Use of clomipramine to treat ritualistic stereotypic motor behavior in three dogs

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- Stereotypic behavior in dogs may be a sign of obsessive-compulsive disorder.
- Treatment with clomipramine, along with behavioral modification and administration of other drugs, may be helpful in ameliorating the inappropriate behavior.
- Considerations in the diagnosis and management of such behavioral problems include exclusion of other causes of clinical signs, treatment of secondary complications of the behavioral disorder, and consideration of adverse effects and human abuse potential when prescribing psychotropic drugs.

A typical, stereotypic motor and locomotor behavior was observed in 3 dogs. Clinical signs varied in each dog: apparently sudden onset of circling, vocalization, and snapping; circling associated with pica and aerophagia; and gradually intensifying, purposeless locomotor behavior stimulated by bright light. The behavior in all dogs was interfering with normal interaction between the clients and the dogs.

A sexually intact male mixed-breed dog (dog 1), weighing 29 kg and estimated to be 1 to 3 years old, was evaluated for an apparently sudden onset of spinning and circling behavior. The spinning (fast, airborne, tight circles) and circling (slow, low, on the ground) was always to the right (clockwise). The behavior had started 4 weeks prior to evaluation, approximately 2 weeks after the dog had been brought into the clients' house. The dog had been a stray and had been fed in the parking lot of the clients' business for more than a month prior to being adopted. None of the clinical signs were apparent to the clients prior to adoption. History of apparent intoxication, injury, or illness was lacking. The dog had been dipped for flea infestation (product unknown) a few days before being brought into the house.

At evaluation, the spinning behavior, once started, was continuous. Bouts of spinning and circling were interrupted or terminated only by physical/manual restraint or collapse of the dog. Frequency was variable, but the minimal number of times that the dog experienced a bout of spinning was twice daily. The clients (husband and wife) were unable to define shortest and longest bouts; every time that the dog stared at its paws, it would spin until exhausted. The shortest bout witnessed at the veterinary hospital was 20 minutes, and was terminated by physically restraining the dog. Spinning bouts were preceded by staring at first the left, then the right forepaw and by high-pitched barking at the paw being observed. This behavior then would progress through crying, yelping, sucking, grabbing, and gently biting both forepaws. As the chewing and sucking progressed, the digits flexed, limbs became rigid, the lumbar spine flexed and the dog would spin. When physically restrained from spinning and chewing, the dog whimpered; wrinkled its peri orbital and cranial skin, lips, and ears; and stared at its paws.

The husband was able to control the dog's behavior by a combination of physical restraint and frequently reinforced verbal commands; however, the dog would exhibit the facial expressions described and apparently desire to engage in the behavior. If placed in the dog pen, the dog would circle and spin, characteristically starting by pacing the length of the kennel. The behavior was observed more frequently in the wife's presence, as reported by the clients and observed by the author; the intensity of the spinning and circling was not different whether or not the wife was present. Husband and wife were effusive in the treatment of the dog; the husband was home during the day with the dog, but neither spent more time with the dog. If the dog had been sleeping and awoke in the presence of the husband, the behavior did not start unless the wife entered the room.

The clients reported that when dog 1 was loose outdoors, it ran and behaved normally, but resumed spinning when it stopped running. When the dog was behaving normally, the other 3 canine housemates on the farm interacted with it, but they avoided it in the kennel when it spun.

Despite being fed large amounts of high-quality dog food, dog 1 had continuously lost weight (approx 5 kg) since adoption. Physical examination

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revealed areas of alopecia and erythema on the distal part of the forelimbs, primarily between the digits at the edges of the pads, which were moist and erythematous. Superficial cervical lymph nodes were large, and popliteal nodes were slightly large. When flexed, the neck became rigid, digits of the forelimbs flexed, and the dog yelped. The same response was elicited to a lesser extent by extension of the neck. The forepaws were hyperesthetic; the dog cried and tried to place them under its body during manipulation. Signs of pain were not apparent on deep palpation of the thoracic and lumbar spine, although pressure at the thoracolumbar junction elicited circling and complementary behaviors. Heart and respiratory rates were substantially increased when circling (140 beats/min and 50 breaths/min, respectively), but were within reference range when the dog was resting (80 beats/min and 30 to 40 breaths/min, respectively).

