin Adjustments if the Nadir Is <60 mg/dL (see also Figure 11.1)

- ◆ Remember that CGM readings do not always accurately reflect the patient's true BG; therefore, low CGM readings in a patient without clinical signs of hypoglycemia should be confirmed using a PBGM before adjusting therapy.
- ◆ If prolonged periods of hypoglycemia (i.e., 1–2 hr) are confirmed in a patient that is not showing clinical signs, offer the patient a small meal. Consider reducing the insulin dose by 10–25% at the next scheduled injection time.
- ◆ If clinical signs of hypoglycemia are present, treat as necessary (i.e., offer a small meal or provide a sugary solution and pursue further veterinary assessment). Withhold additional insulin until the hypoglycemia has resolved and BG is >250 mg/dL. When re-starting insulin, consider reducing the insulin dose by 25–50%.
- ◆ Obtain a BGC after the next dose to identify whether hypoglycemia recurs. If hypoglycemia recurs with the lower dose, continue to decrease the dose and obtain a BGC until hypoglycemia is resolved.
- ◆ Place a CGM or obtain a BGC in 5–14 days.

Insulin Adjustments if the Nadir Is >150 mg/dL

- ◆ If clinical signs of DM are present:
 - Increase insulin dose depending on patient body size and degree of hyperglycemia, commonly done in increments of:
 - 0.5-1 U/dose in small to medium-sized dogs.
 - 1-2 U/dose in medium to large breed dogs.
 - Place a CGM or recheck a BGC in 5–14 days.
- ◆ If clinical signs of DM are not present:
 - If weight is stable, leave the dose unchanged and recheck in 1–3 mo.
 - If weight is decreasing unintentionally, consider additional testing (such as fructosamine) or a trial dose increase. Place CGM or recheck a BGC in 5–14 days.
- ◆ Consider whether a condition causing insulin resistance is present when:
 - Insulin dose is >1.5 U/kg/dose with no response or fails to bring BG below 250 mg/dL.

Insulin Adjustments if the Nadir Is 60–150 mg/dL

- ◆ If clinical signs are controlled, no adjustment is needed.
- ◆ If clinical signs are not controlled, consider the following possibilities:
 - BGC is not reflective of overall control due to high inter-day glycemic variability. Consider placing a CGM.
 - The duration of insulin action is too short. If insulin is given once daily, consider *q* 12 hr therapy. If given *q* 12 hr, consider changing the insulin type.
 - The duration of insulin action is too long. If BG is still decreasing at the end of the insulin dosing interval, a subsequent dose may cause hypoglycemia. If insulin is given *q* 12 hr, consider *q* 24 hr therapy or giving a lower insulin dose in the evening. It would also be acceptable to try changing the insulin type.
 - The clinical signs are caused by another disease (see Section 7, Troubleshooting in Diabetic Dogs).
- ◆ When using basal insulins, nadirs around 70–80 mg/dL are often targeted for good control and may not warrant insulin dose adjustment.

(Continued on next page)

FIGURE 5.1, CONTINUED*Monitoring and Adjusting Intermediate or Long-Acting Bolus Insulin Treatment in Dogs: NPH, Lente, or PZI*

Ongoing Home Monitoring

- ◆ Log food and water daily. Note any abnormal events like vomiting or lethargy.
- ◆ Log insulin doses daily.
- ◆ Note any signs suggestive of hypoglycemia. Reassess if persistent or recurrent.
- ◆ Consider periodically (once weekly) testing urine for negative glucose results in patients with tight BG control.
- ◆ Consider weekly urine glucose and ketone monitoring in patients with poorly controlled DM. If ketones are present, patient should be reassessed.

BG, blood glucose; BGC, blood glucose curve; BP, blood pressure; CBC, complete blood count; CGM, continuous glucose monitor; DM, diabetes mellitus; NPH, neutral protamine Hagedorn; PBGM, portable blood glucose monitor; PZI, protamine zinc insulin; U, unit

FIGURE 5.1, CONTINUED

Monitoring and Adjusting Intermediate or Long-Acting Bolus Insulin Treatment in Dogs: NPH, Lente, or PZI

Section 6: Dietary Management in Diabetic Dogs

Top 3 Takeaways

1. Dietary therapy helps achieve or maintain an ideal body weight and reduces postprandial hyperglycemia.
2. Canned diets offer benefits over dry food.
3. Differentiate intentional weight loss from unintentional weight loss associated with poor DM control.

In diabetic dogs, dietary management complements insulin therapy. The goals of dietary therapy are to achieve or maintain an ideal body weight and assist with reducing postprandial hyperglycemia. Canned diets offer benefits over dry: fewer simple carbohydrates, higher water content, and less caloric density.¹⁴ Diets that contain increased soluble and insoluble fiber can reduce postprandial hyperglycemia and assist with active weight loss goals.¹⁴ If diabetic dogs are fed standard fiber content diets, consider supplementing them with canned pumpkin, green beans, or commercial fiber supplements containing psyllium or wheat dextrin.

Active weight loss should not exceed 2% of body weight per week. Pay attention to differentiating active, intentional weight loss efforts in obese patients from unintentional weight loss associated with lack of DM control. Also consider regular low-intensity exercise as an adjunct to diet in weight loss plans to help reduce postprandial hyperglycemia.

In underweight diabetic dogs, the principal goal of dietary therapy is to normalize body weight, increase muscle mass, and stabilize metabolism. Feed underweight dogs a high-quality maintenance diet

or a diabetic diet that contains soluble and insoluble fiber but is not designed for weight loss. Underweight diabetic dogs should have a selected diet that will encourage weight gain, and high fiber diets are often lower in calories and fat content, which may work against this goal. It can be acceptable to feed a diet with a moderate carbohydrate content that is higher in calories and fat, regardless of dietary fiber content, as fiber can be supplemented as needed.

Dogs with DM can do well on any diet that is complete and balanced, fed at consistent times and in appropriate portions, and has palatability that supports a consistent appetite.

For dietary management of diabetic dogs:

- Calculate the daily caloric requirements based on lean body mass.
- Include the kilocalorie (kcal) contribution of any daily treats or snacks when calculating a patient's total daily caloric intake.
- Measure body weight (using the same scale) and assess body condition score (BCS) and muscle condition score (MCS) (using the same scoring systems) at least once or twice monthly, and adjust dietary intake to maintain optimal weight.
- Aim for 1–2% weight reduction/wk in obese dogs.

Adjust dietary recommendations if concurrent diseases are present (e.g., chronic kidney disease, pancreatitis, malabsorptive intestinal disease). Feeding a diet that is not ideal for diabetic management is acceptable when patient survival time would be negatively impacted without specific dietary intervention for a comorbid condition.

See the *AAHA Nutrition and Weight Management Guidelines* at aaha.org for more information about BCS, MCS, and calculating daily caloric requirements.

Section 7: Troubleshooting in Diabetic Dogs

Top 3 Takeaways

1. Uncontrolled diabetic dogs have persistent clinical signs. Occasional periods of hyperglycemia in clinically well-controlled dogs are acceptable.
2. Ensure that the proper insulin dose is administered subcutaneously with the correct syringe.
3. Consider that dogs who are not responding to insulin may have a concurrent disease causing insulin resistance or may be exhibiting individual variation in response to a specific insulin.

