Antiplatelet effects and pharmacodynamics of clopidogrel in cats

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Objective—To evaluate antiplatelet effects and pharmacodynamics of clopidogrel in cats.

Design—Original study.

Animals—5 purpose-bred domestic cats.

Procedure—Clopidogrel was administered at dosages of 75 mg, PO, every 24 hours for 10 days; 37.5 mg, PO, every 24 hours for 10 days; and 18.75 mg, PO, every 24 hours for 7 days. In all cats, treatments were administered in this order, with at least 2 weeks between treatments. Platelet aggregation in response to ADP and collagen and oral mucosal bleeding times (OMBTs) were measured before and 3, 7, and 10 days (75 and 37.5 mg) or 7 days (18.75 mg) after initiation of drug administration. Serotonin concentration in plasma following stimulation of platelets with ADP or collagen was measured before and on the last day of drug administration. Platelet aggregation, OMBT, and serotonin concentration were evaluated at various times after drug administration was discontinued to determine when drug effects were lost.

Results—For all 3 dosages, platelet aggregation in response to ADP and collagen, and serotonin concentration were significantly reduced and OMBT was significantly increased at all measurement times during drug administration periods. All values returned to baseline values by 7 days after drug administration was discontinued. No significant differences were identified between doses. None of the cats developed adverse effects associated with drug administration.

Conclusions and Clinical Relevance—Results suggested that administration of clopidogrel at dosage ranging from 18.75 to 75 mg, PO, every 24 hours, results in significant antiplatelet effects in cats. (J Am Vet Med Assoc 2004;225:1406–1411)

Cardiogenic arterial thromboembolism (CATE) is common in cats and is usually associated with some form of underlying myocardial disease. Intracardiac thrombus formation is believed to result from endocardial injury and blood stasis, followed by platelet adhesion and aggregation and subsequent activation of the coagulation cascade. Emboli that originate from the thrombus are primarily composed of a fibrin network with interspersed platelets, illustrating the role that platelets play in the pathogenesis of CATE. Furthermore, platelet aggregation is altered in cats with cardiac disease, and collateral blood flow around a site of embolization is reduced in response to platelet release products such as serotonin.

Antiplatelet drugs such as aspirin would appear to be an attractive choice for the prevention of CATE in cats. However, aspirin treatment has not been reported to result in dramatic reductions in the prevalence of CATE. The thienopyridines ticlopidine and clopidogrel are newer antiplatelet drugs that exert their effects through irreversible inhibition of ADP receptors on the platelet membrane; this is uniquely different from the cyclooxygenase inhibiting effect of aspirin. Following administration, the thienopyridines must undergo hepatic transformation to 1 or more active metabolites. Therefore, in vivo and ex vivo studies, such as bleeding times and platelet aggregation studies, respectively, are required to demonstrate their antiplatelet effects. In addition, plasma concentration of the parent drug does not correlate with antiplatelet effect, so pharmacodynamic instead of pharmacokinetic studies are typically used to determine dose and dosing intervals in the species of interest. In humans, the thienopyridines have been shown to significantly reduce the risk of stroke, myocardial infarction, and vascular death, compared with aspirin therapy.

Ticlopidine has been shown to impair platelet function in cats, but its use was associated with adverse effects that precluded use of the drug in clinical patients. In addition, clopidogrel has supplanted ticlopidine in human medicine because of its equal or better clinical efficacy and more favorable safety profile. To our knowledge, the effects of clopidogrel in cats have not been determined. The purpose of the study reported here was to determine antiplatelet effects and pharmacodynamics of clopidogrel in cats. In addition, we wanted to determine whether there were any acute adverse effects associated with clopidogrel administration in cats.

Materials and Methods

Cats—Five purpose-bred, approximately 1-year-old, domestic cats (3 castrated males and 2 sexually intact females) were used in the study.