Ophthalmic and cranial nerve examination did not reveal anisocoria, retinal hemorrhages, strabismus, or nystagmus. Neurologic examination 1 week later revealed grossly normal myotatic reflexes and cranial nerve and postural responses. At that examination, flexion of digits and back, as described earlier, was not part of the sequence of the behavior. The left forepaw appeared hyperesthetic.

After the physical examination, during which the clients were not present, the dog ceased all ritualistic activity for 20 minutes, became attentive and friendly, and appeared clinically normal. The dog had exhibited the spinning and circling prior to the physical examination, but not during it when he was lightly restrained. After 20 minutes, the husband was invited back into the room, and the dog remained normal and greeted him in a friendly manner. When the wife returned 10 minutes later, the dog greeted her enthusiastically and began to spin within 30 seconds of her arrival. If the dog was left alone in the examination room and observed behind one-way glass, the behavior continued, accompanied by pacing the length of the room. The dog could be momentarily distracted by a rap on the glass, but resumed the behavior within seconds.

Fecal examination revealed infection with Toxocara and Trichuris spp. Results of 2 urine metabolic evaluations, separated by 3 weeks, were normal. The CBC revealed mild normochromic anemia (PCV 28%), and eosinophilia (1,804 cells/μl) that was probably associated with the parasites. Serum biochemical analysis revealed mild, unexplained hyperphosphatemia (5.1 mg/dl; reference range, 2.0 to 4.5 mg/dl).

Ehrlichia and Borrelia titers were lacking. Serum IgM distemper titer was lacking; serum IgG titer was 1:25. The CSF distemper titers were lacking. The initial Rocky Mountain spotted fever (RMSF) titer was 1:64; a repeat titer 2 weeks later was 1:16. Radiography of the distal part of the left forelimb did not reveal abnormalities. Analysis of CSF fluid, obtained with the dog anesthetized, did not reveal erythrocytes or precipitated protein, and the number of nucleated cells was within reference ranges. A few lymphocytes, single subarachnoid lining cells, and several small macrophages were detected; the latter were interpreted by the pathologist service and the neurologists to be indicative of cell destruction. These findings could have been of infective organ, given the RMSF results. Spinning and circling did not decrease after injection of naloxone (dosage, 0.015 mg/kg of body weight, iv).2

Differential diagnoses in dog 1 included behavioral and medical conditions which initially were metabolic disease, including hepatoencephalopathy; primary neurologic disease, including encephalitides; attention-seeking behavior; anxiety; hyperactivity; aberrant endorphin metabolism; obsessive-compulsive disorder; and primary brain disease with cerebral thalamic lesions, probably with a right-sided focus. The latter would be progressive or static, given the dog's history. Because dog 1 did not have irregularities in hepatic-associated enzyme activities and because the condition did not appear to be affected by diet or feeding schedule, hepatoencephalopathy was considered unlikely. Dog 1's behavior was consistent with a diagnosis of anxiety, but was much more exaggerated than has been reported in such cases.

Neurologic lesions, primarily encephalitides, have been attributed to tick-borne diseases; however, these lesions are usually mentation changes that affect cognitive abilities during the active phase of the disease, unlike in the dog reported here. Whether this dog was recovering from infection was not clear; a 1:254 titer is required for a presumptive diagnosis of RMSF on the East Coast. Because the second RMSF titer did not have the fourfold increase classic of active disease, the behavioral signs were unlikely to have been caused by active disease. Completely excluding the effects of subclinical or convalescent RMSF on the behavioral signs was impossible prior to examination. However, treatment with antimicrobial medication should be efficacious, whereas treatment with primarily psychotropic medication should have little effect without an antimicrobial agent.

Primary brain disease remained a diagnostic consideration. However, results of neurologic and ophthalmic examinations were within normal limits and were otherwise inconsistent with a diagnosis of primary brain disease. Circling behavior is not a usual sign of spinal cord disease.

Stereotypic behavior by dogs may be a sign of obsessive compulsive-disorder. Obsessive-compulsive disorder in human beings is characterized by stereotypic ritualistic behavior that interferes with the patient's ability to function normally.

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Much of this behavior may be rooted in anxiety or neurologic disorders.  
Obessive-compulsive and anxiety disorders have not been extensively recognized in or characterized for companion animals, although references for zoo animals are numerous. Large numbers of stereotypic behaviors exhibited by animals involving vocalization, locomotion, ingestion, and grooming may be candidates for this classification of disorder. This description characterized dog 1; accordingly, a working diagnosis of obsessive-compulsive behavior was made.

