



An In-Depth Look:

HYPOPARATHYROIDISM

Hypoparathyroidism: Treatment*

Alicia K. Henderson, DVM
Orla Mahony, MVB, DACVIM
Tufts University

ABSTRACT:

Hypoparathyroidism is a disorder caused by parathyroid hormone deficiency, resulting in hypocalcemia and hyperphosphatemia. Treatment can be divided into acute, subacute, and maintenance stages. Calcium supplementation and vitamin D therapy are the hallmarks of treatment. Replacement therapy with synthetic parathyroid hormone is an emerging treatment in humans. Frequent monitoring of the serum calcium concentration is vital to avoid hypercalcemia and its serious consequences, including renal failure. With appropriate treatment and diligent monitoring, the disease carries a favorable prognosis with good-quality, long-term survival.

Primarily hypoparathyroidism is an endocrine disorder characterized by parathyroid hormone (PTH) deficiency, resulting in hypocalcemia and hyperphosphatemia. Clinical signs are primarily neuromuscular in origin and can include tetany and seizures secondary to hypocalcemia. Treatment of primary hypoparathyroidism is individualized depending on the severity of clinical signs, the degree of hypocalcemia, the rapidity of decline in serum calcium levels, and trends toward a further decrease or stability in the serum calcium concentration.¹ Treatment measures can be divided into acute, subacute, and maintenance therapy. Medical treatment includes parenteral and oral calcium as well as oral vitamin D analogue supplementation. Unfortunately, no treatment regimen fully compensates for all of the physiologic actions of PTH.¹ The mainstay of therapy, vitamin D metabolite treatment, corrects low intestinal absorption of calcium but does not completely protect the kidneys from hypercalciuria and does not exert a powerful effect on bone in the absence of PTH.¹

ACUTE THERAPY

Aggressive, immediate treatment, including hospitalization and intravenous calcium infusion, is needed in patients with tetany, seizures, or pronounced hypocalcemia with

*A companion article on pathophysiology and diagnosis appears on page 270.

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or without clinical signs. Both calcium chloride and calcium gluconate can be used for rapid intravenous treatment. The percentage of calcium varies widely between the different calcium salts, although there is no difference in the effectiveness in correcting hypocalcemia when the dose is based on elemental calcium. Calcium chloride has the potential advantage of providing more elemental calcium per milliliter of solution; however, it is extremely irritating to tissues when injected outside a vessel.¹ Calcium gluconate as a 10% solution is the calcium salt of choice because it is not caustic when extravasated outside a vein.^{1,2} The recommended dose of elemental calcium is 5 to 15 mg/kg IV slowly over 10 to 30 minutes to effect. This correlates with 0.5 to 1.5 ml/kg of 10% calcium gluconate.^{1,3} This recommended dose should be used only as a guideline, with patient

which oral vitamin D therapy becomes effective.^{1,2,4} Options for parenteral treatment include intermittent intravenous boluses, continuous intravenous infusion, or subcutaneous injections of calcium salts. Multiple intermittent intravenous injections of calcium salts can be given to control clinical signs; however, this method is not widely recommended because it causes wide fluctuations in serum calcium concentrations.¹

Continuous intravenous infusion of calcium is recommended at 60 to 90 mg/kg/day (2.5 to 3.75 mg/kg/hr) of elemental calcium.¹ Ten percent calcium gluconate contains 9.3 mg/ml of elemental calcium. The dose of intravenous calcium should be slowly tapered as oral calcium salts and vitamin D metabolites become effective.¹ It is important to remember to add calcium salts to saline and not to fluid preparations that contain lactate,

For long-term monitoring, the target serum calcium concentration should be just below the reference range (8 to 9.5 mg/dl) rather than in the middle or upper range of normal.

response being the determining factor for the volume administered.⁴ This rapid emergency therapy is invariably successful, with a response noted within minutes of initiating the infusion.² However, clinical signs such as nervousness, panting, and behavioral changes may persist despite the return of normocalcemia during the acute period, potentially reflecting a lag in cerebrospinal fluid equilibration with the extracellular fluid.¹

