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Chronic Management of Tachyarrhythmias in the Dog

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THE BASIS OF OUR DECISIONS

The truth is we really do not have enough information to practice 'evidenced-based' medicine in the treatment of cardiac arrhythmias in veterinary medicine. Consequently, it behooves us to be less than dogmatic in detailing our opinions. Tremendous volumes of information are available with regards to the treatment of cardiac arrhythmias in humans. We can borrow cautiously, and with likely error from these data, as we struggle to determine the best treatment for the dog with arrhythmias. However, the diseases most commonly studied in humans are associated with myocardial infarction and this particular type of ischemia is not what we see in our patients. Therefore, the vast majority of how we decide to treat an arrhythmia in the dog will not be based on science, but the other arm of medicine: Art. And likely, the truth lies in good luck. What then is the key to knowing how to manage cardiac arrhythmias in veterinary medicine? The key is individual patient follow-up.

WHAT ARRHYTHMIAS DEMAND TREATMENT

Treating Tachyarrhythmias

A little bit of bad is okay. In old textbooks, treatment was recommended for even single premature beats (including both ventricular and supraventricular beats). There were at times, what now seem 'odd recommendations' for treatment (e.g., greater than 17 PVCs per minute treatment is given). The tolerance for other than a sinus rhythm has increased because it has been learned that to eliminate all bad can lead to worse. That is, in certain situations the side effects or proarrhythmic effects of drugs can lead to morbidity or mortality. As a result, new guidelines are proposed when evaluating the need for treatment tachycardias. It should be noted that these recommendations are not founded in fact. We treat fewer dogs for arrhythmias, but the ones we do treat we do so more aggressively and with meticulous follow-up.

Ventricular Arrhythmias

Why should we be selective about what arrhythmias are treated? Because beneficial results may not always occur and adverse effects are possible. Treatment is based upon the assumption that therapy will 1) reduce the risk of death; 2) decrease the frequency of a dangerous arrhythmia; and 3) improve or abolish related clinical signs.[1] Sudden cardiac death can result from ventricular fibrillation (VF), ventricular asystole, or electrical-mechanical dissociation. It was once believed that decreasing the frequency of ventricular extrasystoles would also decrease the risk of sudden death. However, this assumption is not always true. Drugs can have antiarrhythmic effects without antifibrillatory effects and vice versa. If ventricular asystole or electrical-mechanical dissociation is the mechanism of death, then drugs that prevent VF may not affect that outcome. Ventricular premature complexes are **not a surrogate** for sudden death: Is ventricular tachycardia?

Ventricular fibrillation is a common terminal and fatal canine arrhythmia. Rapid VT usually (not exclusively) precedes the development of VF because it destabilizes the more homogeneous electrical state of the myocardium. That is, a dispersion of repolarization develops which is predisposing to VF. Some drugs such as bretylium and beta-adrenergic blockers may be more of an **antifibrillatory than antiarrhythmic.**

How do we know when something is bad? (Can the answer be: "I know it when I see it," Supreme Court Judge—Potter Stewart, 1964). A ventricular arrhythmia is judged to be dangerous 1) when it causes hemodynamic compromise (e.g., low cardiac output with weakness, syncope, or CHF; hypotension; or collapse); or 2) when it may degenerate into a more electrically unstable and fatal arrhythmia. The bottom line is: PVCs are not bad alone. Couplets are usually not bad. Triplets and VT are bad, unless the rate is 'not fast.' Defining the latter is covered in gray. Fast is greater than 160 beats/minute: usually treated. Slow is less than 120

beats/minute: usually not treated. In between is 120–160 beats/minute: may be treated sometimes! These guidelines are subject to change with breed and other factors. Hence, heart rate is the most important determinant of hemodynamic compromise as it pertains to the arrhythmia itself. Compounding the negative effect of a fast rate is underlying myocardial failure. **The general rule: If it is fast, it is bad.**