Experimental protocol—Cats were treated with clopidogrel3 at 3 dosages: 75 mg (mean ± SD, 19.2 ± 4.6 mg/kg [8.7 ± 2.1 mg/lb]), PO, every 24 hours for 10 days; 37.5 mg (9.3 ± 2.5 mg/kg [4.2 ± 1.1 mg/lb]), PO, every 24 hours for 10 days; and 18.75 mg (4.4 ± 1.3 mg/kg [2.0 ± 0.6 mg/lb]), PO, every 24 hours for 7 days. In all cats, treatments were administered in this order, with at least 2 weeks between treatments. The initial dosage (75 mg, PO, q 24 h) was chosen because it is equipotent with a dosage of 250 mg of ticlo-
pudine, PO, every 12 hours, in humans and because ticlopi
dine has consistent antiplatelet effects when administered to
cats at this dosage.19

For each treatment period, platelet aggregation in
response to ADP platelet aggregation in response to collagen,
and oral mucosal bleeding time (OMBT) were measured
before and 3, 7, and 10 days (75- and 37.5-mg dosages) and
7 days (18.75-mg dosage) after initiation of drug administra-
tion. To determine how long antiplatelet effects persisted,
platelet aggregation in response to ADP and platelet aggrega-
tion in response to collagen were measured 3, 7, and 10 days
(75- and 37.5-mg dosages) and 7 days (18.75-mg dosage)
after drug administration was discontinued; OMBTs were
measured 3 and 7 days (75- and 37.5-mg dosages) and 7 days
(18.75-mg dosage) after drug administration was discontinu-
ted. Measurement times were selected on the basis of data
from studies21,22 in humans, which indicate that the onset of
action of clopidogrel is 2 hours, with maximal effects seen
between 3 and 7 days after initiation of drug administration,
and a loss of drug effects seen between 5 and 7 days after
drug administration is discontinued.

As a measure of platelet dense granule secretion, con-
centrations of serotonin in plasma obtained following stimu-
lation of platelets with ADP and collagen were determined
prior to initiation of drug administration, the day drug
administration was discontinued, and 7 days after drug
administration was discontinued. A CBC was performed, and
serum alkaline phosphatase activity, alanine aminotrans-
ferase activity, and total bilirubin concentration were mea-
ured prior to initiation of drug administration and on the
day drug administration was discontinued to monitor for
development of adverse effects similar to those reported fol-
lowing administration of clopidogrel in humans.23

For collection of blood samples and measurement of
OMBTs, cats were anesthetized with a combination of keta-
mide (15 mg/kg [6.8 mg/lb], IM), acepromazine (0.2 mg/kg
[0.1 mg/lb], IM, but not exceeding 1 mg), and atropine
(0.054 mg, IM). This anesthetic regimen reportedly does not
affect results of platelet function tests in cats.19

Cats were examined at least twice a day during treat-
ment periods for evidence of petechiae, ecchymoses, hema-
tochezia, hematuria, and other adverse effects. The experi-
mental protocol was approved by the Purdue University
Animal Care and Use Committee. Cats were adopted by pri-
vate individuals at the end of the study.

**Determination of platelet aggregation**—Whole blood
aggregometry was used to evaluate platelet aggregation
because of the smaller volume of blood needed for each evalu-
oration. In addition, we believe that whole blood aggregome-
try is more representative of platelet aggregation in vivo
than the buccal mucosal bleeding time. This technique
has been found to be accurate and repeatable in cats.20 Briefly,
cats were anesthetized and placed in lateral recumbency, and
the lip was reflected back and held in place by gauze that was
tightly tied around the head. Spring-loaded blade cassettes
were used to create 1-mm-deep and 5-mm-long oral mucosal
incisions above the premolars or molars. The OMbT was
the time from creation of the incisions until bleeding ceased.

**Measurement of OMBT**—Because of the anatomic limi-
tations of the oral cavity in cats, the OMBT was determined,
rather than the buccal mucosal bleeding time. This technique
has been found to be accurate and repeatable in cats.20 Briefly,
cats were anesthetized and placed in lateral recumbency, and
the lip was reflected back and held in place by gauze that was
tightly tied around the head. Spring-loaded blade cassettes
were used to create 1-mm-deep and 5-mm-long oral mucosal
incisions above the premolars or molars. The OMbT was
the time from creation of the incisions until bleeding ceased.