Troubleshooting and Follow-Up Action

The primary goal of treating canine DM is to control clinical signs. Tight glycemic control is rarely possible using twice-daily intermediate-acting insulin. Occasional brief periods of significant hyperglycemia (>300 mg/dL) in patients with good clinical control do not require additional workup.

An uncontrolled diabetic dog has persistent clinical signs (e.g., PU/PD/PP, weight loss). Lack of control may be due to client noncompliance, incorrect insulin handling or administration, dietary noncompliance, presence of insulin resistance, and/or individual variation in response to specific insulin types. The following step-wise approach helps identify the cause of lack of control (see also Figure 11.2):

1. Assess insulin handling and administration.
 - a. Confirm proper insulin storage. Observe the client's insulin handling, including how they are mixing the insulin. Observe the client administering an insulin dose, including the type of syringe used and dose given.
 - b. Assess insulin being used and replace it if it is out of date or if the insulin is flocculent, discolored, or exhibits consistency changes.
2. Review diet and caloric requirements, especially if unintentional weight loss continues despite control of other clinical signs. It is essential for clients to be honest about food additives/toppers and treats offered regularly at home.
3. If not already done, use a CGM (ideally) or a PBGM-based BGC to rule out hypoglycemia (BG <60 mg/dL).⁴ If hypoglycemia is confirmed, decrease the insulin dose.
 - a. Before adjusting the insulin dose, occasional or sporadic nonclinical low glucose readings on a CGM should be confirmed with a PBGM. Low CGM readings can be inaccurate compared with the patient's actual BG, with some patients not truly being hypoglycemic.¹⁵
4. Consider insulin resistance (See Box, Conditions that Contribute to Insulin Resistance) when intermediate-acting or long-acting insulin doses approach or exceed 1.5 U/kg/injection and BG remains greater than 250 mg/dL.

Conditions That Contribute to Insulin Resistance in Dogs

- Obesity
- Unregulated hypothyroidism
- Dental disease
- Infection (e.g., urinary tract infection, pyoderma)
- Hypertriglyceridemia
- Hypercortisolism
- Renal disease
- Pancreatitis
- Pregnancy/diestrus (see Diestrus Diabetes, Section 2, Recognizing and Diagnosing DM)
- Neoplasia
- Acromegaly (mammary tumor-associated)
- Hyperglucagonemia
- Pheochromocytoma

- a. Rule out administration of medications that could cause IR, such as glucocorticoids and progestins. Specifically ask clients about steroid-containing creams, eye or ear drops, and progestins that may be transferred from a family member, such as topical hormone-replacement therapies.
 1. If a short course of the medication is expected, after which it can be discontinued, reassess the patient 2 wk after the medication has been stopped. For example, if the patient receives a short course of steroid eye drops before or after cataract surgery, the insulin dose does not usually need to be changed despite a short duration of increased BG or mild clinical signs.
 2. If the medication cannot be discontinued or the patient transitioned to an alternative medication within 2 wk, the insulin dose may need to be increased.
 3. Consultation with or referral to a specialist may help, particularly if the patient has concurrent immune-mediated disease that is being managed with glucocorticoids.
- b. Rule out concurrent disease.
 1. Repeat a physical examination, including an oral examination to evaluate for dental disease.
 2. Perform baseline laboratory testing (CBC, serum biochemistry with electrolytes and triglycerides, and urinalysis [with culture if indicated]) if not recently completed.
 3. Perform ovariectomy/ovariohysterectomy in intact, diabetic female dogs. Anesthesia is not contraindicated in otherwise healthy, stable, nonketoacidotic diabetic patients. See the Diabetes Management Resource Center at aaha.org for sample protocols for managing diabetic patients under anesthesia.
 4. Consider second-level diagnostics, such as abdominal and thoracic radiographs, abdominal ultrasonography, pancreatic lipase immunoreactivity (spec cPLI), trypsin-like immunoreactivity, B12/folate measurement, and SDMA for IRIS staging of chronic kidney disease. These diagnostics, in conjunction with baseline diagnostics, will help identify many causes of IR.

- a. Acute or acute-on-chronic pancreatitis can destabilize a previously controlled diabetic patient and make it difficult to re-establish consistent glycemic control. Pancreatitis diagnosis can be challenging, so it is critical to evaluate clinical signs in conjunction with examination findings, clinicopathologic abnormalities, cPLI, and abdominal ultrasonography. Dogs with chronic pancreatitis may have insulin requirements that vary over time because of waxing and waning IR associated with flare-ups. If insulin doses are increased, hypoglycemia can occur when IR resolves as pancreatic inflammation resolves. Thus, conservative dose adjustments that are closely monitored, especially for hypoglycemia, are ideal.
5. Consider specific diagnostics for hypercortisolism (HC; Cushing syndrome), especially in dogs whose signs include pathologic panting or significant dermatologic/haircoat abnormalities. HC can cause insulin resistance and persistent PU/PD in diabetic dogs who otherwise appear to be well regulated. ALP is often increased in diabetic dogs, so increased ALP alone does not suggest HC. However, sudden progressive or severe elevations of ALP may suggest HC onset. Generally, endocrine testing for HC should not be performed before diabetic regulation has been attempted for at least 2–4 wk, because unregulated diabetes can lead to false-positive cortisol results. The urine cortisol-to-creatinine ratio is often positive in diabetic dogs and is therefore not recommended as a screening test for HC in this population. The adrenocorticotrophic hormone (ACTH) stimulation test is advantageous for screening uncontrolled diabetic patients as it is more specific (fewer false positives) but less sensitive (more false negatives) than the low-dose dexamethasone suppression test.¹⁶ See the *AAHA Selected Endocrinopathies in Dogs and Cats Guidelines* at aaha.org for more information.
6. Consider specific diagnostics for hypothyroidism, especially in dogs whose signs include significant dermatologic/haircoat abnormalities. Hypothyroidism can cause IR, but diagnosis in diabetic dogs can be challenging. Many euthyroid diabetic dogs will have a decreased TT4 concentration due to euthyroid sick syndrome, so a decreased TT4 alone cannot confirm hypothyroidism. In most cases, delay testing for hypothyroidism for 4–6 wk after DM diagnosis and treatment initiation to decrease the effects of euthyroid sick syndrome. In a diabetic patient, evaluate TT4, free T4 by equilibrium dialysis, and thyroid-stimulating hormone concurrently.¹⁷
7. If a condition causing IR is identified, focus first on controlling that underlying condition before adjusting DM therapies to achieve regulation.
8. If the patient has never been regulated, has no cause of IR identified, and has been given only one insulin type, consider switching to a different insulin formulation. This may be attempted before performing second-level diagnostics (item 4 above), based on the veterinarian's preference. Some veterinarians recommend changing the insulin type when a dog does not show any response to >1.5 U/kg/dose of NPH or porcine lente insulin. See Section 9, Glucose Monitoring, for information on transitioning to a new insulin.
9. Finally, consult with a specialist if the patient cannot be regulated (See the *AAHA Referral Guidelines* at aaha.org).