It is very important to monitor a patient's heart rate and, ideally, electrocardiogram during intravenous infusion of calcium salts.¹ Bradycardia, sudden elevation of the ST segment, shortening of the QT interval, or premature ventricular complexes may indicate the onset of cardiotoxicity from the calcium infusion.^{1,5} If such changes are noted, the injection should be temporarily discontinued and reinstituted at a slower rate.⁶

SUBACUTE THERAPY

The initial effects of intravenous calcium treatment persist for a limited time (1 to 12 hours).^{1,4} Long-term maintenance therapy with oral vitamin D and oral calcium supplementation usually requires a minimum of 24 to 96 hours before an effect is achieved.² Therefore, parenteral calcium must be provided during the post-tetany period between acute bolus treatment and the time at

acetate, bicarbonate, or phosphates because calcium salt precipitates can form.¹ The major disadvantage of this method of parenteral calcium therapy is that 24-hour monitoring of the continuous infusion is needed to ensure that an animal does not receive a life-threatening bolus of calcium.

The final option for parenteral calcium salt administration is intermittent subcutaneous injections of calcium gluconate. Calcium chloride should not be used because it is highly irritating to tissue.^{1,7} Many dosing schemes are available. The first dosing method is to subcutaneously administer (every 6 to 8 hours) the same amount of calcium gluconate that was originally required to control tetany.^{7,8} Alternatively, 1 to 2 ml/kg of 10% calcium gluconate can be given every 8 hours or a dose of 60 to 90 mg/kg/day can be divided and given in subcutaneous fluids several times per day.^{1,7} Regardless of the dosing technique used, calcium gluconate should always be diluted at least 1:1 with saline before administration.⁷

Subcutaneous administration of diluted calcium gluconate has been recommended as an effective, simple, and inexpensive method of supplementing calcium by various veterinary sources.^{2,7,9} In addition, this protocol has been repeatedly successful in supporting calcium

concentration during the lag period until vitamin D therapy becomes effective.² Stated advantages of the subcutaneous route are that intravenous catheter placement and monitoring with hospitalization are not required and owners can be taught to give subcutaneous injections at home, significantly lowering the cost of treatment.⁷ However, in humans, this route is not recommended because of the possibility of tissue necrosis, sloughing, and abscess formation.⁹ In addition, in both the human and veterinary literature, iatrogenic calcinosis cutis has reportedly occurred secondary to percutaneous penetration or absorption of calcium salts.^{7,10}

There are increasing numbers of veterinary case reports of suspected calcinosis cutis secondary to subcutaneous injection of calcium gluconate. For example, in one report, a young adult cat diagnosed with primary hypoparathyroidism developed firm subcutaneous masses in the intrascapular area and dependent axillary area.⁷ This occurred 7 days after appropriately diluted calcium gluconate was administered subcutaneously.⁷ The areas eventually developed full-thickness necrosis.⁷ In another case report, a young dog with primary hypoparathyroidism was treated with diluted calcium gluconate every 8 hours for 2 days.⁹ Again, within 1 week of treatment, the dog became febrile and developed painful subcutaneous fluid accumulation with pal-

subcutaneous administration of calcium gluconate solutions, especially in the presence of hyperphosphatemia, is likely involved.⁹ However, the possibility of iatrogenic calcinosis cutis and skin necrosis should be considered when choosing the administration route of calcium.^{7,9}

Serum calcium concentration should be monitored once to twice daily during subacute therapy, and the dose and rate of calcium administered should be altered to maintain a serum calcium concentration of 8 to 9 mg/dL.^{2,6,8} Once the calcium concentration has remained stable for 48 hours, the infusion rate or frequency of administration should be gradually tapered until parenteral therapy is no longer needed.⁸

