

# Artificial Atrial Fibrillation in the Dog

## AN ARTIFACT?

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### ABSTRACT

R-R interval sequences during artificial atrial fibrillation in dogs were studied in the same way as in patients in a previous study and compared with results obtained in dogs with spontaneous atrial fibrillation. Artificial atrial fibrillation was effected by right atrial stimulation in three closed-chest and one open-chest dog. The ventricular rhythm was studied by histograms and autocorrelograms. The ventricular dysrhythmia during artificial atrial fibrillation generally differed from that during true atrial fibrillation. Therefore the findings during artificial atrial fibrillation cannot be used without reservation to explain electrophysiological (or hemodynamic) phenomena in true atrial fibrillation.

**KEY WORDS**  
randon rhythm

ventricular irregularity  
histograms

atrial stimulation  
autocorrelograms

■ A recent editorial (1) outlined some controversial viewpoints concerning the ventricular dysrhythmia in atrial fibrillation. These controversies arise from the fact that different groups of investigators have found differing patterns of ventricular irregularity in patients with atrial fibrillation (2-6). Our findings (6) indicate that the ventricular rhythm is random, while others have produced evidence for a measurable amount of interdependence between successive R-R intervals (time between R waves of two successive QRS complexes). On the basis of the randomness of the ventricular rhythm in our studies, we offered the hypothesis that randomly spaced atrial impulses of random strength reach the AV node from random directions (6); we felt that the role of the AV conduction system in atrial fibrillation was limited to scaling the rate of the atrial impulses. The electrophysiological justification for the interdependence between successive

R-R intervals which is at times found in patients with atrial fibrillation is based mainly on a paper by Moe and Abildskov using artificial (maintained rapid atrial stimulation) "atrial fibrillation" in dogs (7). In their paper a dominant role is attributed to the AV node. In a previous article (8) we questioned the extrapolation of results from open-chest dogs during artificial atrial fibrillation to patients with autochthonous atrial fibrillation. We felt that a possible solution for the existing controversy (1) might be found if the ventricular rhythm of dogs with artificial atrial fibrillation could be compared with the ventricular rhythm of dogs with truly autochthonous atrial fibrillation.

### Methods

Studies were conducted on seven anesthetized ( $O_2$ ,  $N_2O$ , and halothane inhalation) dogs; three of these had spontaneous atrial fibrillation due to mitral incompetence. In three dogs a bipolar stimulating catheter was introduced into the right atrium via the femoral vein. Artificial atrial fibrillation was produced by regular stimulation of the right atrial appendage with at least twice the threshold current at frequencies between 20 and 50 Hz. One dog was studied under open-chest conditions with bipolar electrodes stitched to the right atrium in the fashion described by Moe and Abildskov (7). In all animals the electrocardiogram was recorded on magnetic tape for 20

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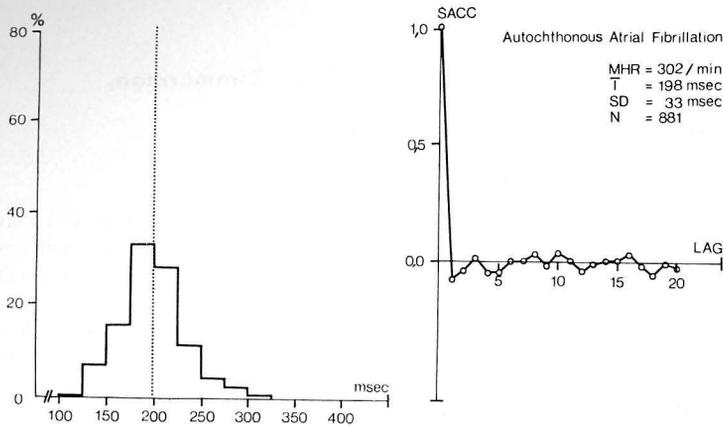


FIGURE 1

Histogram (left) and autocorrelogram (right) of a dog with autochthonous atrial fibrillation. The abscissa of the histogram represents the R-R intervals in classes of 50 msec. SACC = serial autocorrelation coefficient; LAG = coefficient number; MHR = mean heart rate; I = average interval duration; SD = standard deviation of the interval distribution; N = number of intervals.