**Measurement of platelet secretion of serotonin**—To
evaluate platelet dense granule secretion, blood used for
aggregometry was recovered after platelet aggregation and
centrifuged at 5,000 × g for 3 minutes at 4°C to separate the
cellular elements from plasma enriched with serotonin
released from platelets stimulated by ADP or collagen. The
platelet-free plasma was snap frozen in liquid nitrogen and
stored at −80°C until analyzed. Serotonin concentrations
were measured with a commercially available ELISA test kit.3

**Statistical analyses**—Descriptive statistics (mean and
SD or mean and 95% confidence interval [CI]) were calcu-
lated. Maximal inhibition of platelet aggregation was calcu-
lated by use of the following formula: ([baseline percentage
platelet aggregation – lowest percentage platelet aggregation
during drug administration]/baseline percentage platelet
aggregation) × 100. Similarly, maximal inhibition of sero-
tonin release was calculated by use of the following formula:
([baseline serotonin release – lowest serotonin release during
drug administration]/baseline serotonin release) × 100. Mean
platelet aggregation and OMBTs were compared within each
dosage group over time and between dosage groups by means
of ANOVA for repeated measures with a Bonferroni correc-
tion (overall type I error, 0.05). Pairwise comparisons were
performed with the least-significant difference method.
Standard software was used; values of P ≤ 0.05 were consid-
ered significant.

Serotonin concentrations, body weight, and results of
clinopathologic testing obtained prior to drug administra-
tion (baseline) were compared with values obtained on the
last day of drug administration with paired t tests. Standard
software was used; and values of P ≤ 0.05 were considered
significant.

**Results**

**High dosage**—During administration of clopido-
ogrel at the highest dosage (75 mg, PO, q 24 h), platelet
aggregation in response to ADP was significantly reduced, compared with baseline aggregation prior to
drug administration (Table 1). Mean ± SD maximal
inhibition of platelet aggregation was 93.3 ± 1.9%, and
mean values for platelet aggregation on days 3, 7, and
10 of drug administration were not significantly differ-
ent from each other. Platelet aggregation was still sig-
nificantly reduced 3 days after drug administration was
 discontinued, but was no longer significantly different
from baseline aggregation 7 days after drug adminis-
tration was discontinued. Similar results were seen for
platelet aggregation in response to collagen, although
mean maximal inhibition was lower (65.9 ± 3.9%).

Oral mucosal bleeding times were significantly prolonged on all days during drug administration
(Table 1), with OMBTs on days 3, 7, and 10 of drug

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administration being a mean of 5.35, 4.58, and 3.92 times baseline times, respectively. However, OMBTs on days 3, 7, and 10 were not significantly different from each other. Oral mucosal bleeding times were no longer significantly different from baseline times 7 days after drug administration was discontinued.

Sero tin release from platelets activated with ADP or collagen was significantly reduced (mean ± SD maximal inhibition, 92.4 ± 5.5% and 84.9 ± 17.1%, respectively) after 10 days of drug administration (Table 1), but was no longer significantly different from baseline release 7 days after drug administration had been discontinued.

No adverse effects were identified during drug administration. There was a significant decrease in total bilirubin concentration, compared with baseline concentration, after 10 days of administration, but concentrations for all cats were within reference limits (Table 2).