How do we know when something is really bad? Such a question is vital because it is these situations that demand intravenous antiarrhythmic treatment and other support. Again, rate is critical as is the underlying myocardial function. The size of the dogs also influences the tolerance to the tachycardia (a small dog can tolerate a faster rate than a giant breed dog). As a general guide, most dogs will have substantial hemodynamic compromise when the heart rate is **sustained** (greater than 30 seconds) at a rate of greater than 250 beats per minute. In such cases intravenous treatment is ideal. **The general rule: If it is really fast, it is really bad.**

Other ECG features in addition to the fast rate can imply electrical instability and increase the belief that the arrhythmia should be treated. These features include: 1) polymorphic PVCs; 2) a short coupling interval between the PVC and the preceding normal beat (i.e., the premature ventricular complex occurs during the vulnerable period of the T-wave [R on T phenomenon]); or 3) a short cycle length (i.e., rapid heart rate) for the consecutive ventricular complexes even if not VT (this means couplets and triplets). The assumption is that rapid, monomorphic, or polymorphic VT is more likely to degenerate into ventricular fibrillation than slow (< 120 beats/minute), monomorphic VT. We do not have proof that this is a true statement. **Fact often disproves the best theories!**

Elimination of clinical signs should be a goal of antiarrhythmic therapy. This sounds like such a reasonable goal. In truth we rarely have the documentation that this has actually happened. Weakness, exercise intolerance, hypotension, and syncope can be confirmed to have been improved with treatment, but it must be documented to be known and not just 'guessed.' Therefore, adequate follow-up of each patient is mandatory to judge the attainment of this goal. This requires that we really know what the dog was doing before and after treatment was started. For example, if the dog had three horrible collapsing episodes over a 3-month period, then medication was started and the dog has had only one collapse in the next 3 months. Does this justify saying that the treatment worked? Unlikely. Twenty-four hour electrocardiographic (Holter monitoring) coupled with a diary of the pets activities can help correlate an arrhythmia with clinical signs when the association is in question.

When deliberating whether to treat a ventricular arrhythmia, one must consider the clinical presentation, associated disease, and natural history. For example, in humans myocardial dysfunction is the major determinant of risk associated with ventricular arrhythmias in man; the extrapolation is most likely true for dogs as well. The Boxer and Doberman pinscher are known to have sudden death associated with ventricular tachycardia, and thus is antiarrhythmic treatment ideal even if only single PVCs are documented in a dog because of the breed historical characterization? In some of these dogs, however, the cause of death may actually be asystole. German shepherd dogs with rapid VT are at risk of death until 18 months of age, whereas those with only PVCs, or those older than 18 months are at low risk. Such historical information can help 'swing' the decision for treatment in such cases of animals that do not have documented VT, but that are syncopal and have 'important' ventricular arrhythmias documented without other bradyarrhythmias confusing the conclusion.

What are the criteria for documenting a treatment effect when using antiarrhythmics in the treatment of ventricular tachycardia?

It is time that we just said it: You cannot possibly have a clue as to the antiarrhythmic effects of a drug unless a baseline Holter is recorded before treatment and a follow-up Holter is recorded after treatment **and** within a reasonable period of time.

Evaluating drug effect from a routine ECG is just short of useless outside of the critical intensive care monitored patient being given intravenous therapy for sustained VT as you search for conversion to a sinus rhythm.

