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PRESCRIPTION COMPOUNDING FOR

VETERINARY MEDICINE

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We customize individual prescriptions for the specific needs of our patients.
C A N I N E  C O N G E S T I V E  H E A R T  F A I L U R E

The following study found that pimobendan plus conventional therapy prolongs time to sudden death, euthanasia for cardiac reasons, or treatment failure in dogs with CHF caused by MMVD—“Effect of pimobendan or benazepril hydrochloride on survival times in dogs with congestive heart failure caused by naturally occurring myxomatous mitral valve disease: the QUEST study” (J Vet Intern Med. 2008 Sep-Oct;22(5):1124-35).

BACKGROUND: Myxomatous mitral valve disease (MMVD) continues to be an important cause of morbidity and mortality in geriatric dogs despite conventional therapy.

HYPOTHESIS: Pimobendan in addition to conventional therapy will extend time to sudden cardiac death, euthanasia for cardiac reasons, or treatment failure when compared with conventional therapy plus benazepril in dogs with congestive heart failure (CHF) attributable to MMVD.

ANIMALS: Two hundred and sixty client-owned dogs in CHF caused by MMVD were recruited from 28 centers in Europe, Canada, and Australia.

METHODS: A prospective single-blinded study with dogs randomized to PO received pimobendan (0.4-0.6 mg/kg/d) or benazepril hydrochloride (0.25-1.0 mg/kg/d). The primary endpoint was a composite of cardiac death, euthanized for heart failure, or treatment failure.

RESULTS: Eight dogs were excluded from analysis. One hundred and twenty-four dogs were randomized to pimobendan and 128 to benazepril. One hundred and ninety dogs reached the primary endpoint; the median time was 188 days (267 days for pimobendan, 140 days for benazepril hazard ratio = 0.688, 95% confidence limits [CL]=0.516-0.916, P=.0099). The benefit of pimobendan persisted after adjusting for all baseline variables. A longer time to reach the endpoint was also associated with being a Cavalier King Charles Spaniel, requiring a lower furosemide dose, and having a higher creatinine concentration. Increases in several indicators of cardiac enlargement (left atrial to aortic root ratio, vertebral heart scale, and percentage increase in left ventricular internal diameter in systole) were associated with a shorter time to endpoint, as was a worse tolerance for exercise.

CONCLUSIONS AND CLINICAL IMPORTANCE: Pimobendan plus conventional therapy prolongs time to sudden death, euthanasia for cardiac reasons, or treatment failure in dogs with CHF caused by MMVD compared with benazepril plus conventional therapy.” PMID: 18638016

With our state of the art compounding lab and pharmaceutical knowledge and experience, we can compound pimobendan as a flavored oral suspension.

An example of how you might prescribe follows:

<table>
<thead>
<tr>
<th>COMPOUNDED MEDICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pimobendan 0.5mg/ml</strong></td>
</tr>
<tr>
<td><strong>Flavored Oil Oral Suspension</strong></td>
</tr>
<tr>
<td>Quantity 30ml</td>
</tr>
<tr>
<td>Give 0.5mg/kg once a day or as directed</td>
</tr>
</tbody>
</table>
C A N I N E  E P I L E P S Y

The following clinical review was conducted to determine therapeutic serum drug concentrations in epileptic dogs treated with potassium bromide—“Therapeutic serum drug concentrations in epileptic dogs treated with potassium bromide alone or in combination with other anticonvulsants: 122 cases (1992-1996)” (J Am Vet Med Assoc. 1998 Nov 15;213(10):1449-53).

OBJECTIVE: To determine therapeutic serum drug concentrations in epileptic dogs treated with potassium bromide. DESIGN: Retrospective study. ANIMALS: 122 dogs with major motor epilepsy.

PROCEDURE: Medical histories were collected for epileptic dogs treated with potassium bromide with or without phenobarbital sodium or primidone, from which serum was submitted for bromide analysis from May 1992 to May 1996 to the Therapeutic Drug Monitoring Program at Cornell University's College of Veterinary Medicine. A therapeutic response (improved seizure control) was defined as a > or = 50% reduction in seizure frequency following initiation of bromide treatment. Serum bromide and phenobarbital concentrations and therapeutic outcome were determined for all dogs.

RESULTS: 72% of epileptic dogs had a > or = 50% reduction in seizure frequency following initiation of treatment with potassium bromide. Discontinuation of barbiturate treatment was possible in 19% of those dogs originally treated with phenobarbital or primidone. Of those dogs continued on bromide and phenobarbital, 45% maintained seizure control with serum phenobarbital concentrations < 20 micrograms/ml. Significantly higher serum bromide concentrations were required when dogs were initially or eventually treated with bromide alone (mean bromide concentration, 1,906 micrograms/ml) compared with dogs treated with potassium bromide along with a barbiturate (mean bromide concentration, 1,621 micrograms/ml).

