

BROMIDE (Veterinary—Systemic)

There are no commercial human- or veterinary-labeled bromide products in the United States or Canada.

Category: Anticonvulsant.

Indications

Note: The text between ^{ELUS} and ^{EL} describes uses that are not included in U.S. product labeling. Text between ^{ELCAN} and ^{EL} describes uses that are not included in Canadian product labeling.

For this medication, the ^{ELUS} and ^{ELCAN} designations reflect a lack of commercial product availability in the countries indicated. See the *Regulatory Considerations* section below this monograph.

Dogs

Accepted

^{ELUS,CAN}Epilepsy (treatment)^{EL}—Bromide is used in the treatment of epilepsy in dogs (Evidence rating: A-2,3), either as an addition to therapy with other agents, such as phenobarbital, or as the sole anticonvulsant.^(R-1-4) Addition of bromide to phenobarbital therapy can allow significant reduction in the serum concentration of phenobarbital required for seizure control and, therefore, a reduction in the phenobarbital dose.^(R-1; 2; 4) In some patients, phenobarbital therapy can be discontinued for maintenance on bromide alone. Alternatively, treatment of epilepsy may begin with bromide as the sole anticonvulsant.

Cats

Potentially effective

^{ELUS,CAN}Epilepsy (treatment)^{EL}—Caution is advised in the use of bromide for the treatment of seizures in cats (Evidence rating: C-2,3). Retrospective studies have shown that bromide can be effective in the control of seizures in some cats; however, some animals may develop a nonproductive cough, the mechanisms of which have not been clearly defined.^(R-5; 30) Reports are that this effect can be self-limiting over time with discontinuation of treatment; however, it appears to be bronchoalveolar in nature and, therefore, potentially fatal.^(R-5; 30)

Regulatory Considerations

U.S. and Canada—

There are no commercial products; therefore, bromide must be purchased as medicinal or analytic grade potassium or sodium bromide from an approved source and compounded for veterinary use.^(R-1; 5) In the United States, refer to the Animal Medicinal Drug Use Clarification Act,^(R-6) Food and Drug Administration regulations pertaining to compounding (CFR 21 Part 530.13),^(R-29) and the current United States Food and Drug Administration's Compliance Policy Guide on *Compounding of Drugs for Use in Animals*.^(R-7) In Canada, refer to the Health Canada Health Products and Food Branch's *Policy Framework on Manufacturing and Compounding Drug Products in Canada*.^(R-8)

Evidence Quality

- A Good evidence to support a recommendation for use
- B Moderate evidence to support a recommendation for use
- C Insufficient evidence to support a recommendation for use
- D Moderate evidence to support a recommendation against use
- E Good evidence to support a recommendation against use

Chemistry

Chemical group: Elemental halide.^(R-9)

Molecular formula:

Potassium Bromide: KBr.^(R-34)

Sodium Bromide: NaBr.^(R-34)

Molecular weight:

Potassium Bromide: 119.00.^(R-34)

Sodium Bromide: 102.89.^(R-34)

Description:

Potassium Bromide: Colorless crystals or white granules or powder. Melting point is 730 °C.^(R-34)

Sodium Bromide: White crystals, granules or powder. Melting point is 755 °C.^(R-34)

Solubility:

Potassium Bromide: One gram dissolves in 1.5 mL of water, 1 mL of boiling water, 250 mL of alcohol, or 4.6 mL of glycerol. An aqueous solution is neutral.^(R-34)

Sodium Bromide: One gram dissolves in 1.1 mL of water, 16 mL of alcohol, or 6 mL of methanol. An aqueous solution is practically neutral.^(R-34)

Pharmacology/Pharmacokinetics

Mechanism of action/Effect: The mechanism of action of bromide has not been clearly defined. By preferential movement across neuronal membranes via gamma-amino butyric acid (GABA)-activated chloride channels, bromide may aid in controlling seizures through hyperpolarization of neuronal cell membranes, leading to stabilization and decreased sensitivity to epileptic foci.^(R-2; 9; 28)

Absorption: Oral bioavailability—

Dogs: F = 46%, with administration of sodium bromide solution.^(R-11)

Human beings: F = 96 ± 6%, with administration of sodium bromide solution.^(R-16)