Section 8: Client Education

Top 3 Takeaways

1. Clients must understand that managing a diabetic dog requires both time and financial commitment. However, over time the disease will be controlled in most dogs and proper treatment and monitoring often results in a good quality of life.
2. Clients must know how to identify signs of hypoglycemia, how to prevent it, and what to do when it is suspected or observed.
3. Clients need clear instructions on dietary recommendations and timing of insulin administration in relation to meals (depending on the insulin being used). Also discuss the importance of nutrient content, amount, and glycemic impact of any treats they plan to routinely give to their dog.

The goal of client education is to give the pet caregiver a realistic idea of the commitment involved in managing their pet's DM, along with positive encouragement that successful disease management is possible but takes time. Clients need access to veterinary team members trained to readily answer questions and/or troubleshoot common problems. Provide written material for reference when the veterinary team is unavailable. Feature content (e.g., online, hand-outs) that answers frequently asked questions, describes what to watch for at home, and explains how to respond to changes in their dog's condition. Direct clients to reliable online information, including the Diabetes Management Guidelines Resource Center at aaha.org/diabetes-management-dogs. Stress the importance of appropriate nutrition and weight management.

Key client education points:

Insulin

1. Have the client feed the dog and administer a full insulin dose only after the dog has consumed at least 50% of the meal.
 - a. Give one-half of the normal insulin dose if the dog is intentionally fasted or eats less than 50% of their offered meal.
 - b. Contact the veterinarian if the dog does not eat at least 50% of two consecutive meals.
2. Consistent timing and amount of food, treats, and exercise are important. A walk following meals may decrease postprandial hyperglycemia.¹⁸
3. Instruct clients on how to properly handle and mix the specific type of insulin prescribed. Proper mixing of an insulin suspension (non-clear solution) is essential before pulling up a dose.
 - a. Vetsulin should be shaken until a homogeneous, uniformly milky suspension is obtained.¹⁹
 - b. When using other insulin vials (glargine U-100, PZI, NPH), and insulin degludec, roll but do not shake the vial.
4. 0.3- and 0.5-mL insulin syringes or insulin pens are best to facilitate accurate dosing, especially in dogs receiving <5 U per dose.²⁰
 - a. Insulin degludec U-200 and glargine U-300 must be administered using the provided pen. U-200 or U-300 insulin syringes are not available.

- b. See manufacturer recommendations specific to the pen used for information on handling, methods to mix insulin prior to administration, and injection instructions.

Recognizing hypoglycemia

Instruct clients on how to recognize the signs of hypoglycemia (e.g., lethargy, sleepiness, strange behavior, abnormal gait, weakness, tremors, collapse, and seizures) and provide instructions on what to do if they observe these signs in their pet.

1. If the dog is conscious and only shows mild signs, feed a small, high-carbohydrate meal (e.g., rice, bread, pasta, regular diet), then take to a veterinarian.
2. If the dog is showing moderate-to-severe clinical signs, is poorly responsive, or has tremors, soak 3–5 cotton-tipped swabs in 1–2 teaspoons of corn syrup and place or rub in the mouth between the cheek and gums. Clients should not stick their fingers into a hypoglycemic patient's mouth. Some experts use a corn syrup dose of up to 0.25 mL/kg, which can be administered orally using a syringe. Advise the client of the risk of aspiration in an obtunded animal. Take the pet to a veterinarian.
3. If the dog is having seizures, follow steps above and advise clients to seek immediate veterinary medical attention.
4. Empower clients to decrease or skip an insulin dose if clinical hypoglycemia is noted and/or confirmed on a veterinary-calibrated PBGM but emphasize that they should never increase the dose or frequency of insulin administration without clear instructions from their veterinarian.

General Monitoring Considerations

Monitoring diabetic dogs can be challenging. The algorithm in Figure 9.1 (first published in the 2018 AAHA *Diabetes Management Guidelines for Dogs and Cats* and updated for these guidelines)¹ provides a quick reference for three types of DM patients who are receiving insulin—newly diagnosed, previously diagnosed, and previously diagnosed but currently unregulated. Monitoring includes serial assessment of clinical signs and body weight in addition to performing BGCs, CGM, or fructosamine measurements as needed. Occasionally, monitoring urinary glucose may be warranted, such as screening for hypoglycemia, but it is not a recommended routine monitoring tool. Results from different monitoring approaches may conflict. In a review of 53 cases of canine DM, all glycemic assessments and glycated protein concentrations were consistent with good glycemic control in only 60% of dogs classified as being clinically well-controlled.²¹ No single glycemic assessment tool evaluated could consistently differentiate well and poorly controlled DM, highlighting the absolute necessity of interpreting any glycemic data obtained in relation to the presence or absence of clinical signs.²¹

Section 9: Glucose Monitoring

Top 3 Takeaways

1. Glucose monitoring includes BGCs and CGM, which assesses a patient's response to insulin and can identify subclinical hypoglycemia (thus the insulin dose can be decreased before clinical signs occur).
2. Anytime clinical signs recur following initial DM control, glucose monitoring is required to identify the root cause (e.g., low dose, short duration of action) and direct how to safely adjust (increase or decrease) an insulin dose.
3. Situations when glucose monitoring should be performed include:
 - a. At 5–14 days after starting insulin or following any dose change;
 - b. Any time clinical signs recur in a previously controlled patient; and
 - c. When hypoglycemia is suspected.

Blood Glucose Curves

A BGC is typically performed by measuring BG using venous or capillary blood before (baseline, time 0) and then every 2 hr after feeding a normal-sized meal and administering the full insulin dose, for up to 10–12 hr.

A BGC should identify the lowest BG (i.e., the nadir) obtained, the duration of insulin action observed, and the range of BG fluctuation during the dosing interval. The ideal nadir is a BG of 80–150 mg/dL. As most insulin types are administered twice daily, an ideal duration of insulin effect is as close to 12 hr as possible. The BG readings should ideally range between 80 and 300 mg/dL during the BGC as long as the BG remains <200 mg/dL for most of the dosing interval assessed. Identifying these aspects of a BGC allows logical changes to be made to the patient's dosing regimen as needed.

The first aim in regulating a diabetic is to achieve an acceptable nadir. If an acceptable nadir is not achieved, consider increasing the insulin dose. An acceptable nadir with good clinical control may not be obtained if the insulin has a short duration of action. Avoid prolonged periods (i.e., 1–2 hr) of subclinical hypoglycemia (BG <60 mg/dL) and consider reducing the insulin dose if noted or any time a patient exhibits clinical signs of hypoglycemia. Once an acceptable nadir is achieved, duration of action—roughly defined as the amount of time BG is controlled (ideally 80–250 mg/dL)—can be determined. If either insufficient or excessive duration of insulin action is contributing to uncontrolled BG, change the frequency of insulin administration or, more commonly, transition to a new insulin type (See Box, Transitioning to a New Insulin).