For dog 1, the reported behavior occurred in the absence of the owners and followed a pattern inconsistent with attention-seeking behavior, although the wife, but not the husband, was able to elicit the behavior by greeting the dog. Behavior modification that included ignoring the dog when it was spinning and stopping the wife from greeting the dog was unsuccessful in stopping the behavior. The owners of dog 1 were instructed to use behavioral modification so that the dog would not associate the stereotypic behavior with favorable outcomes. They were to reward the dog's calm behavior with affection and treats and to ignore its circling by leaving the room. When the dog came into their vicinity again, they were to ask the dog to lie down and stay before giving it affection. These modifications were intended to rule out any role, however unlikely, of attention-seeking behavior in the ritualistic sequence. In addition, pending normal hepatic-associated enzyme results, the dog was treated with amitriptyline HCl (50 mg, PO, q 12 h), a tricyclic antidepressant that is efficacious in cases of anxiety in dogs. Tricyclic antidepressants act primarily by inhibiting serotonin reuptake and relieve primary anxiety or that secondary to disease. Individual responses to selective serotonin reuptake inhibitors vary greatly; hence, treatment with psychotropic medication usually involves adjustment according to response and more prolonged treatment than that involved for antimicrobial agents.

Intestinal parasites were treated with butamisol HCl and mebendazole. Because of the life cycle of these parasites, much of the dog's problems with its forepaws may have been related to local irritation. After report of the initial RMSF titer of 1:64, treatment with doxycycline HCl (500 mg, PO, q 24 h) was immediately begun and was continued for 21 days. Neither of these treatments had any effect on the behavior.

The attempt to block the behavior with naloxone was unsuccessful. If the dog were stimulating endogenous endorphin production by spinning and circling, as has been postulated, a challenge with a narcotic antagonist should have blocked circulating and CNS endorphins and stopped the behavior. Lack of efficacy suggested that the primary defect did not involve aberrant endorphin metabolism.

After the dog had had 1 week of amitriptyline treatment, the clients reported that the dog's behavior had not improved, but rather, the dog had become worse after steady-state blood drug concentrations had been reached (in 3 to 4 days). When corrected, the dog would circle, rather than spin, but the behavior was difficult to interrupt completely. This response was suggestive of some antianxiety effect of the amitriptyline being experienced by the dog because the dog attempted to control its behavior by modifying that form. Such modification was in response to corrections from the clients, suggesting that during antianxiety treatment, the dog was aware that its behavior was inappropriate or undesirable. These observations agreed with the dog's anxious facial expressions, vocalization, and behavior when restrained. After 3 weeks, treatment with amitriptyline was stopped.

Dog 1 then was treated with another tricyclic antidepressant, clomipramine HCl (25 mg [equivalent to 1 mg/kg], PO, q 12 h) for 14 days, after which the dosage was increased by 1 mg/kg every 2 weeks until it reached 3 mg/kg or 100 mg every 12 hours by week 5 (equivalent to the maximal recommended dosage of 200 mg/d), or less, if the signs were controlled. Treatment at the maximal dosage was continued until week 8 for an assessment of efficacy.

By week 4 (clomipramine dosage, 50 mg, q 12 h), the behavior had largely abated and dog 1 was attentive when addressed, even if mildly spinning. The dog had gained 2 kg; the stereotypic behavior was less pronounced and totally interruptible. If called and interrupted while spinning, the dog would solicit attention and would play with a ball interactively. The dog sometimes would spin if ignored. This behavior was aborted by teaching the dog to lie down with its head and chin on the floor ("head down") for a treat. Such behavior modification had not been successful prior to treatment with clomipramine. The clients then could use this command to abort episodes.

By week 8, the clients noticed distinct changes in the dog's tail carriage that allowed them to predict when the dog would start to spin; they then used the "head down" command to abort the onset of the spinning. The clients reported that stopping the spinning by calling the dog if they missed the tail cue was easy. When the dog spun, it still oriented and tilted its head to the right. During these episodes, the dog no longer appeared rigid, and the vocalizations had almost totally stopped. Clomipramine dosage was increased to 50 mg above the maximal daily dosage, in an attempt to abort these episodes.