Distribution: Bromide is distributed to the volume of the extracellular space and some intracellular sites.^(R-9; 15) With therapeutic dosing, bromide will accumulate in cerebrospinal fluid, in the interstitial tissues of the brain, and in neurons, by crossing neuronal chloride channels.^(R-9) In dogs, the cerebrospinal fluid to serum ratio of bromide concentration at steady state is 0.77,^(R-10) while in human beings this ratio is 0.37.^(R-17) However, cerebrospinal fluid levels do not necessarily correlate with concentrations in the brain.^(R-39)

Volume of distribution—*Dogs:* 0.45 ± 0.07 L/kg.^(R-11)

Protein binding: Bromide is minimally protein bound.^(R-39)

Biotransformation: Bromide is not biotransformed by the liver and is eliminated unchanged, primarily by renal clearance.^(R-9)

Half-life: Elimination—Bromide is freely filtered by the glomerulus, but is reabsorbed by the kidneys, in competition with chloride.^(R-15) Bromide reabsorption will predominate in the absence of a

Evidence Type

- 1 Species-specific evidence from at least one large randomized and controlled trial (RCT) or multiple small RCTs
- 2 Species-specific evidence from a small RCT, disease models, large case studies, pharmacokinetic studies using surrogate endpoints, or evidence from well-designed trials in a different species that is considered appropriate for comparison
- 3 Dramatic results from either well-designed, species-specific trials without controls or small case studies
- 4 Pharmacokinetic studies without surrogate endpoints
- 5 *In vitro* studies
- 6 Opinions of respected authorities on the basis of clinical experience or reports of expert committees

large chloride load, causing a significantly extended elimination half-life in human beings and dogs.^(R-11; 13; 15) Increasing chloride intake by an animal on a low chloride diet will decrease the half-life of elimination.^(R-27; 28)

Steady state serum concentration for a particular dose is expected in less than 8 weeks in cats but up to 4 to 5 months in dogs.

Cats: With an oral dose of 10 mg bromide (15 mg potassium bromide) per kg of body weight every twelve hours for eight weeks— 11.2 ± 1.4 days.^(R-5)

Dogs:

Single oral dose of 14 mg bromide (18 mg sodium bromide) per kg of body weight—
Fed a diet of 0.2% chloride on a dry matter basis: 69 ± 22 days.^(R-28)

Fed a diet of 0.4% chloride on a dry matter basis: 46 ± 6 days.^(R-28)

Fed a diet of 1.3% chloride on a dry matter basis: 24 ± 7 days.^(R-28)

Single dose of 20 mg bromide (25.6 mg sodium bromide) per kg of body weight and fed a diet containing 0.4% chloride—

Intravenous dose: 37 ± 10 days.^(R-11)

Oral dose: 46 ± 9 days.^(R-11)

With an oral dose of 20 mg bromide (30 mg of potassium bromide) per kg of body weight every twelve hours for 115 days and fed a diet containing 0.55 to 0.72% chloride: 15.2 ± 9 days.^(R-10)

Human beings: Fasting twelve hours before administration—

Intravenous administration: 9.4 ± 1.5 days.^(R-16)

Oral administration: 11.9 ± 1.4 days.^(R-16)

Concentrations:

Cats—An oral dose of 10 mg bromide (15 mg potassium bromide) per kg of body weight every twelve hours for eight weeks produced a peak serum concentration of 1.1 ± 0.2 mg/mL at the eighth weekly sample.^(R-5)

Dogs—

A single oral dose of 14 to 20 mg bromide (18 to 26 mg sodium bromide) per kg of body weight produced a peak serum concentration of 0.08 ± 0.01 mg/mL at 0.69 ± 0.13 hours.^(R-11; 28)

An oral dose of 20 mg bromide (30 mg potassium bromide) per kg of body weight every twelve hours for 115 days produced a steady state serum concentration of 2.45 mg/mL (range, 1.78 to 2.69 mg/mL) in Beagles fed a diet containing 0.55% chloride.^(R-10)

Elimination: Renal. Because bromide is widely distributed, minute amounts will also be excreted in saliva, sweat, and feces.^(R-9; 10)

Rate of elimination of bromide will increase in animals that receive a high level of chloride supplementation.

Clearance—**Dogs:** Administered a single intravenous dose of 20 mg of bromide (25.6 mg sodium bromide) per kg of body weight and fed a diet containing 0.4% chloride— 9 ± 3.9 mL/day/kg.^(R-11)

Precautions to Consider

Reproduction/Pregnancy/Lactation

Cats and dogs: The effects of bromide on canine and feline reproduction have not been investigated.