The Somogyi phenomenon, also called hypoglycemia-induced hyperglycemia, refers to hypoglycemia followed by marked hyperglycemia. It can result from a physiological response when an insulin dose causes BG to be <60 mg/dL or when BG concentrations decrease rapidly over a short period of time. In either case, counter-regulatory hormones (e.g., cortisol, epinephrine, and glucagon) are released to increase BG. Rebound hyperglycemia usually occurs rapidly and can

MONITORING GLUCOSE IN DIABETIC DOGS (Not on Basal Insulin)

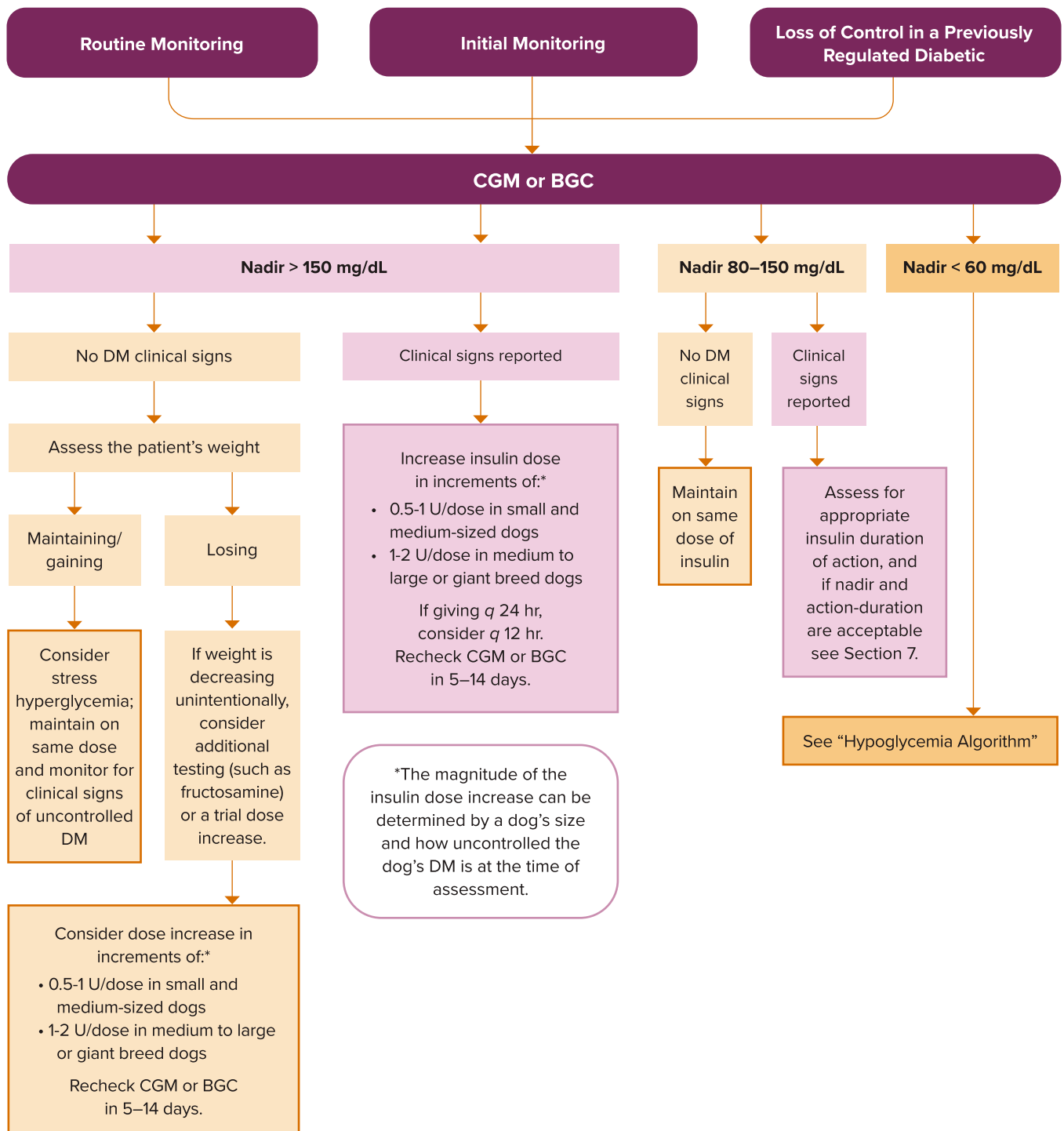


FIGURE 9.1

Monitoring Glucose in Diabetic Dogs (Not on Basal Insulin)

BGC, blood glucose curve; CGM, continuous glucose monitor; DM, diabetes mellitus; U, unit

Transitioning to a New Insulin

The simplest approach to transition a patient to a new insulin type is to initiate the new insulin at the recommended routine starting dose and frequency (Table 4.1). Review time-action details of the new insulin, as changes to dose or frequency of administration may be warranted. For example, transitioning from NPH to PZI may warrant dose and frequency reduction. Other transitions are more complicated. Protocols for transitioning from an intermediate-acting insulin (e.g., NPH or lente insulin) to insulin degludec or glargine U-300 have been described, with initial doses calculated from the dog's previous insulin dose.* Always monitor BG closely following initiation of a new insulin type and consider consultation with a specialist if problems achieving glycemic regulation are encountered.

*See Mott JA, Gal A, Tardo AM, et al. Insulin degludec 100 U/mL for treatment of spontaneous diabetes mellitus in dogs. *J Vet Intern Med* 2025;39(1):e17303 and Tardo AM, Fleeman LM, Fracassi F, et al. A dose titration protocol for once-daily insulin glargine 300 U/mL for the treatment of diabetes mellitus in dogs. *J Vet Intern Med* 2024;38(4):2120–8.

be followed by a period of sustained insulin resistance. If a Somogyi phenomenon is observed, decrease the insulin dose. Once the nadir is >60 mg/dL, counter-regulatory hormones will no longer interfere, and the true duration of effect will become apparent.²²

Glucose curves are not perfect, so always interpret them in light of clinical signs and body weight changes. Responses seen on a BGC vary from day to day and can be affected by deviation from the patient's normal routine.^{23,24} Stress hyperglycemia can occur in dogs but the false increase in BG is generally mild. See the Diabetes Management Guidelines Resource Center at aaha.org/diabetes-management-dogs for examples of interpreting various glucose curves.

IN-HOSPITAL BLOOD GLUCOSE CURVES

A PBGM calibrated for use in veterinary patients (e.g., AlphaTrak 3) may be the most practical and accurate method to serially monitor the BG.^{25,26,27} Although glucometers designed for use in people are readily accessible to pet caregivers, the task force does not recommend their use owing to lack of veterinary-specific calibration, which can lead to inaccurate result reporting.

If the dog is not expected to eat while in hospital, the client can feed and give insulin at home before bringing the dog to the clinic. The BGC duration in dogs receiving intermediate-acting insulin (e.g., NPH, lente) is often up to 12 hr, whereas dogs receiving basal insulin (e.g., degludec, glargine U-300) are best monitored with a CGM to allow >24 hr assessment. Maintaining the patient's routine daily schedule (including mealtimes, insulin administration, treats, and exercise) during the BGC may increase the accuracy of obtained results.

AT-HOME BLOOD GLUCOSE CURVES

Obtaining a BGC at home may be helpful. The steps outlined above for in-hospital BGCs apply to at-home BGCs, and capillary blood is most suitable.²⁸ Common blood collection sites are the lateral ear, buccal mucosa, non-weight-bearing or accessory foot pads, or elbow callus. If using lancing devices designed for pricking human fingertips, choose one with a variable needle depth often ranging from 1.0–5.0 mm. Small dogs may require a 1.0–1.8 mm lancing depth when sampling from the ear, buccal mucosa, or paw pad, while medium to large breed dogs may require at least a 1.5–2.3 mm lancing depth, especially when sampling from thick or cartilaginous ears or paw pads. A hypodermic needle can also be used, especially if blood is collected from the marginal ear vein.²⁹ To ensure result accuracy, the lancing device must produce a droplet of blood sufficient for BG determination without requiring excessive manipulation of the pricked site, such as repeatedly squeezing that area to increase droplet size.