MAINTENANCE THERAPY

The two main treatments widely used in chronic hypoparathyroidism are oral calcium and vitamin D analogues. Thiazide diuretics are also used in humans to increase renal tubular resorption of calcium, although their effects in dogs are debatable.¹¹

Vitamin D Therapy

Vitamin D therapy has been the foundation of long-term management of patients with hypoparathyroidism.⁴ Vitamin D is administered to enhance intestinal calcium transport. Because there is a lag time until

Severe calcinosis cutis and skin necrosis have been suspected in both dogs and cats secondary to subcutaneous calcium salt administration.

pable gritty deposits along the ventral body wall.⁹ Multiple skin biopsies revealed calcinosis cutis with pyogranulomatous dermatitis and dermoepidermal separation.⁹ The lesions progressed to areas with black discoloration, ulceration, and drainage of purulent fluid, with biopsies indicating severe pyogranulomatous panniculitis with mineralization.⁹ Such granulomatous panniculitis and epidermal necrosis and separation have been described in reports involving subcutaneous calcium gluconate administration to human infants and experimental animals.⁹

Therefore, although subcutaneous calcium salt administration is likely safe and efficacious in most dogs and cats, these reports demonstrate the potential for severe cutaneous lesions.⁹ Individual animal sensitivity to the

maximal effect of vitamin D metabolites, therapy should be initiated as soon as tetany is controlled and oral medication can be tolerated.⁶⁻⁷ The need for vitamin D therapy is usually permanent in dogs and cats with primary hypoparathyroidism.^{1,2,8} The most commonly used vitamin D preparations in veterinary medicine include ergocalciferol, dihydrotachysterol, and calcitriol (1,25-dihydroxycholecalciferol¹; Table 1).

Vitamin D₂

Vitamin D₂ (i.e., ergocalciferol) is widely available and relatively inexpensive. Ergocalciferol and its immediate metabolite have minimal vitamin D-receptor avidity; therefore, large doses must be administered. Large doses are also initially required to saturate fat deposits because

Table 1. Vitamin D Preparations for Treating Hypoparathyroidism

Preparation	Forms	Commercial Name	Doses	Time for Maximal Effect	Time for Toxicosis to Resolve
Vitamin D ₂ (ergocalciferol)	Capsules: 25,000 and 50,000 IU	Calciferol (Rorer) Drisdol (Winthrop-Breon) Deltalin (Lilly)	Initial: 4,000–6,000 U/kg/day	5–21 days	1–18 wk
	Oral syrup: 8,000 U/ml	Drisdol (Winthrop-Breon)	Maintenance: 1,000–2,000 U/kg once daily to once weekly		
	IM Injectable: 50,000 U/ml	Calciferol (Rorer) Vitadee (Gotham)			
Dihydrotachysterol	Tablets: 0.125, 0.2, and 0.4 mg	Dihydrotachysterol (Philips-Roxane)	Initial: 0.02–0.03 mg/kg/day for 2 days or to effect	1–7 days	1–3 wk
	Capsules: 0.125 mg	Hytakerol (Winthrop- Breon)	Maintenance: 0.01–0.02 mg/kg q24–48h		
	Oral solution: 0.25 mg/ml	Hytakerol (Winthrop- Breon)			
1,25-dihydroxy- vitamin D ₃ (calcitriol)	Capsules: 0.25 and 0.5 µg	Rocaltrol (Roche)	Initial: 0.02–0.03 µg/kg/day for 3–4 days	1–4 days	1–14 days
			Maintenance: 0.005–0.015 µg/kg/day		

ergocalciferol is highly lipid soluble.² Vitamin D₂ therapy has multiple negative aspects that clinicians must consider before administering it. First, there is significant individual variation in the dose needed to induce normocalcemia.¹² In addition, it has a long onset of action, taking up to 5 days to 3 weeks after initiation of therapy for effect. More important, if hypercalcemia occurs, it is not easily reversed because of the long half-life of vitamin D₂. As a result, hypercalcemia may persist for 1 to 18 weeks after vitamin D₂ has been discontinued.¹³ Because of such potential problems and the availability of other treatment options for hypoparathyroidism, vitamin D₂ is no longer used by most practitioners.