Holter monitoring is not without its own limitations. Post treatment Holters must be made within a 'short' period because of spontaneous daily variability in arrhythmia frequency which is more likely to occur the longer time that passes between recordings. In humans, even with 24 hours of ECG recorded, the day-to-day variability of ventricular arrhythmias is high. Variability is greater when the frequency is low (< 200 PVC beats/hour) than when the PVC counts are high (> 1000 beats/hour). Moreover, when the interval between recordings is increased, the spontaneous variability in arrhythmias varies even more. Therefore, to clearly demonstrate a positive drug effect: 1) reexamination should be made within a short period of time; 2) the percent reduction of arrhythmias should be stringent; 3) long term follow-up may include examination off drug treatment; and 4) and the limitations of monitoring, as they apply, must be factored in. Nevertheless, Holter monitoring provides critical assessment for managing ventricular arrhythmias. Holter monitoring is better than the minute amount of information provided by a 1- or 2-minute rhythm strip. Holter monitoring is a valuable investment because of the cost benefit of the information obtained. Based on 48 hours of Holter monitoring in man, suppression of approximately 90% of repetitive ventricular ectopic beats (nonsustained VT) and 75% reduction in PVCs must be achieved to comfortably ascribe an antiarrhythmic effect. This type of criteria is effective only when ventricular arrhythmias are frequent.

Currently, specific recommendations to judge treatment success have not been validated for dogs; accordingly, (once again) we must assess the individual patient. In the evaluation of our patients, we may discover questions and gain insight into the problems in arrhythmia control. For example, in Table 1 an example of the tabulation that we keep on each dog treated for ventricular arrhythmias is shown. As shown in this table we will assess not only the arrhythmia counts, but the type of arrhythmia, their rates, and the underlying sinus rate. When certain drugs are given such as those with beta-adrenergic blocking properties we can determine the degree of adrenergic blockade as reflected in the average heart rate and the amount of time spent in sinus rhythm less than 120 beats/minute. In contrast, we can identify when excessive adrenergic blockade has occurred as judged by the number and length of sinus pauses. Back to the arrhythmia counts; note that although the PVC numbers are essentially unchanged, the amount of VT has decreased by greater than 90% with the higher dose of sotalol. However, note that with the low dose sotalol the VT has actually increased. Is this a proarrhythmic effect of the low dose sotalol or just variability? This question cannot be answered. Further follow-up is demanded at the apparent antiarrhythmic dose to make the hopefully the correct conclusion. The dog shown in Table 2 demonstrates a reduction in arrhythmias that is not as confusing.

No lies, no tape please, and Holter monitoring. If we are to make judgments about the success of treatment of treatment based on 24-hour ambulatory ECG monitoring; then the Holter analysis must be accurate. **For the dogs with the worst arrhythmias that require this information the most, it can be stated that most analysis reports of the counts of ectopic complexes by most Holter analyzers are wrong.** It is critical that the accuracy of these reports be evaluated. The use of digital high frequency recordings (1000 Hz) coupled with special manipulations of the analyzer can drastically improve this information. For example, we have documented that at a sampling frequency of 200 Hz beat annotation can be greater than 65% incorrect in the assessment of a recording with frequent ventricular arrhythmias. To correct such a tape requires greater than 20 hours of manual editing. With a simultaneously recorded Holter at 1000 Hz and the application of changes in the algorithm the inaccuracy is reduced to 32% which then required 2.5 hours of manual editing to have correct counts. **The key points: Holter analysis is not to be evaluated by looking at the report page that some company or person has given you without you inquiring in detail about the accuracy of the data. This accuracy can only be evaluated by looking at the quality control and knowledge of Holter analysis in canine recordings.** Be suspicious of

results unless you have evidence of accuracy. On a good note, perhaps very soon we will have the recommendations to take this worry away with improved analysis possible. The future lies in high frequency digital recordings. Holter recordings made on tape is okay for analyses of recordings that you don't really need to do.

What dogs do we most commonly treat for ventricular arrhythmias and what are the first line drugs? Dogs with ventricular tachycardia are the focus of treatment efforts. The most common drugs used today are mexiletine plus atenolol, sotalol, and amiodarone. The later is more commonly used in Europe than in the US in dogs. At Cornell we most commonly use either mexiletine plus atenolol or sotalol. How do we decide between these two? At this point, we have no logical explanation to provide. We have had situations where we will start with one drug, and the animal develops side effects (such as 'dog is not acting normal') and we then switch to another drug. Each of these treatment regimens is costly (mexiletine plus atenolol ~ $1.40/day, sotalol $2/day, amiodarone $1.25/day).