Not all clients are suited to the task of obtaining a home BGC. Clients frequently encounter problems such as needing more than one puncture to obtain a sufficient sample, inability to restrain a pet without help, and the pet's resistance to blood sampling.²⁸ Over time, this can negatively impact the human-animal bond.

Even home BGCs can vary from day to day and must always be interpreted considering clinical signs.³⁰ Refer to the Diabetes Management Guidelines Resource Center for more detailed information and resources on at-home monitoring using BGCs.

CONTINUOUS GLUCOSE MONITORING SYSTEMS

Standard BGCs have limitations, as they provide only a snapshot in time, insulin responses can vary considerably from day to day, and hypoglycemia during the night may go undetected. CGM systems record interstitial glucose readings continuously and over multiple days, which can help identify problems and allow more comprehensive assessments of BG control over several days. CGM sensors are easily placed—even in less cooperative patients—and are generally as affordable as a BGC. A CGM also provides more physiological information for interpretation as glycemic activity is monitored in the patient's home environment and captures all aspects of their daily routine, such as meals, treats, and exercise.

The FreeStyle Libre (FSL) is a CGM system that reports an average interstitial glucose concentration every 15 min for up to 15 days. Successful use of the FSL has been reported in veterinary patients.³¹ A 2019 study also reported its use in hospitalized dogs with diabetic ketoacidosis.³² Although not calibrated for veterinary patients, CGMs are typically used to follow glycemic trends over time and appear to be relatively accurate.³³ However, there can be discrepancies between glucose results reported by the FSL and AlphaTrak in some patients, and very high or low glucose readings on FSL often warrant further confirmation.³³ It is especially important to train clients to confirm low FSL readings with a veterinary-calibrated PBGM before altering therapy, particularly when the dog is not exhibiting clinical signs of hypoglycemia. A “Notes” feature in the FSL system allows clients to document timing of meals, insulin, exercise, or unexpected events (such as vomiting) to aid the veterinarian’s assessment.

Inform clients that the sensors do not always function or remain attached to the patient for the full 15 days, but the information gained from even a few days of reporting is usually more helpful than a standard BGC. Reinforcing the FSL sensor adhesive pad with adhesive wipes or tissue glue and/or using a commercial sensor cover, T-shirt, or stockinette may help prevent inadvertent sensor removal. Tissue glue use is anecdotally associated with causing more skin irritation, therefore apply conservatively and consider using an adhesive remover, such as acetone, when removing sensors.

Clients with real-time access to their pet’s glycemic information can become noncompliant or overly dependent on needing to know their pet’s glycemic status. For example, they may feel compelled to alter prescribed therapies based on glucose readings independent of veterinary guidance or seek frequent veterinary consultation by calling with every slightly abnormal reading. Additionally, clients can become preoccupied with the need to always know their pet’s glucose readings. These clients may want an FSL placed on the patient continuously, even when it is not medically necessary. Educating clients and setting proper expectations can help mitigate these concerns. The task force recommends clinicians only use a CGM when medically necessary for their assessment of a patient’s BG control. Placing a CGM simply to satisfy a client’s desire to know their pet’s glycemic results often increases the likelihood of problematic behaviors.

Section 10: Other Methods for Monitoring Glycemic Status

Top 3 Takeaways

1. Routine urine glucose measurement is not typically recommended because of low accuracy for determining glycemic control.
2. Fructosamine trends tend to be more useful than isolated values.
3. Control of clinical signs is an essential goal of diabetic treatment, and the presence or absence of clinical signs is often a more useful marker of clinical DM control than any glycemic data obtained.

HOME MONITORING OF CLINICAL SIGNS

Client perception of clinical signs in combination with serial monitoring of body weight is crucial to effective DM monitoring. The presence or absence of clinical signs is often a more accurate marker of DM control than any glycemic data obtained, suggesting that consideration of clinical sign control is an essential aspect of each diabetic evaluation.²¹ Encourage clients to keep a daily log documenting trends in their pet’s appetite, thirst, urinary habits (e.g., increased, normal, decreased), feeding (e.g., diet type, amount) and insulin dose administered, which can be brought in for review during each in-clinic recheck examination. When the patient has no clinical signs and the body weight is steady or increasing, DM is likely well controlled. Because clinical signs are subjective or possibly difficult to fully appreciate (such as water intake), the use of automated devices monitoring food or water intake may improve objectivity as opposed to solely relying on client observations.

GLYCOSYLATED PROTEINS

The glycosylated proteins include fructosamine and glycosylated hemoglobin (A1C). Fructosamine, the glycosylated protein commonly measured in veterinary medicine, is formed by nonenzymatic, irreversible binding of glucose to serum proteins, mainly albumin.³⁴ The rate of formation is proportional to the average BG level, so the higher the mean BG concentration is over time, the greater the fructosamine concentration should be. Because fructosamine concentration is also affected by the half-life of albumin, it reflects glycemic control over the previous 7–10 days.³⁵ Unfortunately, well-controlled diabetics can have elevated fructosamine concentrations. Conversely, pets with uncontrolled diabetes can have normal levels.³⁶ For these reasons, fructosamine trends are often more useful than isolated values. Declining fructosamine values indicate the average BG concentration is decreasing over time, whereas increasing values indicate the opposite. A fructosamine concentration below the reference range is highly suggestive of chronic hypoglycemia, which should be verified with a BGC or CGM assessment.

Because fructosamine development involves binding with proteins, conditions that increase the rate of protein turnover (e.g., hyperthyroidism) or reduce serum protein levels (e.g., protein-losing enteropathy) can falsely decrease the fructosamine concentration.

Hemoglobin A1C (i.e., glycated hemoglobin) is commonly used to monitor diabetes in humans and has been described in veterinary medicine as a marker of mean BG over the preceding 2–3 mo.^{37,38,39} A dried blood spot mail-in test card is commercially available for use in dogs but has not yet been validated in diabetic dogs.

URINE GLUCOSE MEASUREMENTS

Urinary dipsticks have a relatively low accuracy in dogs, often underestimating the urine glucose concentration (UG).^{40,41} The UG concentration is only a reflection of the average BG over the time

interval since the bladder was last voided. Therefore, routine UG measurement is not typically recommended.

Patients that have consistently negative UG should be screened for hypoglycemia. This finding may indicate tight BG regulation or that insulin dosages are excessive. However, a negative UG reading only means that BG was below the renal threshold (i.e., BG could have been 150 mg/dL or 40 mg/dL). The only way to know is to serially assess the BG.⁴²

Section 11: Algorithms for Managing Hypoglycemia and Troubleshooting in Dogs

The following algorithms were originally published in the 2018 AAHA *Diabetes Management Guidelines* and updated for these guidelines. These algorithms can assist with managing hypoglycemic patients and troubleshooting patients on insulins other than basal insulin.