5Elavil, Stuart Pharmaceuticals, Wilmington, Del.
6Styquin, Miles Inc, West Haven, Conn.
7Lederle Laboratories, Pearl River, NY.
8Anafranil, Ciba-Geigy Corp, Summit, NJ.
residual behaviors. After 3 weeks this attempt was unsuccessful, and the dosage was decreased to 100 mg, po, every 12 hours.

For the next 2.5 months, dog 1 was maintained on clomipramine at the maximal dosage, with continuing decrease in the frequency and severity of the stereotypic behavior and continuing improvement in the ease with which this behavior was interrupted. The clients reported that dog 1 had increased frequency of normal social behavior with the other dogs, and an increase in affectionate and attentive behaviors. Clients continued to use the “head down” command to dissuade the dog from engaging in the behavior or to abort the behavior, with great success. After maintaining treatment at this therapeutic dosage for 3 months, clomipramine was gradually discontinued because of expense and difficulty in obtaining the drug. The owners reported that they had not noticed any worsening of the behavior and were still able to control any ritualistic outburst with behavior modification. During a 1.5-hour reexamination visit 1 month after stopping the clomipramine treatment, the dog progressed from being calm and outgoing to exhibiting circling behavior. The behavior gradually changed from broad circles, to staring and snapping at the paws and vocalization. This behavior was interruptible by play, attention, calling the dog, or the “head down” command. Once the dog was told that they were going home, the behavior ceased and the dog appeared normal. The dog was castrated 2 months later and inappropriate motor activity or vocalization was not observed during the short hospital stay.

Evaluation 6 and 12 months later revealed periodic stereotypic behavior, as described, when the dog was in an unfamiliar location or circumstance. All such behavior was controlled and aborted by the “head down” command and reassuring interaction. The clients avoided subjecting the dog to unfamiliar circumstances. As dog 1 became able to control its motor activity, it has become friendlier with the other dogs in the household. Cardiac or physiologic abnormalities resulting from medication have not been detectable.

A 4-year-old castrated male 50.5-kg Rottweiler (dog 2) was evaluated for unusual motor behavior that was more common at night, but could be induced during most times of the day if someone scratched the dog’s back. At the start of these bouts, the dog would extend its neck and began to lick its lips. The dog then would make appetitive movements with its lips and explore the room, snuffling. During this exploration any object that the dog encountered was ingested, including plastic, pens, paper, paper clips, and squeaking toys. If the dog’s hair had been exfoliated when its back was scratched, the dog would immediately ingest that hair. During the bout, the dog appeared to become progressively more excited about pacing the room, circling, snuffling at the ground, and ingesting anything found.

The bouts usually lasted from 10 minutes to > 4 hours; the longest bout lasted almost 24 hours. The most prolonged bouts developed in the middle of the night. The shorter bouts were observed during the day; at the first sign of ingestive movements, the owners would restrain the dog. The frequency of the bouts was variable; sometimes the dog experienced them daily, yet sometimes bouts were separated by as much as a month. If the dog was interrupted early in the course of the behavior and encouraged to perform another behavior (relax, chew on a nylon bone), the bout could be aborted. The longer bouts developed at night; this may have correlated with lack of early interruption because the clients were asleep. The clients usually awoke when the pacing and snuffling had intensified. At that point, physical restraint could not abort the dog’s motor and vocal behavior; it would whine, become agitated, rock back and forth, and continue its ingestive movements, which then would be confined to apneophagia. The clients then would administer acepromazine to sedate the dog. During every bout, the dog became apneophagic and flatulent.

In the 18 months that the dog had exhibited this behavior, it had had 2 gastrostomies for foreign body removal. During the last gastrostomy, gastroscopy was performed as prophylaxis for gastric dilatation and torsion. Results of a previous neurologic examination had been grossly within normal limits, and the tentative diagnosis of psychomotor epilepsy had been made. The dog had been treated for approximately 1 year with phenobarbital (130 to 230 mg, po, q 12 h) without success. The dose had been increased after initial blood concentrations (≤ 7.5 μg/ml) were deemed too low for therapeutic effect. Blood phenobarbital concentration increased to within therapeutic range at the higher dose, but the behavior did not abate.

The stereotypic behavior had started when the dog was about 2.5 years old, approximately 6 months after the dog was adopted. One month after adoption and prior to development of the stereotypic behavior, mild dominance aggression and food-related aggression had been diagnosed in this dog. Both components had been successfully managed by use of behavior modification and removal of real bone and rawhide from the dog’s diet. Whether the food-related aggression and the motor behavior associated with ingestive movements were related was not clear; the dog was never aggressive during the motor events.