Mexiletine

The electrophysiologic properties of mexiletine are similar to lidocaine. Therefore, untoward effects are comparable. While VT treated successfully with lidocaine is usually effectively treated with mexiletine, the acute effects of lidocaine do not always predict patient response to mexiletine.

Currently, mexiletine (4 to 8 mg/kg, q 8 hours PO) combined with atenolol (0.5 mg/kg, q 12 to 24 hours PO) can be used for chronic oral therapy of dangerous ventricular arrhythmias. Untoward effects (trembling, seizures, depression) are similar to lidocaine at toxic doses. Gastrointestinal side effects can usually be limited by giving mexiletine with food. Mexiletine has less proarrhythmic effects than the commonly used class Ia drugs.

Mexiletine is usually combined with atenolol for chronic treatment. The adjunctive use of β-adrenergic blockers to treat VT has been acclaimed in humans based on evidence that they decrease mortality. β-adrenergic blocker monotherapy may be inadequate for controlling PVCs, although they are particularly effective when PVCs and VT are associated with high circulating catecholamine levels (e.g., during anesthesia with halothane and with exercise). In human studies, β-adrenergic blocker monotherapy only modestly affected the incidence of PVCs, but decreased mortality. The latter may be due to the antifibrillatory effects of β-adrenergic blockade. Applicability of these studies to clinical veterinary medicine has not been determined. Caution must be used if β-adrenergic blockers are given when myocardial failure is present.

The dose of β-adrenergic blockers must be titrated to achieve the desired therapeutic end points and this can be aided by the heart rate response as judged by Holter analysis (see Tables 1 and 2).

Sotalol

Experimentally, sotalol was effective in suppressing VT in dogs induced by increased sympathetic influences. We most commonly use a dose that is between 1–2 mg/kg twice daily. The clinically available compound, d,l-sotalol, is not purely a class III agent, but has class II (beta-adrenergic activity). The β-adrenergic blocking activity of d,l-sotalol is about 30% that of propranolol. Sotalol differs from amiodarone in that sotalol does not bind to plasma proteins; is eliminated through the kidneys; is hydrophilic; lacks active metabolites; has no effect on digoxin concentrations; and long-term administration does not alter its kinetics with plasma concentrations proportional to the dose. When treating with sotalol the heart rate, QT interval, and P-R interval should be monitor. Remember that the QT interval needs to be corrected for heart rate for true comparison. The best formula that balances practicality with the least over or under correction is that developed by L. S. Fridericia. This

formula does tend to have some overcorrection. That means it will tell you that the QT is a bit longer at slow rates than it really is. The formula is as follows:

$$QTc = \frac{QT}{\sqrt[3]{\frac{60}{Heartrate}}}$$

Sotalol does not affect serum digoxin levels. Moreover, drug interactions with sotalol are infrequent because of its lack of protein binding and biotransformation. However, sotalol can cause a worsening of congestive heart failure, lethargy, and bradycardia which is related to its β-adrenergic blocking activity (see Table 1 and 2).

Amiodarone

When administered orally, amiodarone has a delayed and highly variable onset of action which makes dosing and determination of clinical response difficult. Amiodarone is very lipophilic, protein bound, and has a high volume of distribution. Because it can cause severe side effects, the goal should always be to use the lowest effective dose. Clinical experience has been very limited. A loading dose of 10 mg/kg/24 hours was given to dogs in experimental studies for 7 to 10 days followed by a maintenance dose of 5 mg/kg daily or three times per week. Others have used smaller doses and claimed success.

Dose related side effects with amiodarone include gastrointestinal, pulmonary fibrosis, hyperthyroidism, hypothyroidism, ocular opacities and hepatic failure. The use of low dose amiodarone has been vaunted as a means of achieving antiarrhythmic activity without untoward effects.