CLINICAL IMPLICATIONS: When dogs are treated with bromide and phenobarbital, a reasonable therapeutic range for serum bromide concentrations is 810 to 2,400 micrograms/ml, and for bromide treatment alone, the range is 880 to 3,000 micrograms/ml. When phenobarbital is used in combination with bromide, a reasonable therapeutic range for serum phenobarbital concentrations is 9 to 36 micrograms/ml, although in some dogs treated with bromide, phenobarbital can eventually be discontinued. PMID: 9828942

With our state of the art compounding lab and pharmaceutical knowledge and experience, we can compound potassium bromide into a flavored oral solution; in a variety of strengths to meet the unique needs of each of your animals.

An example of how you might prescribe follows:

<table>
<thead>
<tr>
<th>COMPOUNDED MEDICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Potassium Bromide 250 mg/ml</strong></td>
</tr>
<tr>
<td><strong>Flavored Oral Solution</strong></td>
</tr>
<tr>
<td>Quantity 30 ml</td>
</tr>
<tr>
<td>Directions: Give 1 ml (250mg) daily or as directed</td>
</tr>
</tbody>
</table>
The following study suggests that cisapride may be useful in the treatment of cats with idiopathic megacolon: "Cisapride stimulates contraction of idiopathic megacolonic smooth muscle in cats" (J Vet Intern Med. 1997 Nov-Dec;11(6):313-8).

“We have previously shown that cisapride, a substituted piperidinyl benzamide, stimulates contraction of healthy feline colonic smooth muscle. The purpose of the present investigation was to determine the effect of cisapride on feline idiopathic megacolonic smooth muscle function. Longitudinal smooth muscle strips from ascending and descending colon were obtained from cats with idiopathic megacolon, suspended in a 1.5 mM Ca(2+)-HEPES buffer solution (37 degrees C, 100% O2, pH 7.4), attached to isometric force transducers, and stretched to optimal muscle length (Lo). Control responses were obtained at each muscle site with acetylcholine (10(-8) to 10(-4) M), substance P (10(-11) to 10(-7) M), or potassium chloride (10 to 80 mM). Muscles were then stimulated with cumulative (10(-9) to 10(-6) M) doses of cisapride in the absence or presence of tetrodotoxin (10(-6) M) and atropine (10(-6) M), or in a 0 calcium HEPES buffer solution. In cats with idiopathic megacolon, cisapride stimulated contractions of longitudinal smooth muscle from both the ascending and the descending colon. Cisapride-induced contractions were similar in magnitude to those induced by substance P and acetylcholine in the ascending colon, but were less than those observed in the descending colon. Cisapride-induced contractions in megacolonic smooth muscle were only partially inhibited by tetrodotoxin and atropine, but were virtually abolished by removal of extracellular calcium. We concluded that cisapride-induced contractions of feline megacolonic smooth muscle are largely smooth muscle mediated and dependent on influx of extracellular calcium. Cisapride-induced contractions in megacolonic smooth muscle are only partially dependent on enteric cholinergic nerves. Thus, cisapride may be useful in the treatment of cats with idiopathic megacolon.” PMID: 9470153


“The disposition of cisapride in seven healthy cats was determined following administration of either a single oral (2 mg/kg body weight) or intravenous (i.v.) (1 mg/kg body weight) dose. Cats were studied using a random crossover design. After administration of the oral capsule, maximum plasma drug concentration (Cmax) +/- standard deviation (SD) was 73.32 +/- 16.59 ng/ml, and bioavailability +/- SD was 29.0 +/- 22.6%. Following i.v. administration, extrapolated peak cisapride concentration (C0) +/- SD was 421.30 +/- 155.37 ng/ml, and clearance +/- SD was 15 +/- 0.67 ml/kg per minute. Elimination half-life (T1/2) was similar for both routes of administration (T1/2(oral) +/- SD was 5.27 +/- 3.16 hr, T1/2 (i.v.) +/- SD was 5.19 +/- 3.77 hr). Adverse effects were not observed. Based on these results, a dose of 1 mg/kg body weight per os (PO) every eight hours or 1.5 mg/kg body weight every 12 hours is expected to result in plasma drug concentrations within the therapeutic ranges established for humans.” PMID: 9358420

With our state of the art compounding lab and pharmaceutical knowledge and experience, we can compound cisapride into a flavored oral suspension.

An example of how you might prescribe follows:

**CISAPRIDE 10mg/ml**

**Flavored Oral Suspension**

Quantity 30ml

Give 2mg/kg once a day or as directed
### Canine Congestive Heart Failure
- **Pimobendan 0.5mg/ml**
  - Flavored Oil Oral Suspension
  - Quantity: 30ml
  - Directions: Give 0.5mg/kg once a day or as directed

### Canine Epilepsy
- **Potassium Bromide 250mg/ml**
  - Flavored Oral Solution
  - Quantity: 30ml
  - Directions: Give 1ml (250mg) daily or as directed

### Feline Megacolon
- **Cisapride 10mg/ml**
  - Flavored Oral Suspension
  - Quantity: 30ml
  - Directions: Give 2mg/kg once a day or as directed

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**Directions**

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Prescriber's Signature____________________________________ Refills: 1 2 3 4 5 6 7 8 9 10 11 12 NR