Dihydrotachysterol

Dihydrotachysterol is a potent synthetic vitamin D analogue that is widely available but more expensive than vitamin D₂. It has an onset of action and half-life between those of vitamin D₂ and calcitriol.¹ The more

rapid onset of action and increased effectiveness of dihydrotachysterol are a result of its stereochemistry. The A ring of the sterol structure is rotated 180° so that the hydroxyl group in the 3 position serves as a pseudo-1-hydroxyl group.¹³ Therefore, after administration, this drug requires hepatic 25-hydroxylation but does not require renal 1α-hydroxylation. The ability to bypass this step explains the rapid action and potency of dihydrotachysterol in treating hypoparathyroidism, in which renal 1α-hydroxylase activity is deficient in the absence of PTH.¹²

Dihydrotachysterol should initially be administered at a dose of 0.03 mg/kg/day PO for 2 days or until effect, then 0.02 mg/kg/day PO for 2 days, and finally 0.01 mg/kg/day PO in divided doses.^{2,3,13} Using a loading dose reduces the time needed to achieve maximal calcemic effects.^{1,6} Similar doses can be administered to cats. In a study of five cats with primary hypoparathyroidism, the final maintenance doses ranged from 0.004 to 0.04 mg/kg/day PO with a mean of 0.015 mg/kg/day.¹⁴

Table 2. Calcium Preparations for Treating Hypoparathyroidism

	Drug	Preparation	Available Calcium	Dose
Injectable	Calcium gluconate	10% solution in 10-ml vials	9.3 mg of calcium per milliliter	0.5–1.5 ml/kg (5–15 mg/kg) slow IV to effect 1–2 ml/kg diluted 1:1 with saline SC
	Calcium chloride	10% solution in 10-ml vials	27.2 mg of calcium per milliliter	5–15 mg/kg/hr IV
Oral	Calcium carbonate	Tablets: 500, 650, and 1250 mg	200, 260, and 500 mg of calcium per tablet	25 mg/kg q8–12h of elemental calcium
	Calcium lactate	Tablets: 325 and 650 mg	42 and 85 mg of calcium per tablet	
	Calcium chloride	Powder	27.2%	
	Calcium gluconate	Tablets: 325, 500, 650, and 1,000 mg	30, 45, 60, and 90 mg of calcium per tablet	
	Calcium carbonate–gluconate	700 mg	25 mg of calcium per tablet	

The maximal effect of dihydrotachysterol can be seen within 1 to 7 days.³ If toxicosis occurs, the lag period between discontinuing dihydrotachysterol and noting a fall in the serum calcium concentration is typically 4 to 21 days. Compared with vitamin D₂, the more rapid onset of action and shortened period of toxicosis in patients treated with dihydrotachysterol allow veterinarians to have more control over therapy and avoid prolonged periods of hypo- and hypercalcemia.^{2,3,13}

Dihydrotachysterol is available as tablets, capsules, and an oral solution.⁶ The latter is convenient for long-term treatment of hypoparathyroid cats and small dogs.⁶ Certain cats and dogs have reportedly appeared resistant to the tablet form of the drug but readily respond to the liquid form.²

Calcitriol

Calcitriol (i.e., 1,25-dihydroxyvitamin D₃) is the vitamin D compound of choice for managing hypoparathyroidism because it offers the advantages of the most rapid onset of action and the shortest biologic half-life (i.e., 2 to 4 days).^{1,5} Because 1,25-dihydroxyvitamin D₃ does not require activation by the kidneys, physiologic doses maintain normocalcemia.^{2,12}