Amiodarone has a minor but definite negative inotropic effect which is offset by the lengthening of the action potential duration and associated prolongation of contraction. Consequently, long-term treatment with amiodarone does not worsen or induce congestive heart failure because it has little or no net negative inotropic effects. However, concomitant use of digoxin with amiodarone can cause plasma digoxin levels to increase.

What happened to procainamide? Well, we just stopped using it because we did not think that it worked (testimonial results only). Also, many dogs developed side effects. And finally, it is likely that it will be pulled from the market because of proarrhythmic effects in humans. Too bad, it was cheap and maybe it worked in a few dogs, but we really don't know.

Name the dogs. Of course, there are going to be dogs of no particular breed that will have VT that needs treatment; however, the breeds that we see most commonly that demand chronic treatment are boxers and Doberman Pinchers. When young German shepherds are identified with the malignant VT that characterizes the dogs at risk for sudden death, these animals need treatment, but we do not as yet know how to eliminate these arrhythmias.

A recent study headed by Dr. Kate Meurs demonstrated in the boxer that a combination of mexiletine plus atenolol was the most effective treatment with sotalol a fairly close second.[2] Procainamide was not effective. Boxers are usually presented with a history of syncope which is associated with a rapid monomorphic VT that is likely of right ventricular origin. The substrate for these arrhythmias may be similar to that for arrhythmogenic right ventricular dysplasia (communication Drs. PR Fox and K Meurs), although a disease similar to that described as right ventricular outflow tract tachycardia in humans cannot be excluded at this point. Moreover, some boxers have myocardial failure that can develop years later or which is the presenting clinical feature. Whether all these boxers have the same disease is not clear. A note to remember, Holter recordings from fainting boxers have revealed the coexistence of VT and long sinus pauses (even longer than we permit in a brachiocephalic dog) of greater than 5 seconds. Therefore, be alerted that such boxers may be fainting because of the bradyarrhythmia rather than the tachyarrhythmia or due to both. We have had to pace and treat medically such dogs.

Doberman pinchers with myocardial dysfunction can die suddenly. The cause of death usually is suspected of being VT that degenerates into VF, although bradycardias may be the cause in some cases. Ventricular tachycardia in the Doberman does not have a distinctive morphologic characteristic. Monomorphic or polymorphic VT is common. Successful antiarrhythmic management can be difficult because of concurrent heart failure and systolic dysfunction. Therefore, use of antiarrhythmic drugs (i.e., d-sotalol) which have potential negative inotropic effects may result in decompensation. Consideration of potential drug interactions is important. Advice in this circumstance is to control the congestive heart failure, and then treat with antiarrhythmic as needed with close follow-up to assess response.

German shepherds with sudden arrhythmic death are likely hiding in the population.[3–5] A rapid polymorphic VT characterizes the rhythm of dogs likely to die suddenly. Death occurs in the young (usually 12–52 weeks of age), usually at rest, but not exclusively. This is an inherited disorder that can manifest clinical arrhythmias in an entire litter or only a few dogs. The vast majority of dogs have no clinical signs (rare dog may have syncope). Consequently, a diagnosis is by serendipity (ECG screening before neutering, auscultation of an arrhythmia during a routine examination). Mildly affected dogs can produce severely affected offspring. Although lidocaine can abolish the arrhythmias, an effective oral medication has not been identified. Only dogs with VT documented by Holter analysis are at risk for death. Many German shepherds have been identified with only premature complexes indicating that they carry the genotype with some phenotypic expression, but such dogs do not need treatment. These dogs will likely not have any arrhythmias detected after 18 months of age. Severely affected dogs, if they survive the window of vulnerability can also outgrow the arrhythmias (or have greatly reduced numbers) after about 24 months of age.