MANAGING HYPOGLYCEMIA IN DIABETIC DOGS

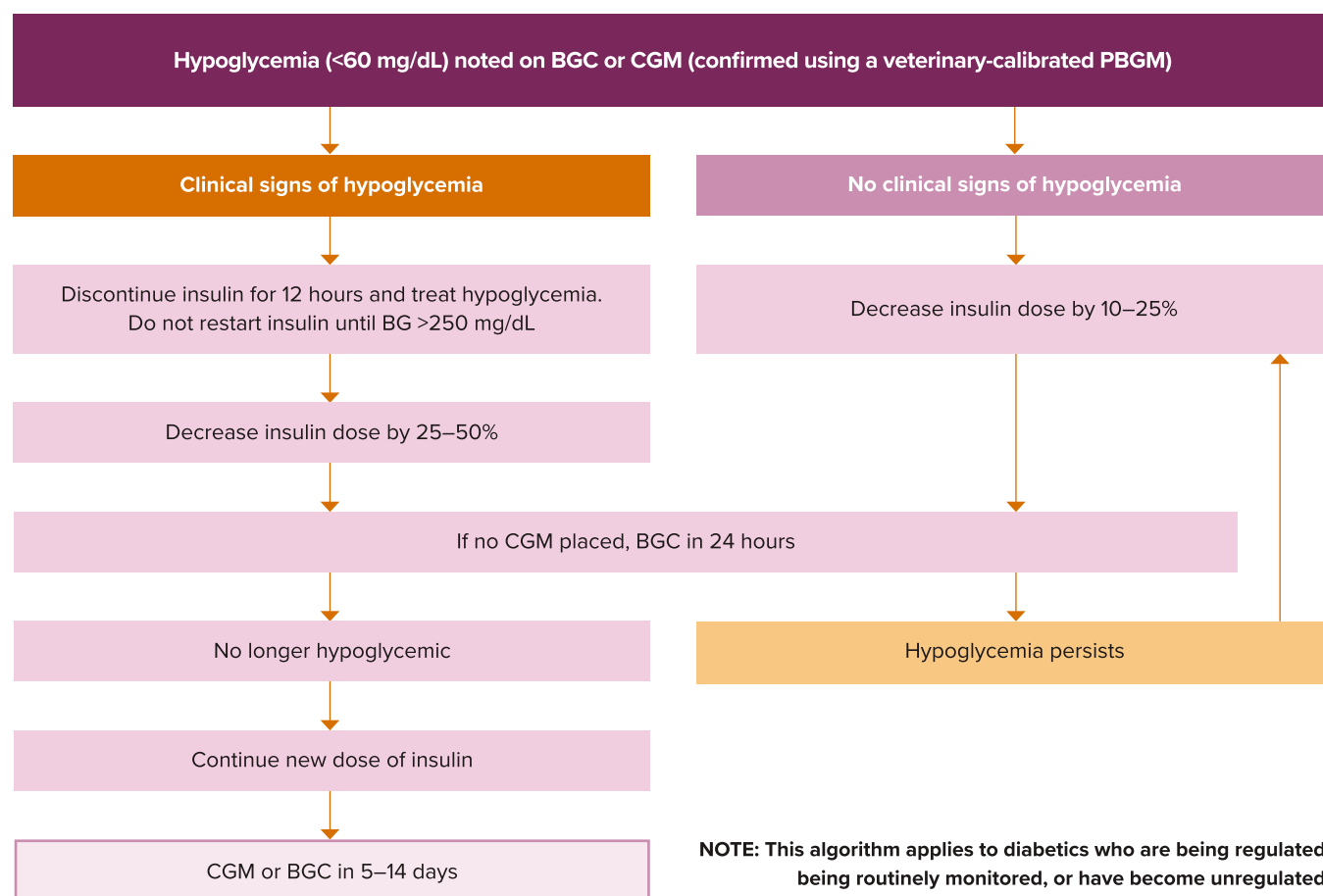


FIGURE 11.1

Managing Hypoglycemia in Diabetic Dogs

BG, blood glucose; BGC, blood glucose curve; CGM, continuous glucose monitor; PBGM, portable blood glucose monitor; U, unit