Physical examination in dog 2 was unremarkable. The CBC and earlier biochemical panels obtained prior to the 2 surgeries and prior to the treatment discussed earlier were mostly within reference ranges; serum alkaline phosphatase activity was slightly high (193 U/L; reference range, 35 to 169 U/L). During phenobarbital treatment, blood phenobarbital concentrations were monitored and found to be low or in the therapeutic range; adverse effects were not detected as a result of this treat-
ment. Blood lead concentration was within acceptable limits (6 μg/d; reference range, ≤ 40 μg/d). Repeated urine metabolic evaluations did not reveal abnormalities. Histologic examination of a gastric biopsy specimen, obtained during the second gastrotomy, revealed eosinophilia; on the basis of a presumptive diagnosis of eosinophilic gastritis, dog 2 was fed a special diet, without resultant behavioral change.

Many of the same differential diagnoses discussed for dog 1 were applicable for dog 2. The lack of response to phenobarbital in dog 2, coupled with the sedation that the dog experienced when treated with acepromazine, lowered the likelihood that epilepsy was the cause of the behavior. Worsening of the stereotypic motor behavior as each bout progressed suggested that a primary or secondary anxiety component may have been operative. Dog 2 had locomotive, ingestive, and grooming behaviors, all of which have been associated in stereotypic contexts with anxiety behavior in and with the subset of anxiety disorders classified as obsessive-compulsive disorders.

The owners of dog 2 were taught to ask the dog to lie down and relax at the first signs of any unusual buccal or ingestive behavior. Rewarding the dog for physical signs of relaxation was emphasized. At the first signs of any panting, the dog was to lie down with its head down. Dog 2 also was treated with amitriptyline HCl (1 mg/kg, po, q 12 h). After 10 days, the dog’s nocturnal behavior had not changed. After an additional 10 days of treatment with the dose doubled to 100 mg (2 mg/kg, q 12 h), the dog’s condition was still unchanged and the drug was stopped.

During the next reexamination, naloxone was given IV while the dog was engaged in a bout of motor and ingestive activity; the behavior did not change. Dog 2 then was treated with clomipramine HCl; the dosage was 1 mg/kg, po, every 12 hours, and was increased to 3 mg/kg over 6 weeks. Eight weeks after clomipramine treatment was begun, the dog was sleeping though the night. Breakthrough nocturnal bouts were frequent at first, but became few after a few months of treatment. These nocturnal bouts were easily interrupted and seldom necessitated use of tranquilizers. The aerophagia and flatulence decreased dramatically.

Dog 2 was maintained on clomipramine for 18 months. The only change in the dog’s serum biochemical values were a slight increase in serum creatinine concentration (1.5 mg/dl; reference range, 0.1 to 1.2 mg/dl; previous value, 1.0 mg/dl). The increase in serum creatinine may have been attributable to clomipramine treatment, but was considered nondiagnostic because the dog did not have clinical signs of renal impairment.

Dog 2 has had periodic week-long recurrences of its former behavior, which seem to be associated with 2 events: the examination periods of one of his owners (who is a student), and episodes of seasonal atopy, during which the dog is agitated when pruritic. That client noticed that the episodes were less violent if she did not change her schedule, remained calm, and slept regular hours. Treating the dog’s atopy with antipruritic medication (hydroxyzine HCl or doxepin HCl) greatly reduced, but did not ablate, the intensity and frequency of these events. During the first recurrence, controlling the dog’s behavior by increasing the dosage of clomipramine to 300 mg/d was attempted; this change paradoxically made the dog more volatile and treatment with this higher dosage was discontinued after 3 weeks. After 18 months of maintenance treatment without major motor events for months, the owners decided to gradually wean the dog from the clomipramine and rely on behavior modification to control the dog. Six weeks after the last dose of clomipramine, dog 2 had experienced total recrudescence; gradual return to maintenance treatment with clomipramine again controlled the behavior.