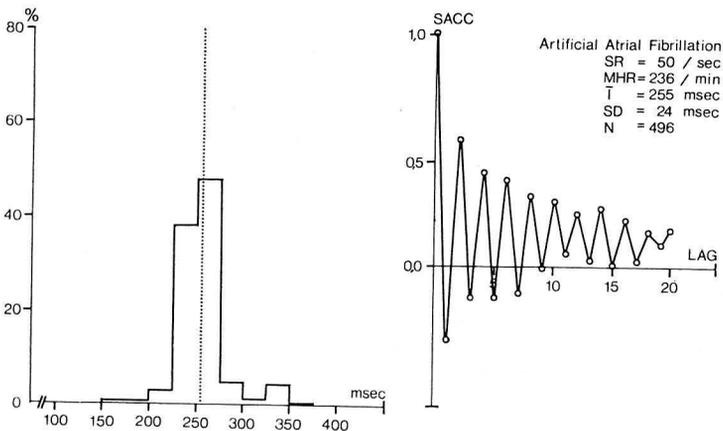


FIGURE 2

Histogram and autocorrelogram of a dog with artificial atrial fibrillation, stimulated at 50 Hz. The autocorrelogram reflects the tendency of relatively short intervals to alternate with relatively long intervals. SR = stimulation rate; other abbreviations as in Figure 1.

minutes and processed with the autocorrelation techniques described in our recent paper on atrial fibrillation in man (6).

As in that study, we computed R-R interval histograms and autocorrelograms, using at least 450 consecutive R-R intervals. Computations were performed on a PDP 9 computer. For details on data handling and statistical methods refer to the previous paper (6).

### Results

Figure 1 presents the autocorrelogram and

the corresponding R-R interval histogram for 881 successive R-R intervals from a dog with autochthonous atrial fibrillation. It can be seen that, except for coefficient zero, all coefficients are well within the 5% confidence limits of zero value. The histogram is unimodal and slightly skew. The findings in the other two dogs with autochthonous atrial fibrillation were similar to those presented in Figure 1.

Figure 2 presents the autocorrelogram and

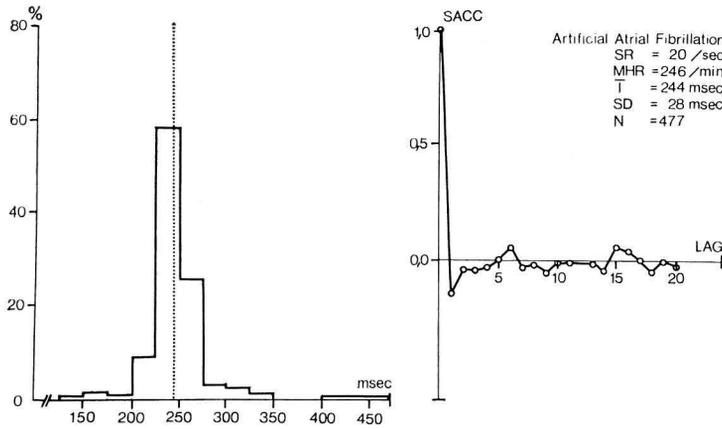


FIGURE 3

*Histogram and autocorrelation of a dog with artificial atrial fibrillation, stimulated at 20 Hz. The ventricular rhythm is nearly random.*