A calcitriol dose of 0.03 to 0.06 µg/kg/day PO has traditionally been recommended.³ However, it has recently been suggested that this dose may be satisfac-

tory as a loading dose but too high for long-term maintenance therapy.¹ A loading dose of 0.02 to 0.03 µg/kg/day PO for the initial 3 to 4 days and a maintenance dose of 0.005 to 0.015 µg/kg/day PO are now recommended.¹ The dose of calcitriol is divided twice daily to ensure sustained effects on intestinal epithelium for calcium uptake.¹

The maximal effect of calcitriol can be seen in 1 to 4 days.³ If hypercalcemia results from overdosing, it can be rapidly corrected by temporarily discontinuing the drug, with toxicosis subsiding in 1 day to 2 weeks.^{1–3,12} In addition, the dose of calcitriol can be adjusted more frequently because there is less lag time for effect on serum calcium.¹

The major disadvantages of calcitriol are its expense and the potency of the available capsule sizes.^{6,12} The capsules are designed for human use and are not well formulated for small dogs and cats.⁶ Therefore, it is often necessary to have a compounding pharmacy reformulate calcitriol to a dose suitable for smaller patients.¹

Calcium Supplementation

Calcium supplementation is an important component in the treatment regimen for hypoparathyroidism (Table 2). When calcium intake is low, active intestinal transport mechanisms under the control of calcitriol are responsible for calcium uptake. However, when calcium

intake is high, vitamin D-independent, passive, intestinal absorption of calcium occurs.¹ Clinicians can take advantage of this passive mechanism for intestinal calcium transport early in the treatment of hypoparathyroidism before adequate vitamin D concentrations are reached.¹ Therefore, supplementation should be implemented as soon as possible. Once vitamin D therapy is in effect, supplementing calcium in conservative doses is also recommended to ensure that sufficient calcium is available for increased intestinal absorption by vitamin D, which is the chief mechanism by which vitamin D raises serum calcium.^{2,6} This is especially important in anorectic animals.¹

Oral supplementation can be provided by administering calcium as a gluconate, lactate, chloride, or carbonate salt.^{2,3} The most common mistake in administering calcium salts is thinking in terms of weight of salt rather than quantity of elemental calcium.¹² There are large differences in calcium content among the various salts, although there is little difference in effect when equimolar amounts are given.¹² For example, calcium gluconate contains 9% calcium and 1 g yields 4.5 mEq, whereas calcium carbonate is 40% calcium and 1 g yields 20 mEq of calcium. The differences in calcium content of the salts can help determine the best option for ease of administration. Because calcium gluconate and lactate tablets contain relatively small quantities of elemental calcium, relatively large numbers of tablets must be given.² Calcium chloride and carbonate tablets contain large quantities of calcium, allowing fewer pills to be administered. However, calcium chloride tablets tend to produce gastric irritation.² Calcium carbonate is considered the preparation of choice in treating human hypoparathyroidism because of its high percentage of calcium (i.e., 40%), wide availability, low cost, and lack of gastric irritation.⁴ Although no specific research is available to support a recommendation of one calcium supplement over another in veterinary patients, calcium carbonate is the most widely used for the aforementioned reasons. The oral dose of elemental calcium is 25 mg/kg bid or tid.⁸ These recommendations are approximate; the actual dose given should be altered according to the individual patient's serum calcium concentration.

Normal dietary intake of a commercial pet food provides sufficient calcium to maintain adequate serum calcium levels in the presence of vitamin D metabolite treatment for most patients.^{1,2} Consequently, as the vitamin D dose and serum calcium concentration reach a steady level, the dose of oral calcium can be gradually

tapered and discontinued over a period of 2 to 4 months.^{4,8}

Although specific therapy for lowering serum phosphate levels is typically not needed, it should be considered when phosphate levels are high despite adequate control of calcium. Another advantage of using calcium carbonate as an oral calcium supplement is that it acts as an intestinal phosphate binder.^{1,3} Therefore, calcium carbonate can be continued indefinitely if serum phosphorus levels remain increased.¹ If additional therapy is necessary, low-phosphorus diets and oral phosphate binders such as aluminum hydroxide may be used.³ A lower serum phosphorus concentration may allow increased endogenous synthesis of calcitriol because the phosphate-mediated inhibition of the renal tubular 1α -hydroxylase system is decreased.¹