SUPRAVENTRICULAR ARRHYTHMIAS

Actually, the various supraventricular tachycardias (SVT) that are seen in dogs can be more intriguing than the ventricular arrhythmias. In humans, lovely algorithms to specifically identify the type of supraventricular arrhythmia will have you measuring and determining if the RP is longer than the PR to classify the specific type of SVT. This may help you narrow the field from 10 differential mechanisms to 5, but that is about it. Then, there is the uncommon dog with a by-pass-tract SVT that definitely provides intrigue, excitement, and dialog for all. We can sort out dogs with AV nodal dependent and independent SVT which may set us up for what we can expect if treatment is successful. That is, the potential to control the arrhythmia versus control the ventricular response rate. However, the common drugs that we use (atenolol, diltiazem, and digoxin) can sometimes have surprising antiarrhythmic effects in some dogs when we just expected to control the ventricular response rate. When these are not effective, we can launch out to the use of drugs such as propafenone, sotalol (treats everything), or amiodarone (treats everything too). A common type of SVT seen in the dog is one which is not AV node reentrant (positive P wave is present in the caudal leads), does not exactly stop abruptly, frequently has a slight increase in the PR interval or a single P wave just before the arrhythmia breaks, and is usually intermittent and paroxysmal (to a point). With these arrhythmias a trial with the medications is required to find control. Remember that when evaluating each drug it is important to maximize the dose for each drug (before side effects) before moving to the next drug as titration to effect is important and varies greatly between dogs. Having said this, it is completely acceptable to begin with combination therapy so long as close follow-up is done.

Atrial fibrillation, the most common arrhythmia that you are likely to treat, can be treated with various drugs. Once again, follow-up is the key to success. At this point in small animal medicine the treatment goal has been to control the ventricular response rate and not try for cardioversion (getting the heart back to a sinus rhythm). The adage has been that 'dogs with atrial fibrillation cannot be converted or to maintain a sinus rhythm after conversion is not possible.' This approach has not been the goal in the horse or in humans; the goal here has been cardioversion. In the horse the atria is structurally normal, cardioversion usually works and is most often

maintained. Similarly, some humans can have lone atrial fibrillation a normal structure, while others have pathology of the atria. As recent reports in the dog (Dr. Janis Bright, ACVIM 2002) have challenged the statement that cardioversion in the dog is not likely or sustainable, large clinical trials (See website: R-6-722777-5975104-2-709-US1-E5D24951@xmr3.com for these trials. PIAF study Lancet 2000, AFFIRM trial 2002, RACE trial, PAF II trial, and the STAF trial) in humans are providing evidence that it is actually the rate control over cardioversion that determines long-term well-being. Thus, given the specialized equipment required for cardioversion in the dog, rate control still is a highly desirable goal.

How is rate control in atrial fibrillation defined? This is an important issue which has, of course, no specific 'one size fits all' recommendation. The factors to consider in deciding the **target heart rate** include: 1) size of the dog; 2) underlying disease (lone arrhythmia with normal heart, dilated cardiomyopathy, volume overload with maintained myocardial function); and 3) side effects to medication in an individual patient. Tachycardiomyopathy is a well-established consequence of excessive heart rates. The faster the heart rate, the faster the myocardial failure will develop. Alteration in function can develop within days and clinical signs within a few weeks. But, does the heart rate need to be the average normal rate for that breed? The smaller dogs will tolerate a higher rate than the giant breeds. Perhaps a dog with myocardial failure should have a higher rate to maintain cardiac output and blood pressure, whereas a dog with lone atrial fibrillation can reach the normal heart rates. For example, a Doberman pincher with dilated cardiomyopathy and a ventricular response rate of 260 bpm should have a rate goal of 130 bpm and not 100 bpm?

Another issue is how the rate is determined. 1) Heart rate from auscultation in the examination room? 2) Heart rate as determined by the owner at home? 3) Heart rate from the routine ECG? 4) Heart rate from Holter recordings? Consistency must be maintained when addressing changes in the heart rate. Although the Holter recording can provided documented heart rates for an extended time, long term monitoring by the owner complements this evaluation as treatment is adjusted. We provide the owners with a table to fill out the heart rate based on auscultation. Most owners can do this and it is amazing how valuable this information can be in determining the proper dosage.