### Table 1—Results of platelet function studies in 5 cats treated with clopidogrel.

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Platelet aggregation (%)</th>
<th>Serotonin (mg/mL)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ADP</td>
<td>Collagen</td>
</tr>
<tr>
<td><strong>High</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>34.0 ± 15.4</td>
<td>43.8 ± 17.9</td>
</tr>
<tr>
<td>3</td>
<td>3.0 ± 3.7</td>
<td>16.6 ± 6.2</td>
</tr>
<tr>
<td>7</td>
<td>2.0 ± 2.3</td>
<td>14.8 ± 6.7</td>
</tr>
<tr>
<td>10</td>
<td>1.8 ± 2.5</td>
<td>13.2 ± 5.3</td>
</tr>
<tr>
<td>13</td>
<td>10.0 ± 7.6</td>
<td>19.8 ± 11.9</td>
</tr>
<tr>
<td>17</td>
<td>24.4 ± 18.4</td>
<td>40.2 ± 23.4</td>
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<tr>
<td>20</td>
<td>35.0 ± 4.6</td>
<td>48.0 ± 14.6</td>
</tr>
<tr>
<td><strong>Moderate</strong></td>
<td></td>
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<tr>
<td>Baseline</td>
<td>35.0 ± 4.6</td>
<td>49.0 ± 14.5</td>
</tr>
<tr>
<td>3</td>
<td>1.0 ± 1.0</td>
<td>13.2 ± 4.2</td>
</tr>
<tr>
<td>7</td>
<td>0.5 ± 1.4</td>
<td>12.6 ± 2.5</td>
</tr>
<tr>
<td>10</td>
<td>0.8 ± 0.8</td>
<td>15.4 ± 3.2</td>
</tr>
<tr>
<td>13</td>
<td>13.2 ± 12.3</td>
<td>22.8 ± 17.5</td>
</tr>
<tr>
<td>17</td>
<td>22.6 ± 15.0</td>
<td>45.4 ± 3.0</td>
</tr>
<tr>
<td>20</td>
<td>25.5 ± 13.1</td>
<td>45.6 ± 2.7</td>
</tr>
<tr>
<td><strong>Low</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>27.2 ± 2.9</td>
<td>47.8 ± 6.2</td>
</tr>
<tr>
<td>7</td>
<td>1.8 ± 2.5</td>
<td>19.0 ± 7.6</td>
</tr>
<tr>
<td>14</td>
<td>25.8 ± 12.8</td>
<td>43.4 ± 2.1</td>
</tr>
</tbody>
</table>

Data are given as mean ± SD. Cats were treated with clopidogrel at a dosage of 75 mg PO, every 24 hours for 10 days (high); 37.5 mg PO, every 24 hours for 10 days (moderate); and 18.75 mg PO, every 24 hours for 7 days (low). In all cats, treatments were administered in this order, with at least 2 weeks between treatments.

*Concentration of serotonin in plasma obtained after stimulation of platelets with ADP or collagen. OMBT = Oral mucosal bleeding time. ND = Not done.

**Significantly (P ≤ 0.01) different from baseline value. **Significantly (P ≤ 0.05) different from baseline value. ***Significantly (P ≤ 0.001) different from baseline value.

### Table 2—Results of clinicopathologic testing in 5 cats treated with clopidogrel.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Low dosage</th>
<th>Moderate dosage</th>
<th>High dosage</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Day 7</td>
<td>Baseline</td>
<td>Day 10</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hematocrit (%)</td>
<td>Collagen</td>
<td>ADP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>WBC (× 10^6/mL)</td>
<td>OMBT</td>
<td>Collagen</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neutrophils (&lt;10^6/mL)</td>
<td>Platelets (&lt;10^6/mL)</td>
<td>ALT (U/L)</td>
</tr>
<tr>
<td>Baseline</td>
<td>35.4 ± 1.7</td>
<td>33.2 ± 2.7</td>
<td>25.5 ± 2.6</td>
<td>25.4 ± 3.4</td>
</tr>
<tr>
<td>3</td>
<td>8.96 ± 2.87</td>
<td>9.27 ± 2.46</td>
<td>6.72 ± 1.40</td>
<td>6.17 ± 1.41</td>
</tr>
<tr>
<td>7</td>
<td>5.41 ± 2.02</td>
<td>5.02 ± 1.07</td>
<td>4.34 ± 1.24</td>
<td>3.56 ± 0.68</td>
</tr>
<tr>
<td>10</td>
<td>390.5 ± 32.3</td>
<td>396.0 ± 97.2</td>
<td>393.5 ± 62.8</td>
<td>383.4 ± 72.5</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>360.2 ± 17.5</td>
<td>362.8 ± 18.5</td>
<td>365.4 ± 20.6</td>
<td>368.6 ± 22.5</td>
</tr>
<tr>
<td>ALP (U/L)</td>
<td>56.0 ± 11.7</td>
<td>57.2 ± 13.5</td>
<td>58.4 ± 15.7</td>
<td>59.6 ± 17.9</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>0.22 ± 0.04</td>
<td>0.18 ± 0.13</td>
<td>0.20 ± 0.00</td>
<td>0.10 ± 0.00</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>4.6 ± 1.3</td>
<td>4.6 ± 1.3</td>
<td>4.2 ± 1.1</td>
<td>4.3 ± 1.0</td>
</tr>
</tbody>
</table>

ALT = Alanine aminotransferase. ALP = Alkaline phosphatase.