Parathyroid Hormone Treatment

Unlike hypoadrenocorticism, hypothyroidism, and diabetes mellitus, hypoparathyroidism is one of the few remaining hormonal deficiencies in which physiologic replacement therapy is unavailable.¹⁵ Conventional therapy with vitamin D analogues increases intestinal calcium absorption, which increases serum calcium levels, but does not correct the lack of renal tubular calcium resorption.¹⁵ Chronic hypercalciuria may lead to nephrocalcinosis and renal insufficiency.¹⁵

Approximately 35 years ago, early efforts to treat human hypoparathyroidism with bovine PTH failed because of rapid development of resistance that was likely due to anti-PTH antibodies.^{15,16} Since then, however, multiple studies have been conducted using synthetic human PTH (1-34). In recent human studies, once- and twice-daily subcutaneous synthetic human PTH (1-34) were compared with conventional calcitriol therapy.^{15,16} The studies evaluated calcium levels in serum and urine, creatinine clearance, markers of bone turnover, and bone mineral density.^{15,16} It was found that PTH maintained serum calcium in the normal range with decreased urine calcium excretion compared with calcitriol treatment.^{15,16} Both treatments produced similar bone mineral changes, although increased levels in bone turnover markers were seen only in the PTH group.^{15,16} Although there were no apparent adverse bone changes in these studies, another recent report in PTH-treated rats showed an increased dose-dependent risk of osteosarcoma.¹⁵

Replacement therapy with synthetic PTH appears promising for the future. Its use to treat veterinary

patients is conceivable because the active amino-terminal portion of the molecule appears to have been highly conserved in evolution and would therefore be unlikely to elicit an immune response.¹ However, clinical use of this therapy remains questionable and a great deal of research is still needed to evaluate the long-term skeletal impact, the potential for decreased renal dysfunction, and the development of antibodies.¹⁶

MONITORING AND COMPLICATIONS

Hypoparathyroidism requires intense patient monitoring to maintain adequate control and decrease the potential for serious complications. The key to successful treatment is frequent monitoring of the serum calcium concentration during both the acute phase of hypocalcemia and long-term maintenance therapy. During initial stabilization, patients should be hospitalized with daily calcium monitoring until their serum calcium concentration remains at 8 to 9 mg/dl without parenteral support.⁴ At that point, patients can be discharged with weekly calcium measurements until vitamin D and calcium therapy is regulated. From then on, patients should be checked every 1 to 3 months indefinitely.³

Both hypocalcemia and hypercalcemia are common problems encountered during therapy.⁴ The target level for serum calcium should be just below the reference range (8 to 9.5 mg/dl) rather than in the middle or upper range of normal.^{1,6} This concentration is high enough to prevent signs of hypocalcemia but lessens the likelihood that hypercalcemia and its deleterious side effects will develop. In the absence of PTH, renal tubular resorption of calcium is abnormally low, and much of the calcium absorbed from the gastrointestinal tract is lost in the urine.⁶ If the serum calcium concentration is normalized to concentrations of 10 mg/dl or greater, the filtered load of calcium increases, resulting in severe hypercalciuria and increased risk of urolithiasis, nephrocalcinosis, and progression of renal disease.^{2,6}

If the serum calcium concentration remains below the target level, the dose should be adjusted in 10% to 20% increments.^{1,6} It is important to change the dose of the vitamin D metabolite gradually and to make sure that enough time has been given to see its maximal effect before the dose is changed again.¹ The time lag for this effect varies, depending on which vitamin D metabolite is being administered.¹