What drugs and what dosage? The first line drug for rate control has long been digoxin. And it works most of the time. The times when digoxin does not work as much as we would hope is during exercise, excitement, or when the heart rates are very high (greater than 220 bpm). In these situations, digoxin needs a little help. This assistance can be provided by calcium channel blockers (such as diltiazem usually given as the long acting preparation) or beta blockers. Sometimes we use either of the latter two drugs alone without digoxin to control the rate. There are several ways to get a slow down, there is not just one way to do this, and combinations can be successful.

What are the problems when striving for rate control? Remember, you can get the heart to slow down if you use enough drug. But there lies the problem. There is a 'ceiling' effect with digoxin. That is, the therapeutic/toxic dose of digoxin prevents titrating up to get the desired slowing in some dogs. The amount of digoxin required to slow the heart enough is beyond the therapeutic level. With calcium channel blockers or beta-blockers there is more room for titration; however, at the doses needed to reach the heart rate goal the negative inotropic effect or drop in systemic blood pressure can bring disappointment to reaching the target heart rate. The latter is not as much of a problem in dogs that have lone atrial fibrillation with maintained myocardial function. In these dogs, the dose of either diltiazem or atenolol can be titrated to achieve the desired heart rate. Dogs with myocardial failure, particularly Dobermans, have much less tolerance to the 'drug push.' Also, the matter is complicated by the disease and concurrent drugs. For example, diltiazem will cause the blood pressure to drop and if other drugs (diuretics, vasodilators) with this effect are given, which they usually are in these circumstances, the additive effect is too much. Hypotension will result with the dog displaying weakness and a

decreased blood pressure. Ironically, the situation can lead to a dog being given both diltiazem or digoxin and dobutamine simultaneously.

In a dog with acute heart failure due to dilated cardiomyopathy and marked tachycardia due to atrial fibrillation the goals must be prioritized. Most of the time severe dyspnea is present in addition to the tachycardia of the atrial fibrillation. The dog must breathe before all else. Aggressive, short-term treatment with diuretics is given with acute treatment with nitroglycerin, and some may use nitroprusside in that handful of places that have continuous monitoring. The dilemma is whether to treat the dog with dobutamine or diltiazem. The dobutamine for the inotropic support or diltiazem to slow the heart rate (some of these dogs have rates greater than 240 bpm). A second dilemma concerns the route of drug administration. In the dog that is in complete distress, the intravenous route is obvious, but not all dogs are in this most dire position. Some could be treated orally. Response to oral diltiazem can be rapid; however, if there is a negative response the effects can be more protracted if the long acting preparations are used such as Dilacor@. The effects of regular diltiazem do not last long in the dog and require frequent administration; however, this can be an advantage in a dog where the IV route is not selected, but the long acting medication is not desirable as yet. The unfortunate problem is that with calcium channel blockers and beta-adrenergic blockers the heart rate control comes with the price of hypotension in many dogs with myocardial failure. This is why a combination of treatment with digoxin can be advantageous, but the dog must live long enough to have all the medications achieve adequate blood levels. Once this is achieved, titrating to the oral doses can begin. A switch to long acting medication can be made once the drug dosage is determined. Although some modifications are likely, there is a general guide to follow when changing from a regular oral medication to the long acting. Take the total daily dose for the regular medication and this should equal the total daily dose for the long acting; just given that the medication is not divided up as frequently. (For example, a dog was receiving 30 mg of regular diltiazem every 6 hours for a total daily dose of 120 mg. Therefore, the dog would receive 60 mg of Dilacor twice daily). It has been our experience that if a dog is not in severe myocardial failure, the dose of diltiazem is higher than expected to achieve heart rate control. We commonly use 2 to 4 mg/kg twice daily of Dilacor for long-term treatment. This type of dose however is lowered with concomitant treatment with digoxin. Some persons may prefer to use atenolol as the adjunctive treatment to digoxin and we do use this as well. In fact, treatment with beta-adrenergic blockers may be preferred as according to studies in humans there was a better outcome with beta-adrenergic blockers over calcium channel blockers. How much we can borrow from this is unknown.