Pediatrics

The safety of administering bromide to neonates and young animals has not been evaluated.

Human information—Although bromide has been replaced by newer anticonvulsant medications in adult human beings, there are reports it may be used in infants or children with seizures refractory to other medications if the side effects (acne, fatigue,

loss of appetite, weight loss) are considered tolerable for these patients.^(R-31-33)

Drug interactions and/or related problems

The following drug interactions and/or related problems have been selected on the basis of their potential clinical significance (possible mechanism in parentheses where appropriate)—not necessarily inclusive (» = major clinical significance):

Note: Combinations containing any of the following medications, depending on the amount present, may also interact with this medication.

Chloride-containing medications and foods

(bromide and chloride compete for reabsorption by the kidneys; consumption of increased amounts of chloride can promote loss of bromide in the urine, leading to a lowering of serum bromide concentrations; conversely, decreased chloride consumption will promote increased renal reabsorption of bromide; see also the *Veterinary Dosing Section*)^(R-2; 9; 11; 13; 15; 28)

Halothane anesthesia

(a percentage of inhaled halothane is metabolized by dogs, horses, and other species, to produce bromide, along with other compounds; peak serum concentration occurs within about a day and, in one group of dogs, ranged from 0.04 to 0.088 mg/mL and persisted, with some diminishment, for at least ten days;^(R-21; 22) although this increase is unlikely to be a significant risk for bromide toxicity in most dogs, it may be a consideration in animals that must have repeated anesthesia or that already require high serum bromide concentration for seizure control)

Laboratory value alterations

The following have been selected on the basis of their potential clinical significance (possible effect in parentheses where appropriate)—not necessarily inclusive (» = major clinical significance):

With physiology/laboratory test values

Serum chloride

(some laboratory chloride tests may show serum chloride concentration to be higher than the actual concentration while an animal is on bromide therapy; this effect has been utilized in the preliminary diagnosis of bromide toxicity in human beings and has been reported in a dog with bromide toxicity)^(R-2; 17; 19)

Medical considerations/Contraindications

The medical considerations/contraindications included have been selected on the basis of their potential clinical significance (reasons given in parentheses where appropriate)—not necessarily inclusive (» = major clinical significance).

Except under special circumstances, this medication should not be used when the following medical problems exist:

For cats

Airway disease, including

Asthma

(because bromide is associated with development of a cough that seems to be bronchoalveolar in nature, cats with existing airway disease could be at risk for worsening signs if treated with bromide)^(R-5; 30)

Risk-benefit should be considered when the following medical problems exist:

Renal dysfunction

(because bromide is eliminated by the kidneys, insufficient renal function can lead to unexpected bromide concentrations; careful induction and monitoring is recommended;^(R-26) some clinicians recommend one-half the initial recommended dose)^(R-35)

For potassium bromide only

Hypoadrenocorticism

(sodium bromide administration is preferred for animals with disorders in potassium regulation)

For sodium bromide only

Congestive heart disease
Hypertension

(the sodium content of this salt can be significant for patients that are intolerant of sodium, particularly when considering administration at high doses to quickly achieve therapeutic serum concentration)

Patient monitoring

The following may be especially important in patient monitoring (other tests may be warranted in some patients, depending on condition; » = major clinical significance):

Observation for signs of bromide toxicity

(all animals receiving bromide should be monitored for signs of toxicity, particularly during initial tailoring of dose; see also the *Side/Adverse Effects* and *Overdose* sections)

Serum bromide concentration

(serum bromide concentration monitoring should begin within 4 weeks of the start of therapy in dogs and within the first two weeks in cats; due to its long half-life, the time of sample collection in relation to dosing is not critical; however, if loading doses are administered, serum bromide concentrations can be monitored at the completion of a loading dose protocol; see the *General Dosing Information* section in this monograph for more information)^(R-18)

Side/Adverse Effects

The following side/adverse effects have been selected on the basis of their potential clinical significance (possible signs in parentheses where appropriate)—not necessarily inclusive:

Those indicating need for medical attention

Incidence unknown

Cats^(R-5; 30)

Coughing; polydipsia; sedation; vomiting; weight gain

Note: *Coughing* has been reported to occur in as many as 35 to 42% of cats administered bromide for the treatment of seizures.^(R-5; 30) This effect can appear within a few weeks to two years after bromide therapy is begun and does not appear to be related to serum concentration.^(R-5; 30) While the pathogenesis is not well defined, thoracic radiographs, when available, have shown mild to marked peribronchial infiltrates.^(R-30) Bronchoalveolar lavage performed in two cats suggested eosinophilic inflammation.^(R-30) Coughing appears to be generally self-limiting if bromide treatment is discontinued but may require many months to over a year to resolve.^(R-5; 30) Because this effect appears to be bronchoalveolar in nature, it is considered potentially fatal.