An 11-month-old castrated male 25-kg English Bulldog (dog 3) was evaluated because the owner had observed the dog seeking and following lines (such as those on football fields or highways) and attacking bright lights or reflective surfaces. This behavior began at 5 months of age; over 3 to 4 months, it gradually worsened until such episodes became daily. By the time of evaluation, every time dog 3 encountered any of the discussed situations the dog would become very active and vocal and try to attack and grab the reflective surface or line. It would follow the lines until exhausted. If the dog was pursuing a light, it would walk into furniture or other objects in its path. This stereotypic motor behavior lasted from 20 minutes to several hours. During the execution of this behavior, the dog was unresponsive to most external stimuli (excluding reflective surfaces) and did not respond to vocal commands, mild physical restraint and correction, or distraction. The dog had to be removed from the circumstances surrounding the event and not stimulated for a few hours to return to normal behavior.

At all other times, dog 3 was placid. The dog had attended obedience school at 7 months of age; although it learned the basic commands, the owner reported that the dog appeared frightened most of the time and stayed near her.

Physical examination in dog 3 was unremarkable, except for a slightly inflamed left hip with signs of pain that had resulted from a collision with a stationary object. Results of an ophthalmologic examination, recommended because the dog would walk into stationary objects during stereotypic events and appeared stimulated only by bright or reflective surfaces, were within normal limits.

In dog 3, CBC and biochemical panel were

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*Sinequan, Roerig Division, Pfizer Inc, New York, NY.
within reference limits. Repeated urine metabolic evaluations revealed a slight increase in glutamine and taurine concentrations that appeared uncorrelated with liver disease and remained unexplained. Because the dog was growing normally and the blood tests were unremarkable, the clients declined an ammonia tolerance test for the dog.

A month of treatment with amitriptyline HCl (1 mg/kg, PO, q 12 h for 10 days; then 2 mg/kg, PO, q 12 h for 20 days)\(^{27,29}\) did not affect the behavior in dog 3, nor did behavior modification using relaxation alone. Two months after initiating treatment with clomipramine HCl (1 mg/kg, PO, q 12 h, increasing to 3 mg/kg, PO, q 12 h) the dog had improved. If it saw bright lights or reflective surfaces, it always would start to become agitated, but could be distracted and would return to normal behavior. The clomipramine dosage was adjusted over time to be maintained at 3 mg/kg as the dog grew.

Six months after starting the clomipramine treatment, concomitant with the arrival of a new infant in the household, dog 3 began to experience episodes of nocturnal motor and vocal activity from which it was difficult to wake; the owners reported that during such times, the dog's bark was different. In addition, the dog began to seek out light again. Neurologic examination at that time was unremarkable. Such breakthroughs are not unusual in human beings treated for obsessive-compulsive disorder\(^{33,34}\); in some cases, combination treatment with fluoxetine\(^1\) or buspirone\(^1\) is beneficial.\(^{33,35}\) Hence, 6 months after the initial clomipramine treatment, adjunct treatment with fluoxetine (25 mg, PO, q 24 h) was begun.\(^{27,29}\) After 1 month, the dog's locomotor behavior was increasing in frequency and the nocturnal activity and vocalizations were increasing in intensity. The dog was weaned off the fluoxetine over 2 weeks; by 3 weeks, the behavior had returned to the prefluoxetine levels.

Five months later, this behavior had increased such that dog 3 was not sleeping through the night. Buspirone (10 mg, PO, q 24 h)\(^{27,29,36,38}\) was added to the clomipramine, in an effort to control this behavior. One month after starting the buspirone treatment, the dog had experienced some, but not complete, remission in the nocturnal activity. The clomipramine dosage was increased to 100 mg, PO, every 12 hours, which was slightly more than 3 mg/kg, in an effort to magnify this effect. After 1 month, this treatment appeared to have stabilized the dog's condition.

Five months later, while being treated with the drug combination, the dog was again beginning to experience breakthrough locomotory activity and vocalization. Buspirone treatment was withdrawn, without concomitant behavioral change. Ad-

juvant treatment with carbamezepine\(^40,41\) (4 mg/kg, PO, q 12 h) for 3 weeks did not alleviate these clinical signs, but the client complained that the dog was more excitable after the fifth day of carbamezepine treatment. After 3 weeks, carbamazepine treatment was discontinued.