**Significantly (P < 0.05) different from baseline value. See Table 1 for remainder of key.**
administration was discontinued. Serotonin release from platelets activated with ADP or collagen was significantly reduced (mean maximal inhibition, 94.1 ± 4.0% and 88.4 ± 12.6%, respectively) on day 10 of drug administration (Table 1), but was no longer significantly different from baseline release 7 days after drug administration had been discontinued. None of the cats developed any adverse effects associated with drug administration, and results of clinicopathologic tests performed after 10 days of drug administration were not significantly different from baseline values except for a significant decrease in mean total bilirubin concentration (Table 2). However, for all cats, total bilirubin concentration was within reference limits. Platelet function test results when cats were treated with clopidogrel at the moderate dosage were not significantly different from results obtained when cats were treated with clopidogrel at the high dosage.

**Low dosage—**Platelet function test results during administration of clopidogrel at the low dosage (18.75 mg, PO, q 24 h) were comparable to those obtained during administration at the high and moderate dosages. Platelet aggregation (mean ± SD maximal inhibition of platelet aggregation in response to ADP or collagen, 93.4 ± 9.2% and 60.3 ± 15.9%, respectively) and serotonin release (mean maximal inhibition of serotonin release from platelets activated with ADP or collagen, 90.4 ± 7.3% and 86.7 ± 22.5%, respectively) were significantly decreased, and OMBT was significantly increased (mean of 3.95 times baseline time) after clopidogrel had been administered for 7 days, but values were no longer significantly different from baseline values 7 days after drug administration was discontinued (Table 1). None of the cats had any adverse effects associated with drug administration, and results of clinicopathologic tests performed after clopidogrel had been administered for 7 days were not significantly different from baseline values 7 days after drug administration was discontinued (Table 2). Platelet function test results when cats were treated with clopidogrel at the low dosage were not significantly different from results obtained when cats were treated with clopidogrel at the high or moderate dosage.

**Discussion**

The thienopyridines ticlopidine and clopidogrel induce irreversible inhibition of ADP_2Y12_ receptors on the platelet membrane and, in people, have more potent antplatelet effects than aspirin. Additionally, they inhibit ADP-induced conformational change of the glycoprotein IIb/IIIa receptor complex, reducing binding of fibrinogen and von Willebrand factor. There is also evidence that the thienopyridines have vasomodulatory effects, impair myointimal proliferation in vascular smooth muscle, and, possibly, have some thrombolytic effect in people. Whereas aspirin acts as an indirect antithrombotic agent, only interfering with secondary platelet aggregation, the thienopyridines are direct antithrombotic agents in people, inhibiting primary and secondary platelet aggregation in response to multiple agonists as well as inducing dramatic prolongation of bleeding times and inhibiting secretion of platelet release products such as serotonin. These compounds do not exert any direct antplatelet effects but must undergo hepatic metabolism to 1 or more active metabolites. In humans administered clopidogrel long-term, the parent compound and primary metabolite are excreted in the urine (41% to 46%) and feces (35% to 57%).

Results of the present study indicated that administration of clopidogrel caused significant impairment of platelet function in cats. When cats were given clopidogrel at the moderate (37.5 mg, PO, q 24 h) or high (75 mg, PO, q 24 h) dosage, platelet aggregation in response to ADP or collagen was significantly reduced and the OMBT was significantly increased after 3 days of drug administration, and these alterations continued throughout the 10-day drug administration period. Serotonin release from platelets was significantly suppressed after 10 days of drug administration. Similarly, when cats were given clopidogrel at the low dosage (18.75 mg, PO, q 24 h), platelet aggregation and serotonin release were significantly decreased and OMBT was significantly increased after 7 days of drug administration. At all dosages, values for platelet aggregation, OMBT, and serotonin release were no longer significantly different from baseline values 7 days after drug administration had been discontinued. No significant difference in results of platelet function tests was found among dosages.