Dogs

Ataxia;^(R-2; 10; 17) **gastric irritation**—with oral bromide; **lethargy/sedation;**^(R-3; 14) **pancreatitis;**^(R-25) **personality changes;**^(R-36) **polydipsia/polyuria;**^(R-2; 36) **polyphagia**^(R-2; 36)

Note: See also the *Overdose* section in this monograph for information on bromide toxicity, which can occur in some dogs with therapeutic serum concentrations (1.5 to 3 mg/mL). During initial administration of bromide, temporary *ataxia* and/or *sedation* may occur and resolve in the first few weeks; however, similar signs can appear with the onset of bromide toxicity as serum concentrations rise. Because of the relatively long time frame to steady state serum concentration and the range of individual responses to a particular dose, each dog on bromide therapy should be monitored for signs of toxicity.

Pancreatitis has been reported to be more frequent in dogs on concurrent phenobarbital and bromide therapy than dogs on phenobarbital alone.^(R-25) There is no information on the relative frequency of this adverse effect in association with bromide therapy alone.

There are reports of *personality changes* occurring occasionally in dogs on bromide, including attention seeking, irritability or aggression, and aimless pacing.^(R-36)

Because many of the efficacy studies of bromide in the treatment of seizures in dogs investigated its use in addition to phenobarbital, there is little published documentation of *polydipsia* and *polyuria* associated with bromide therapy alone in dogs. Polydipsia and polyuria are considered less common with bromide therapy than with phenobarbital therapy.^(R-36)

There are anecdotal reports that about 25% of dogs on bromide therapy develop some degree of *polyphagia*.^(R-36)

Overdose

For more information in cases of overdose or unintentional ingestion, **contact the American Society for the Prevention of Cruelty to Animals (ASPCA) National Animal Poison Control Center** (888-426-4435 or 900-443-0000; a fee may be required for consultation) **and/or the drug manufacturer**.

Note: Signs of bromide toxicity are dose-related but dogs differ in their sensitivity. Bromide toxicity has rarely been reported in dogs with serum bromide concentrations of less than 1.5 mg/mL. Signs have been reported in some dogs with relatively low serum concentration (2.75 mg/mL) while not appearing in other dogs with significantly higher concentration (4 mg/mL).^(R-10; 36) Dogs on concurrent bromide and phenobarbital therapy may be prone to bromide toxicity at lower serum concentrations than when bromide is administered alone. Other physical factors may also play a role in sensitivity.^(R-10)

Short-term bromide toxicity is considered completely reversible.^(R-36) However, mild bromide toxicity can progress to more severe nervous system dysfunction with ongoing high serum concentration of bromide.^(R-17)

Other species—Bromide toxicity has also been reported in cattle, goats and horses fed hay that contained bromide ion residue from accidental treatment with methyl bromide. Signs included ataxia, hind limb weakness, joint swelling, and sedation or recumbency.^(R-20)

Human toxicity—The use of bromide therapy in human epileptics has been limited since the early 1900s. Reported adverse effects have included acne, dermatitis, lethargy, loss of appetite, necrotizing panniculitis (abdominal pain, elevated sedimentation rate, fever, hepatosplenomegaly, subcutaneous nodules), and weight loss. Reported signs and symptoms of toxicity have included ataxia progressing to quadriplegia with ongoing toxicity, excessive salivation, headache, mental changes (confusion, delusions, disorientation, hallucination, memory loss), mydriasis, and slurred speech.^(R-2; 4; 19; 21; 23; 24; 33) Bromide is believed to more readily pass into the cerebrospinal fluid in dogs than in man; thereby allowing more effective delivery of bromide to target cells with lower serum concentration.^(R-4; 9; 10)