Obsessive-compulsive disorder affects at least 2% of the human population, and this is believed to be an underestimate.\(^{41,43}\) At least one form of obsessive-compulsive disorder has a familial genetic component; however, most instances of human obsessive compulsive disorders appear to be sporadic. Obsessive-compulsive disorders are poorly defined in companion animals. For a disorder to be considered obsessive-compulsive in human beings, it must be ritualistic and sufficiently invasive, cognitively or physically, to interfere with normal function. These criteria are applicable to canine behavior that includes stereotypic, ritualistic circling, spinning, and pacing; some howling; some hallucinatory and ingestive behaviors; and many self-mutilation/grooming disorders such as acral lick granuloma.\(^{0,11,14,23,44,46}\) The development of specific breeds and the practice of inbreeding within those breeds suggests that the incidence of obsessive-compulsive disorders in dogs could be higher than the 2% reported for human beings.\(^{41,43}\) Accordingly, a diagnosis of obsessive-compulsive disorder should be a consideration for animals exhibiting such forms of behavior.

Human obsessive-compulsive disorder has been postulated to be caused by aberrant serotonin metabolism.\(^{30,47}\) Accordingly, treatment has been directed at affecting serotonergic metabolism; pharmacological agents used for treatment are fairly specific and affect subclasses of serotonin receptors. In the dogs of this report, clomipramine, a selective serotonin-reuptake inhibitor, suppressed and controlled the inappropriate behavior, but did not totally eliminate it; this response is not uncommon.\(^{48,51}\) Rather than fully suppressing all symptoms in humans beings, such drugs act to allow many patients to use behavioral modification to fully control the behavior associated with their particular syndrome. In human patients who stop taking medication for obsessive-compulsive disorder, relapse is common; this would be expected if the abnormality is aberrant serotonin metabolism in the region of the caudate nucleus/limbic system.\(^{52}\) The dogs of this report did not fully relapse, but were more reactive in any anxiety-provoking situation when deprived of clomipramine. The owners of dog 1 fully understand the importance of a controlled, nonprovoking environment and continue behavioral modification to control this dog's stereotypic motor behaviors. Dog 1 may again require medication to control its behavior. In similar cases, magnetic resonance imaging and histologic examination of brain tissue also might be beneficial. The owners of dog 2 intend to continue the dog's clom-

\(^{1}\)Prozac, Eli Lilly & Co, Indianapolis, Ind.
\(^{2}\)BuSpar, Mead Johnson Laboratories, Division of Bristol-Myers Squibb Co, Princeton, NJ.
\(^{3}\)Tegretol, Ciba-Geigy Corp, Summit, NJ.
ipramine treatment for life. The owners of dog 3 continue to pursue amelioration of the behavior through behavioral, pharmacologic, and environmental modification.

Behavioral diagnoses of attention-seeking behavior, anxiety, and hyperactivity can be difficult to distinguish and may share features in common with other disorders, including obsessive-compulsive disorder. Boredom is an often-touted and little-demonstrated cause for such behavior. Dogs that are confined, receive little human attention or canine interaction, and have minimal stimulation and exercise may spin or chase their tails because they are bored. In such cases, increased stimulation through exposure to human or canine companions, toys, music, exercise, or rooms with views of activity should diminish or stop this behavior. Boredom was not implicated in the problems in the dogs of this report.

Some animals with anxiety may chase their tail, chew, and suck on themselves or fabrics. Previous injury to an extremity may result in chasing or attacking that region. These animals are often neurologically normal. Repetitive vocalization and self-mutilation also can develop in separation anxiety; affected animals may panic if they are left alone or are separated from a particular human being. Other canine and feline companionship is usually not sufficient to stop the behavior in separation anxiety. Generalized, nonspecific anxiety may be observed with or without the owner, but may not be evident if the animal is engaged in a competing behavior.

Animals learn quickly that if they are not receiving the desired attention from positive, quiet behavior, they can obtain it from behavior that owners find less attractive (e.g., jumping, barking, howling). Attention-seeking behavior includes subtle acts, such as leaning against a human being to passively solicit attention, or rambunctious behavior like jumping and pawing to actively solicit attention. Withdrawing of attention usually results in escalation of the behavior until the owner acquiesces to the pet or banishes it. Banishment may not extinguish the behavior if a pattern has been established: it may provoke more intense attention-seeking behavior such as pawing, howling, scratching, stealing, destruction, and barking. Attention-seeking behavior could include spinning, tail-chasing, sucking, and fly-biting.