TROUBLESHOOTING DIABETIC DOGS RECEIVING HIGH DOSES OF INSULIN

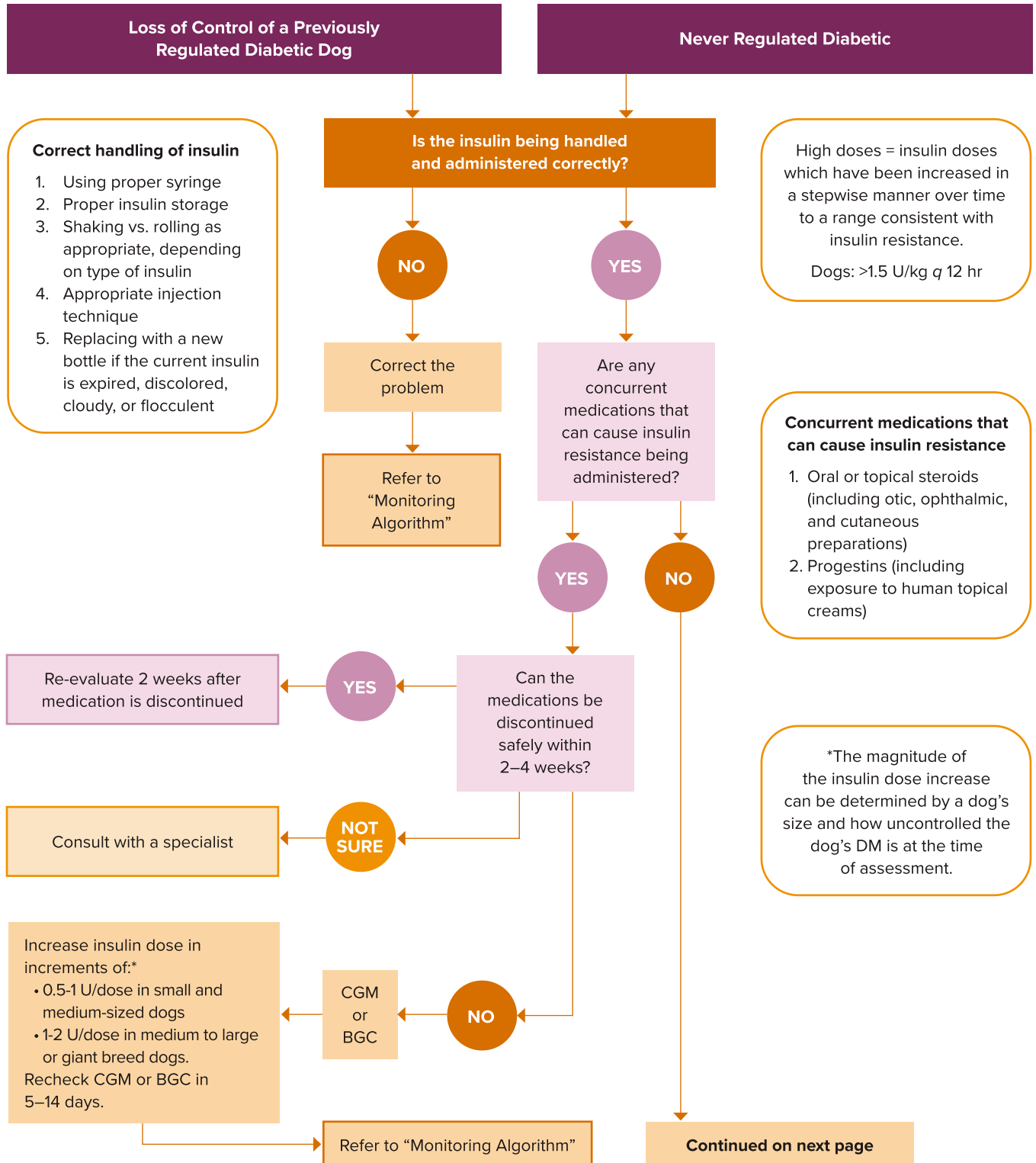


FIGURE 11.2

Troubleshooting Diabetic Dogs Receiving High Doses of Insulin

TROUBLESHOOTING DIABETIC DOGS RECEIVING HIGH DOSES OF INSULIN

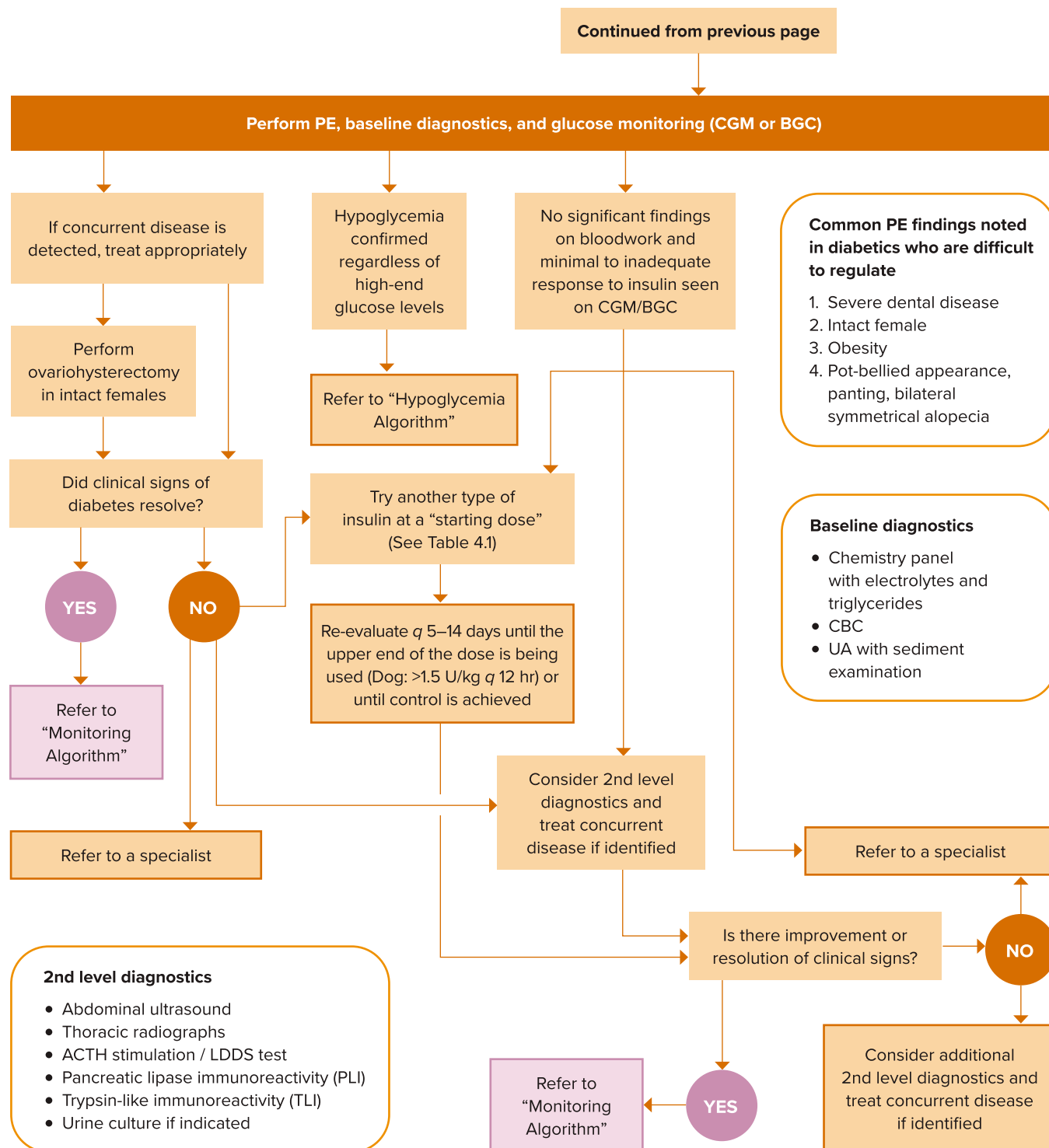


FIGURE 11.2, CONTINUED

Troubleshooting Diabetic Dogs Receiving High Doses of Insulin

BGC, blood glucose curve; CBC, complete blood count; CGM, continuous glucose monitor; PE, physical examination; U, unit; UA, urinalysis

Conclusion

Diabetic dogs can live a good quality of life with few or no clinical signs, facilitated by clients' and veterinary teams' commitment to attentive patient care and monitoring, dietary and schedule adjustments, and diligent follow-up evaluations. A rewarding outcome for diabetic pets starts with realistic but positive and honest conversations between clients and their veterinary teams about expectations relating to treatment, patient monitoring, communication, goals, and the commitments required from those involved in the patient's care. Many dogs who develop diabetes can live comfortably with their condition for the rest of their lives and tolerate insulin therapy well.

Diligent patient observation both at home and via intermittent evaluations in-clinic are essential to gauge therapeutic response, indicate when changes to treatment plans or additional diagnostic tests are needed, and detect or avoid complications associated with the disease or inadequate treatment. Enhancing patients' and clients' quality of life relies on resolving clinical signs through adequate, and not necessarily perfect, glycemic control. ■