Hypercalcemia is a common but serious complication of vitamin D therapy and may be delayed and pro-

longed, depending on the type of vitamin D analogue used.⁵ Hypercalcemia can result in death or chronic renal damage severe enough to cause renal failure.¹ Owners should be instructed to watch for signs of hypercalcemia, including polydipsia, polyuria, anorexia, vomiting, and depression, so that prompt treatment can be initiated to prevent hypercalcemic complications.^{1,6} If hypercalcemia develops, calcium and vitamin D treatment should be temporarily withdrawn until the serum calcium concentration is normal.^{1,3,6} Vitamin D metabolites should then be reinstituted at a lower maintenance dose. Patients with severe hypercalcemia should be hospitalized for diuresis induced by intravenous saline and furosemide administration.^{1,6} A combination of steroids and potentially bisphosphonates or calcitonin may also be indicated in severe cases.¹

PROGNOSIS

The prognosis for dogs and cats with hypoparathyroidism largely depends on the owner.^{2,8} With a dedicated owner, proper lifelong therapy, and conscientious monitoring, the prognosis for patients with primary hypoparathyroidism is good.^{4,7,8}

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ARTICLE #3 CE TEST



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1. Hypoparathyroidism is an endocrine disease characterized by

- a. hypocalcemia and hypophosphatemia.
- b. hypocalcemia and hyperphosphatemia.
- c. hypercalcemia and hypophosphatemia.
- d. hypercalcemia and hyperphosphatemia.

2. Which statement concerning treatment of hypoparathyroidism is false?

- a. Vitamin D fully compensates for the physiologic actions of PTH.
- b. The mainstay of therapy is vitamin D metabolites.
- c. Once diagnosed, animals require lifelong treatment.
- d. Vitamin D therapy is primarily used to increase intestinal calcium transport.

3. Calcium _____ is the oral preparation of choice because of its wide availability, lack of gastric irritation, and large quantity of calcium, allowing fewer pills to be administered.

- a. gluconate
- b. lactate
- c. chloride
- d. carbonate

4. Calcitriol

- a. has an onset of action between those of vitamin D₂ and dihydrotachysterol.
- b. requires activation in the liver and kidneys.
- c. is inexpensive and easy to dose.
- d. has a maximal effect within 1 to 4 days of initiation.

5. Which statement regarding dihydrotachysterol is false?

- a. Because of its stereochemistry, it does not require renal 1 α -hydroxylation.
- b. Its maximal effect occurs within 1 to 7 days.
- c. Because of its long half-life, toxicosis results in prolonged hypercalcemia (i.e., 1 to 18 weeks).
- d. A loading dose is used to achieve maximal effects.

6. Which statement regarding phosphorus is false?

- a. Specific therapy for lowering phosphate levels is often needed.
- b. Calcium carbonate is an oral calcium supplement that also acts as a phosphate binder.
- c. A lower serum phosphorus concentration may allow increased synthesis of calcitriol.
- d. The risk of tissue calcification with subcutaneous calcium administration is greater in patients with hyperphosphatemia.

7. Calcium supplementation

- a. must be continued for the remainder of an affected patient's life.
- b. is recommended to ensure that calcium is available for intestinal absorption by vitamin D.
- c. is not important if an animal is anorectic.
- d. is available only in parenteral form.

8. The target level for serum calcium during maintenance is _____ mg/dl.

- a. 7 to 8.5
- b. 8 to 9.5
- c. 9 to 10.5
- d. 10 to 11.5

9. Which statement regarding subcutaneous administration of calcium is false?

- a. Calcium chloride is considered safe.
- b. Calcium gluconate should be diluted to a 1:1 ratio with saline.
- c. Severe cutaneous lesions have occurred after subcutaneous administration of calcium.
- d. The same dose that acutely controlled tetany can be given every 6 to 8 hours.

10. Which vitamin D analogue has the most rapid onset of action and the shortest biologic half-life?

- a. vitamin D₂
- b. calcitriol
- c. synthetic PTH
- d. dihydrotachysterol