Owners whose animals periodically have intense bouts of motor activity, with or without ingestion of nonfood objects, commonly ask whether the animal could have hyperactivity. Truly hyperactive dogs are rare; most dogs evaluated for hyperactivity are overtactive or are high-energy dogs who do not get enough exercise. Dogs in the latter categories will respond to increased attention and exercise, and to schedules and possibly, diets, more suited to their needs. Hyperactive dogs are almost incapable of relaxation unassociated with exhaustion and collapse; such dogs have physiologic signs of hyperactivity, including dilated pupils, increased heart and respiratory rates, flushing of skin, and congestion of scleral vessels, even when restrained. Hyperactive dogs may respond to treatment with methylphenidate (5 mg, PO, q 12 h; up to 20 to 40 mg daily) or amphetamine (dextroamphetamine: 0.2 to 1.3 mg/kg, PO, as needed; levoamphetamine: 1.0 to 4.0 mg/kg, PO, as needed). These drugs cause nonhyperactive dogs to become stimulated, but have a calming effect for hyperactive dogs. Response to these drugs can be diagnostic in questionable cases. Dog 1 was not physiologically abnormal at rest and was capable of quiescent periods; accordingly, hyperactivity was ranked low on the list of differential diagnoses.

Although 2 of the 3 dogs of this report were treated with anticonvulsant medication and were unresponsive, all dogs benefitted from clomipramine treatment. For dog 3, however, the benefit was not as great as that in the other dogs. Clinical signs did not totally resolve in any of these dogs with medical treatment alone.

Two of the 3 dogs became worse when the clomipramine dosage was decreased or treatment was stopped. These findings are common in human psychiatry: without some cognitive changes resulting from behavioral modification, recidivism and breakthrough rates are high. Treatment of obsessive-compulsive disorder is invariably ongoing, and focuses on management and remission, rather than cure.

Although buspirone has been reported to paradoxically increase the frequency and intensity of some obsessive-compulsive (stereotypic) behaviors in human beings, particularly when this drug is paired with fluoxetine, intensity or frequency of stereotypic behavior was not noticed to increase in dog 3 when buspirone was given with clomipramine. It is unclear why fluoxetine treatment, in addition to clomipramine, resulted in worsening of clinical signs in dog 3; paradoxical treatment has also been reported for fluoxetine.

Most psychotropic medication may potentially affect thyroid hormone concentrations, potentiate arrhythmias, engender epileptiform seizures, and increase most hepatic-associated enzyme activities, particularly alkaline phosphatase. Treatment with tricyclic antidepressants at high doses has been associated with sick euthyroid syndrome. Transient effects that may potentiate other behavioral problems include increased thirst and appetite and potential weight gain. Rational pharmacologic treatment for psychologic/psychiatric disorders in companion animals mandates frequent physiologic monitoring.

Punishment was not used in the course of treatment in any of the dogs in this report; punishing fearful or anxious dogs can worsen the problem.

Ritalin, Ciba-Geigy Corp, Summit, NJ.
Behavior modification was continued concomitant with drug treatment.

Differential diagnosis in dogs such as those discussed here should include metabolic diseases, primary neurologic diseases, nutritional diseases, infectious diseases, and behavioral disorders. The latter are postulated to be caused by anxiety disorders and have been variously classified.14 Behavioral disorders frequently are accompanied by overt medical signs, such as those from bacterial infection secondary to trauma, that further complicate diagnosis. Treating all secondary complications of the ritualistic behavior and excluding any underlying medical cause is critical prior to presumptive treatment of a behavioral disorder. Treatment of behavioral conditions invariably involves psychotropic medications that are anxiolytic or that ameliorate obsessive-compulsive behavior. These medications, which are usually not approved for use in nonhuman species, are not benign, may mask or worsen metabolic or neurologic problems, can be toxic in inappropriate dosages, and may have potential for abuse. Rational implementation of psychotropic medication for companion animals necessitates evaluation of these aspects.

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**Book Review:**

**Clinical Textbook for Veterinary Technicians. Third Edition**

Clinical Textbook for Veterinary Technicians (third edition) by McCurnin is well written, easily read, and thorough, and is the type of text that would be well worth the money for students to have in their library. The illustrations provide excellent diagrams to better explain the text. After total review, I liked the book so much, I have adopted it for use in several of my classes. I think the author did an excellent job putting this book together.—[Clinical Textbook for Veterinary Technicians. Third Edition. By Dennis M. McCurnin. 674 pages; illustrated. WB Saunders Co, The Curtis Center, Independence Square West, Philadelphia, PA 19106-3399. 1994. Price $55.00.]—TERRY D. CANERDY

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