There are also new approaches to rate control. Carvedilol may become the drug of choice in the treatment of congestive heart failure. The beta-adrenergic blocking action of this drug may assist in rate control. Also, amiodarone has been used extensively in humans with atrial fibrillation and it may find its way into our common treatment. The myocardial suppression effects may be less than diltiazem or atenolol. Perhaps we should select this first? We don't know.

SUMMARY

Here are some key points concerning all of this discussion:

- The correct treatment for arrhythmias has not been determined in humans and this is despite millions of dollars in research, tens of thousands of patients, thousands of investigators, and several decades of focus. Consequently, we cannot be too disappointed if with a few bucks, a few dozen patients, and a handful of veterinary cardiologists trying to perform investigations for a few years that we too do not have all the answers.
- The important thing to do is educate ourselves as to the pros and cons of each drug, try to understand better the diseases that we are treating, and to do follow-up evaluations on every patient that we are able to treat. We must individualize treatment and honestly evaluate thoroughly the response to that treatment.

Table 1. Before and After Treatment Worksheet: 24-hour Ambulatory ECG Monitoring

Owner's Last Name:	Depolarization	Breed:	Boxer
Patient's Name:	"Early"	Age:	6 yr
Case #:	101010	Gender:	Fs
Medication times:	R1–7pm/8am, R2–7:30 BID		

Holter	Date of HLTR	Duration of HLTR (Hrs)	Drug	Dosage	Drug Duration	HR avg	HR min	HR max	Artifact %
Baseline	5–9–02	24:37	x	x	x	130	69	200	<1
Reck 1	5–23–02	19:20	Sotalol, also Digoxin, Enalapril, Lasix, Carnitine	40 mg BID, 0.125 mg BID, 15 mg BID, 2 mg/kg, 2g BID	2 wks	117	44	211	<1
Reck 2	6–13–02	24:12	Sotalol, also others–same	80 mg BID, same	3 wks	103	56	169	<1

Holter	Total VE Singles	VE Singles/ Hr	Total VE Couplets	VE Couplets /Hr	Total VE Runs	VE Runs/Hr	Total QRS	Total VE	VE %	HR>120 (Hrs)
Baseline	31,279	1271	10,081	410	3,332	135	192,887	64,000	31	20:30:36
Reck 1	16,465	852	5,789	300	6,456	334	143,441	53,029	37	10:08:25
Reck 2	29,534	1220	1,124	46	202	8	148,589	32,456	22	6:35:01

Table 2. Before and After Treatment Worksheet: 24-hour Ambulatory ECG Monitoring

Owner's Last Name:	Circuit	Breed:	Boxer
Patient's Name:	"Reentry"	Age:	9 yr
Case #:	O101010	Gender:	Fs
Medication times:	R1–8:30 BID, R2–8:30 BID		

Holter	Date of HLTR	Duration of HLTR (Hrs)	Drug	Dosage	Drug Duration	HR avg	HR min	HR max	Artifact %
Baseline	3–7–02	24	x	x	x	100	40	222	<1
Reck 1	3–20–02	24:37	Sotalol	60 mg BID	2 wks	83	34	197	<1
Reck 2	4–2–02	24:23	Sotalol	90 mg BID	2 wks	81	32	185	<1

Holter	Total VE Singles	VE Singles /Hr	Total VE Couplets	VE Couplets /Hr	Total VE Runs	VE Runs/Hr	Total QRS	Total VE	VE %	HR>120 (Hrs)
Baseline	13,848	577	723	30	512	21	137,056	17,088	12	7:34:17
Reck 1	3,899	158	96	4	11	0.4	116,633	4,149	4	2:24:18
Reck 2	2,222	91	46	2	6	0.2	115,989	2,334	2	1:50:41

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