Clinical effects of overdose

The following effects have been selected on the basis of their potential clinical significance (possible signs in parentheses where appropriate)—not necessarily inclusive:

Dogs

Signs may appear with serum bromide concentration >1.5 mg/mL in dogs on bromide therapy alone, but become more common as serum concentration increases, and most common when serum concentration exceeds 4 mg/mL in dogs receiving bromide as the only anticonvulsant (2 to 3 mg/mL in dogs receiving concurrent phenobarbital and bromide).^(R-10; 36)

Ataxia, progressing to quadriplegia with normal reflexes;^(R-2; 10; 17) **hind limb weakness, stiffness, and/or swelling**—often seen without ataxia;^(R-3; 10; 17) **lethargy/sedation, progressing to stupor**

and coma;^(R-3) **mental changes** (disorientation, irritability or aggression)^(R-26)

Reported with a dose of 200 to 500 mg/kg a day for 4 to 26 weeks.^(R-26)

Ataxia; diarrhea; hematochezia; salivation, excessive; shivering; skin lesions; stupor, progressing to coma and death

Treatment of bromide toxicity

Recommended treatment consists of the following:

- For dogs on concurrent phenobarbital and bromide therapy with mild sedation, ataxia, or hindlimb weakness, a 10 to 25% reduction in phenobarbital may resolve the signs within five to seven days.^(R-37)
- For dogs receiving bromide as the only anticonvulsant, administration may be discontinued for a few days to a week while monitoring serum concentration, to see if mild signs of toxicity will resolve.
- If necessary, renal excretion of bromide may be accelerated by increasing sodium chloride consumption or administering 0.9% sodium chloride intravenously over twelve hours, depending on the severity of the toxicity.^(R-15; 17; 26)
- Supportive treatment

Client Consultation

In providing consultation, consider emphasizing the following selected information:

- Not changing the animal's regular diet without consultation with the veterinarian.
- Keeping water readily available during the treatment period to avoid dehydration
- Familiarizing clients with signs of bromide toxicity, including hind limb ataxia or paresis,^(R-10) and instructing them to contact their veterinarian when signs are observed

General Dosing Information

Therapeutic serum concentration

Bromide

Cats—Target serum concentration for the treatment of seizures in cats has not been well defined. In one study, an average potassium bromide dose of 24.2 ± 11.23 mg per kg of body weight (mg/kg) a day produced a mean serum bromide concentration of 1.5 ± 0.7 mg/mL (18.8 ± 8.7 millimoles/liter [mmole/L] = 150 mg/dL = 1500 mcg/mL).^(R-5) A serum bromide concentration of 1 to 1.6 mg/mL was sufficient to control seizures in 7 of 15 cats treated with bromide or bromide and phenobarbital.^(R-5) About 35% of cats in this study developed a nonproductive cough; however, this effect was not considered dose-dependent.^(R-5) See the *Side/Adverse Effects* section in this monograph for more information.

Dogs—A retrospective study of 122 epileptic dogs investigated the serum concentrations of bromide and phenobarbital or bromide alone in dogs with successfully controlled seizures.^(R-1) Based on this and other data,^(R-2-4) the following initial target serum concentrations are recommended:

When added to phenobarbital therapy in dogs: 1 to 2.5 mg/mL (12.5 to 31 mmole/L = 100 to 250 mg/dL = 1000 to 2500 mcg/mL).^(R-1; 38)

For dogs beginning treatment with bromide alone: 1 to 3 mg/mL (12.5 to 37.5 mmole/L).

For dogs already receiving bromide that continue to have uncontrolled seizures, the upper end of the range (>2 mg/mL [>25 mmole/L]) may be a more effective target.^(R-1; 10) Some clinicians consider the high end of the therapeutic range in dogs being treated with bromide alone to be defined by the point where an individual dog develops adverse effects;^(R-35; 36) however, serum bromide assays may not be accurate above 4 mg/mL.^(R-37)

Additional information:

Because responses to bromide may vary among animals, treatment should be tailored to the individual, based on serum bromide concentration, seizure control, and the occurrence of adverse effects.^(R-3; 10)

Steady state serum concentrations are not expected in dogs for 4 to 5 months; however, therapeutic concentrations may be reached before steady state. For dogs, serum bromide concentration may be tested about 4 weeks after the beginning of treatment to assess the response to dose and, if results are satisfactory, tested next when steady state is estimated, typically about 4 months.^(R-18) If the dog is doing well and serum concentration is in the therapeutic range, serum concentration may then be tested every 6 to 12 months. When an animal is started with a loading dose, serum bromide is typically evaluated at the end of the loading period, then as described above.