Platelet release products, specifically serotonin, appear to play a primary role in the clinical signs associated with CATE, and in the present study, clopidogrel administration resulted in significant reductions in serotonin secretion from activated platelets. Previous studies have found that clopidogrel reduces the contractile response of pulmonary and femoral arterial ring preparations to multiple vasoconstrictive agents, including serotonin. Thus, it is possible that administration of clopidogrel, through its anti-serotonin effects, could result in less severe clinical signs of CATE, even if thromboembolization itself was not prevented.

Adverse effects associated with clopidogrel administration in humans have been identified. In a study of 9,599 patients, the discontinuation rate among those taking clopidogrel was 11.94%; however, this was not significantly different from the discontinuation rate for those taking aspirin (11.92%). Clopidogrel was associated with a significantly lower prevalence of adverse gastrointestinal tract events than was aspirin (27.14% vs 29.82%) but with a significantly higher prevalence of adverse dermatologic events (15.81% vs 13.08%), including pruritus and rash. In subgroup analyses of adverse gastrointestinal tract events, only diarrhea was more common with clopidogrel than with aspirin (4.46% vs 3.36%). There was no significant difference in prevalence of hemorrhagic events (9.27% vs 9.28%), and gastrointestinal tract bleeding was significantly less common with clopidogrel (1.99% vs 2.66%). Neutropenia and thrombocytopenia, both of which have been identified in patients treated with ticlopidine, were rare in patients treated with clopidogrel (0.10% and 0.26%, respectively), and their prevalence in patients taking clopidogrel was not significantly different from prevalence in patients taking aspirin.
(0.17% and 0.26%). Thrombotic thrombocytopenic purpura is a severe and often fatal adverse event that has been associated with ticlopidine administration, but only 11 cases have been reported (out of > 3 million patients treated), and there is some debate whether all of these cases were actually caused by ticlopidine, as some patients had clinical conditions or were receiving other drugs known to cause thrombotic thrombocytopenic purpura. Additionally, the expected incidence of idiopathic thrombotic thrombocytopenic purpura in the general population is 3.7 per million persons. Hepatocellular injury and cholestasis have also rarely been associated with clopidogrel administration in people. Adverse gastrointestinal tract events including vomiting, nausea, and weight loss have been associated with ticlopidine administration in dogs and cats. None of the cats in the present study had any adverse clinical effects, bleeding tendencies, or alterations in clinicopathologic data during any of the dosing periods. However, the period of drug administration was short in this study; and a longer clinical safety trial is needed to determine the safety of long-term clopidogrel administration in cats.

We were unable to determine the minimum effective dose of clopidogrel in the present study. In humans, the recommended maintenance dosage for clopidogrel is 75 mg, PO, every 24 hours. At this dosage, maximal inhibition of ADP-induced platelet aggregation ranges from 40% to 60% and maximal prolongation of bleeding time is 1.5 to 2.0 times the baseline value. There were no significant differences seen between dosages in this study, and the lowest dosage (18.75 mg, PO, q 24 h) resulted in mean maximal inhibition of ADP-induced platelet aggregation of 93.4% and a 3.9-fold prolongation of OMBT. Thus, it appears that a lower dosage could be used to provide adequate platelet inhibition. However, clopidogrel is currently only available in the United States as 75-mg tablets. Although these tablets are not exceedingly small, splitting tablets into fractions smaller than quarters was not realistic, and administration of smaller dosages would have required compounding of the drug. Currently, 30 tablets of the commercially available form of clopidogrel cost approximately $120. Therefore, administration to a cat at a dosage of 18.75 mg, PO, every 24 hours, would cost approximately $30/mo. Even given the additional cost associated with compounding, administration of smaller dosages may be less expensive. Thus, additional studies to determine the minimum effective dosage of clopidogrel in cats are warranted.

Results of the present study suggest that it may be reasonable to administer clopidogrel at a dosage of 18.75 mg, PO, every 24 hours, in cats to prevent CATE and to determine the minimum effective dosage.

References