REFERENCES

- Behrend E, Holford A, Lathan P, et al. 2018 AAHA diabetes management guidelines for dogs and cats. *J Am Anim Hosp Assoc* 2018;54(1):1–21.
- Davison LJ, Ristic JM, Herrtage ME, et al. Anti-insulin antibodies in dogs with naturally occurring diabetes mellitus. *Vet Immunol Immunopathol* 2003;91(1):53–60.
- Hess RS, Kass PH, Ward CR. Breed distribution of dogs with diabetes mellitus admitted to a tertiary care facility. *J Am Vet Med Assoc* 2000; 216(9):1414–7.
- Niessen SJM, Bjornvad C, Church DB, et al; ESVE/SCE membership. Agreeing Language In Veterinary Endocrinology (ALIVE): diabetes mellitus-a modified Delphi-method-based system to create consensus disease definitions. *Vet J* 2022;289:105910.
- Fall T, Hedhammar Å, Wallberg A, et al. Diabetes mellitus in elkhounds is associated with diestrus and pregnancy. *J Vet Intern Med* 2010;24(6): 1322–8.
- Pöpl ÁG, Lopes JXL, Nogueira TB, da Silva DI, Machado BDS. Progesterone-related diabetes mellitus in the bitch: Current knowledge, the role of pyometra, and relevance in practice. *Animals (Basel)*. 2024;14(6):890.
- Fall T, Johansson Kreuger S, Juberget Å, et al. Gestational diabetes mellitus in 13 dogs. *J Vet Intern Med* 2008;22(6):1296–300.
- Davison LJ. Diabetes mellitus and pancreatitis-cause or effect? *J Small Anim Pract* 2015;56(1):50–9.
- Stojanovic V, Ihle S. Role of beta-hydroxybutyric acid in diabetic ketoacidosis: a review. *Can Vet J* 2011;52(4):426–30.
- Tardo AM, Fleeman LM, Fracassi F, et al. A dose titration protocol for once-daily insulin glargine 300 U/mL for the treatment of diabetes mellitus in dogs. *J Vet Intern Med* 2024;38(4):2120–8.
- Mott J, Gal A, Tardo AM, et al. Insulin degludec 100 U/mL for treatment of spontaneous diabetes mellitus in dogs. *J Vet Intern Med* 2025; 39(1):e17303.
- Miller M, Pires J, Crakes K, et al. Day-to-day variability of porcine lente, insulin glargine 300 U/mL and insulin degludec in diabetic dogs. *J Vet Intern Med* 2021;35(5):2131–9.
- Berg AS, Jacob ME, Mamo LB, et al. Refrigerated multi-dose insulin vials remain sterile through 6 months of use. *J Small Anim Pract* 2023;64(12): 776–80.
- Parker VJ, Hill RC. Nutritional management of cats and dogs with diabetes mellitus. *Vet Clin North Am Small Anim Pract*. 2023;53(3):657–674.
- Tardo AM, Crews C, Mott J, et al. Accuracy of the Freestyle Libre 3 continuous glucose monitoring system in hypo- and euglycemic cats. *J Vet Intern Med* 2025;39(2):e70048.
- Behrend E. Canine hyperadrenocorticism. In: Feldman ED, Nelson RW, Reusch CE, et al, eds. *Canine and Feline Endocrinology*. 4th ed. Elsevier; 2015:403–5.
- Mooney C. Canine hypothyroidism. In: Ettinger SJ, Feldman EC, Cote E, eds. *Textbook of Veterinary Internal Medicine*. 8th ed. Elsevier;2017: 1731–42.
- Mampe JR, Stefanovski D, Hess RS. Aerobic exercise decreases interstitial glucose concentrations up to 2 h after exercise in dogs with insulin-treated diabetes mellitus: a preliminary study. *Front Vet Sci*. 2025;12: 1595952.
- Vetsulin. [package insert.] Merck Animal Health. Available at: www.vetsulin.com/cats/wp-content/uploads/sites/180/2022/03/Vetsulin-Package-Insert.pdf.
- Keith K, Nicholson D, Rogers D. Accuracy and precision of low-dose insulin administration using syringes, pen injectors, and a pump. *Clin Pediatr (Phila)* 2004;43(1):69–74.
- Briggs CE, Nelson RW, Feldman EC, et al. Reliability of history and physical examination findings for assessing control of glycemia in dogs with diabetes mellitus: 53 cases (1995-1998). *J Am Vet Med Assoc* 2000; 217(1):48–53.
- Feldman EC, Nelson RW. Insulin-induced hyperglycemia in diabetic dogs. *J Am Vet Med Assoc*. 1982;180(12):1432–1437.
- Fleeman LM, Rand JS. Evaluation of day-to-day variability of serial blood glucose concentration curves in diabetic dogs. *J Am Vet Med Assoc* 2003;222(3):317–21.
- Ristic JM, Herrtage ME, Walti-Lauger SM, et al. Evaluation of a continuous glucose monitoring system in cats with diabetes mellitus. *J Feline Med Surg* 2005;7(3):153–62.
- Cohen TA, Nelson RW, Kass PH, et al. Evaluation of six portable blood glucose meters for measuring blood glucose concentration in dogs. *J Am Vet Med Assoc* 2009;235(3):276–80.
- AlphaTrak 3 [package insert.] Zoetis. Available at: https://www.zoetis.com/content/_assets/docs/vmips/package-inserts/alphatrak-3-package-insert.pdf. Accessed November 12, 2025.
- Behrend EN, Suchowersky ND, Carlson EA, et al. Comparison between glucose measurements in canine whole blood, serum, and plasma. *J Vet Intern Med* 2017;31:1271–2.
- Casella M, Wess G, Hassig M, et al. Home monitoring of blood glucose concentration by owners of diabetic dogs. *J Small Anim Pract* 2003; 44(7):298–305.
- Van de Maele I, Rogier N, Daminet S. Retrospective study of owners' perception on home monitoring of blood glucose in diabetic dogs and cats. *Can Vet J* 2005;46(8):718–23.
- Alt N, Kley S, Haessig M, et al. Day-to-day variability of blood glucose concentration curves generated at home in cats with diabetes mellitus. *J Am Vet Med Assoc* 2007;230(7):1011–7.
- Corradini S, Pilosio B, Dondi F, et al. Accuracy of a flash glucose monitoring system in diabetic dogs. *J Vet Intern Med* 2016;30(4):983–8.
- Malerba E, Cattani C, Del Baldo F, et al. Accuracy of a flash glucose monitoring system in dogs with diabetic ketoacidosis. *J Vet Intern Med* 2020;34(1):83–91.

33. Berg AS, Crews CD, Adin C, Alfonso-Castro A, Hill SB, Mott J, Gilor C. Assessment of the FreeStyle Libre 2 interstitial glucose monitor in hypo- and euglycemic cats. *J Vet Intern Med* 2023;37(5):1703–9.
34. Miller E. Long-term monitoring of the diabetic dog and cat: clinical signs, serial blood glucose determinations, urine glucose and glycated blood proteins. *Vet Clin North Am Small Anim Pract* 1995;25(3):571–84.
35. Link KR, Rand JS. Changes in blood glucose concentration are associated with relatively rapid changes in circulating fructosamine concentrations in cats. *J Feline Med Surg* 2008;10(6):583–92.
36. Kuzi S, Mazaki-Tovi M, Ahmad WA, Ovadia Y, Aroch I. Clinical utility of serum fructosamine in long-term monitoring of diabetes mellitus in dogs. *Vet Rec.* 2023;192(2):e2236.
37. Loste A, Marca MC. Fructosamine and glycated hemoglobin in the assessment of glycaemic control in dogs. *Vet Res* 2001;32(1):55–62.
38. Kim NY, An J, Jeong JK, et al. Evaluation of a human glycated hemoglobin test in canine diabetes mellitus. *J Vet Diagn Invest* 2019;31(3):408–14.
39. Norris O, Schermerhorn T. Relationship between HbA1c, fructosamine and clinical assessment of glycemic control in dogs. *PLoS One* 2022;17(2):e0264275.
40. Behrend EN, Tapia J, Welles EG, et al. Evaluation of a conventional urine glucose test strip method for detection of glucosuria in dogs and cats [abstract]. *J Vet Intern Med* 2008;22:790a.
41. Aldridge CF, Behrend EN, Smith JR, Welles EG, Lee HP. Accuracy of urine dipsticks and urine glucose:creatinine ratios for assessment of glucosuria. *J Am Vet Med Assoc* 2020;257:391–6.
42. Greco DS, Broussard JD, Peterson ME. Insulin therapy. *Vet Clin North Am Small Anim Pract* 1995;25(3):677–89.