Cats are expected to reach steady state serum concentration within 4 to 6 weeks.^(R-5) An initial serum bromide concentration may be done within 2 weeks of beginning treatment, and another when steady state is estimated, to assess the response to dose.

Phenobarbital

Dogs—The amount of phenobarbital administered may be decreased or discontinued in some dogs after the addition of bromide therapy. In one retrospective study, serum concentrations of phenobarbital could be reduced to 9 to 36 micrograms/mL in dogs that continued to need phenobarbital while on bromide therapy to control seizures.^(R-1)

To reduce phenobarbital after initiating bromide therapy, some clinicians recommend setting a serum bromide concentration target of >2 mg/mL and waiting from the start of bromide administration until either sedation develops or until serum bromide is in the therapeutic range. Then the phenobarbital dose is reduced by 10 to 25% every four to six weeks,^(R-37) while monitoring the level of seizure control.

Diet (chloride intake)

A stable level of chloride consumption is recommended during bromide therapy. Because bromide is eliminated in competition with chloride, increasing the amount of chloride in the diet consumed by an epileptic dog can significantly decrease serum concentrations of bromide by increasing elimination. Conversely, significantly decreasing the amount of chloride in the diet will promote increased renal absorption of bromide.^(R-10; 15) The authors of one study examining this relationship suggest that dogs fed high chloride diets ($\geq 1.3\%$ chloride on a dry matter basis) would require at least twice as much daily bromide as those fed low chloride diets (0.2 to 0.4% chloride) to maintain therapeutic bromide concentrations.^(R-28) Some therapeutic diets will contain chloride at concentrations approaching or at the high end of this range. This should be taken into consideration during initial treatment.^(R-28) Also, the addition to the diet of certain dog treats alone can lead to breakthrough seizures due to inadequate serum concentration.^(R-35) Animals that must be switched to high chloride diets once seizures are controlled may need an increase in bromide administered, depending on the degree of change in chloride intake; serum bromide concentrations may require careful monitoring to avoid potential loss of seizure control.^(R-27)

Intravenous administration

Potassium bromide is not recommended for intravenous injection because of the risk of cardiotoxicity from rapid potassium infusion.^(R-11)

Dosing and Dosage Forms

Note: The text between ^{ELUS} and ^{EL} describes uses not included in U.S. product labeling. Text between ^{ELCAN} and ^{EL} describes uses that are not included in Canadian product labeling.

For this medication, the ^{ELUS} and ^{ELCAN} designations reflect a lack of commercial product availability in the countries indicated.

bromide concentrations are not reached, additional intravenous sodium bromide may be administered.^{(R-14)EL}

DOSAGES

^{ELUS,CAN} Dogs—

For *Potassium Bromide Oral Solution, Veterinary*

Note: The following dosages are expressed in terms of the potassium bromide salt (not bromide base). Potassium bromide is 67% bromide by weight.^(R-11; 34)

Dosing is adjusted by sampling serum bromide concentration, observing for any signs of toxicity, and recording response to therapy.

Epilepsy—Oral, 30 to 40 mg of potassium bromide per kg of body weight a day.^(R-3; 11; 37) When administered in addition to ongoing phenobarbital, the lower end of the dosage range is typical, while the higher dose is used when starting on bromide alone. This dose may be divided and administered every twelve hours with food to reduce nausea.^(R-35)

Administration of oral solution is reported to cause less vomiting than administration of capsules.^(R-40)

For dogs on diets that are high in chloride (>1% on a dry matter basis)—Oral, 50 to 80 mg of potassium bromide per kg of body weight a day may be necessary to reach therapeutic serum concentration.^(R-37)

Loading dose—

Accelerated: When bromide therapy is begun as the sole anticonvulsant—Oral, 40 to 50 mg of potassium bromide per kg of body weight a day has been used in some patients to more quickly reach therapeutic serum concentrations when beginning administration of potassium bromide alone to control seizures.^(R-14; 37) In one study, a dose of 30 mg/kg every twelve hours was found to produce a serum concentration of 1.8 to 2.7 mg/mL in 60 days.^(R-10)

Rapid: The following dose is typically reserved for dogs with severe and/or frequent seizures when beginning bromide therapy alone or when a previous anticonvulsant must be quickly discontinued due to adverse effects. To rapidly reach minimum therapeutic serum concentration (1 to 1.5 mg/mL)—Oral, 400 to 600 mg of potassium bromide per kg of body weight, divided into four doses, mixed with food, and administered over twenty-four to forty-eight hours.^(R-14; 35-37)

Note: Potassium bromide can cause gastric irritation, especially when a large amount is given orally at one time, as for a loading dose.^(R-9)

For *Sodium Bromide Oral Solution, Veterinary*

Note: The following dose is expressed in terms of the sodium bromide salt (not bromide base). Sodium bromide is 78% bromide by weight.^(R-11; 34)

Epilepsy—Oral, 26 to 34 mg of sodium bromide per kg of body weight a day.^(R-3; 11) When administered in addition to ongoing phenobarbital, the lower end of the dosage range is typical, while the higher dose is used when starting on bromide alone.

For *Sodium Bromide Injection, Veterinary*

Note: The following dose is expressed in terms of the sodium bromide salt (not bromide base). Sodium bromide is 78% bromide by weight.^(R-11; 34)

Epilepsy—Loading dose: To rapidly achieve minimum therapeutic concentration (1 to 1.5 mg/mL)—Intravenous, 600 to 1200 mg per kg of body weight, diluted in a solution and administered over eight hours.^(R-11; 14) If target serum

^{ELUS,CAN} Cats—

For *Potassium Bromide Oral Solution, Veterinary*

Note: The following dose is expressed in terms of the potassium bromide salt (not bromide base). Potassium bromide is 67% bromide by weight.^(R-11; 34)

Epilepsy—Although the safety and efficacy have not been established, an oral dose of 30 mg of potassium bromide per kg of body weight a day has been used in the treatment of seizures in cats.^(R-5) This dose may be divided and administered every twelve hours with food to reduce nausea.^(R-35)

See also the *Side/Adverse Effects* section of this monograph for more information on potentially serious effects in cats.

Loading doses may be unnecessary for cats. Therapeutic serum concentration could be reached in many cats in about two weeks without loading.^{(R-5)EL}

DOSAGE FORMS

Oral

POTASSIUM BROMIDE ORAL SOLUTION, VETERINARY

Strength(s) usually available: Potassium bromide is not available as a commercial product in the United States or Canada.

Therefore, it must be formulated from analytic grade material into capsules or an oral solution for veterinary use. Utilizing the services of a qualified compounding pharmacist to formulate an appropriate dosage form and strength for a specific animal is recommended.

Packaging and storage: Store in a refrigerator between 2 and 8 °C (36 and 46 °F), in a tight container, unless otherwise specified by a qualified compounding pharmacist.

USP requirements: See the *Potassium Bromide Oral Solution, Veterinary USP* (proposed) monograph.

SODIUM BROMIDE ORAL SOLUTION, VETERINARY

Strength(s) usually available: Sodium bromide is not available as a commercial product in the United States or Canada. Therefore, it must be formulated from analytic grade material into a dosage form for veterinary use. Utilizing the services of a qualified compounding pharmacist to formulate an appropriate dosage form and strength for a specific animal is recommended.

Packaging and storage: Store in a refrigerator between 2 and 8 °C (36 and 46 °F), in a tight container, unless otherwise specified by a qualified compounding pharmacist.

USP requirements: See the *Sodium Bromide Oral Solution, Veterinary USP* (proposed) monograph.

Parenteral

SODIUM BROMIDE INJECTION, VETERINARY

Note: A maximum intravenous dose of 150 mg of bromide per kg of body weight an hour was used to set *USP NF* endotoxin limits for this dosage form.

Strength(s) usually available: Sodium bromide is not available as a commercial product in the United States or Canada. Therefore, it must be formulated from analytic grade material into a dosage form for veterinary use. Utilizing the services of a qualified compounding pharmacist to formulate an appropriate dosage form and strength for a specific animal is recommended.

Packaging and storage: Store in a refrigerator between 2 and 8 °C (36 and 46 °F), unless otherwise specified by a qualified compounding pharmacist.

USP requirements: See the *Sodium Bromide Injection, Veterinary USP* (proposed) monograph.

Developed: 06/24/05

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