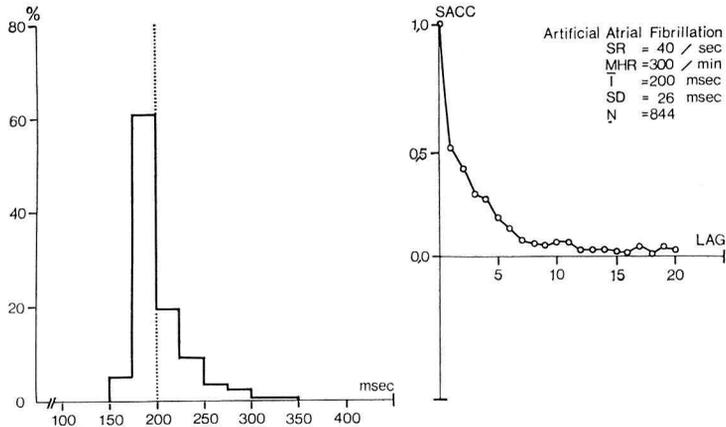


FIGURE 4

*Histogram and autocorrelagram of a dog with artificial atrial fibrillation, stimulated at 40 Hz. The high coefficients for small lags suggest some kind of chaining.*

the corresponding interval histogram of 496 successive R-R intervals from a closed-chest dog with artificial atrial fibrillation (the atrium was stimulated at 50 Hz). The autocorrelagram demonstrates a strong oscillatory behavior; the histogram is bimodal.

Figure 3 presents the results of analysis of a recording from the same dog as Figure 2 during atrial stimulation at 20 Hz. The pattern of the ventricular dysrhythmia is clearly different from that during stimulation at 50 Hz and thus evidently dependent on the rate of the applied atrial stimuli. The autocorrel-

gram resembles that in Figure 1, but the histogram is different. Here the autocorrelagram indicates a random interval sequence.

In Figure 4 the autocorrelagram and the histogram of the open-chest dog is shown at an atrial stimulation rate of 40 Hz. Again, autocorrelagram and histogram are different from those in Figures 2 and 3, reflecting a repetition pattern in which long intervals tend to follow long intervals and short intervals to follow short ones.

All these artificially induced irregularities differ substantially from those obtained in

dogs with true atrial fibrillation (Fig. 1). Although only the results of stimulating the atrium at rates of 20, 40, and 50 Hz are shown, the whole gamut of frequencies between 20 and 50 Hz was tested in steps of 5 Hz, producing a variety of different ventricular rhythms. The results in the open-chest dog with artificial atrial fibrillation were identical to those in the closed-chest animals. Thus the ventricular rhythm during rapid atrial stimulation is not influenced by opening the chest or by electrodes stitched to the atria.

### Discussion

We have presented the autocorrelograms and corresponding histograms of the ventricular rhythm of dogs with clinical symptoms of true atrial fibrillation and of dogs with artificial atrial fibrillation.

In a previous paper (6) we presented autocorrelograms and histograms of the ventricular rhythm of patients with clinically undeniable atrial fibrillation. Both men (6) and dogs (Fig. 1) with true atrial fibrillation show the same random pattern of successive R-R intervals. Thus the ventricular dysrhythmia during true atrial fibrillation as represented by autocorrelogram and histogram seems to be identical in man and dog. This strongly suggests that atrial fibrillation is identical in both species.

The dogs with artificial atrial fibrillation show R-R interval patterns which are dependent on the applied atrial stimulation rate (Figs. 2-4). It is interesting to note that at a certain stimulation rate, in this case 20 Hz (Fig. 3), the autocorrelogram indicates a randomness of the ventricular rhythm pattern. The autocorrelogram in Figure 4 at 40 Hz, however, depicts a situation in which "long cycles tend to favor the occurrence of subsequent long cycles" as found by Moe and Abildskov (7).

Up to now the electrophysiological explanation for the ventricular irregularity in patients with atrial fibrillation was based on observations in dogs with artificial atrial fibrillation. The results in this paper show that this is a questionable extrapolation. We wish to repeat

our former statement (6) that nonrandom portions in the ventricular response during atrial fibrillation in man point to the fact that this response cannot be ascribed to atrial fibrillation per se. Additional (nodal?) foci are presumably active. If this is true, it may have certain clinical (therapeutic) implications. At this moment rather elaborate methods are required to judge whether a ventricular rhythm in atrial fibrillation is truly random. The minimal number of R-R intervals required to define a ventricular rhythm is under study. Furthermore, our findings demonstrate that physiological and especially electrophysiological explanations derived from artificially induced atrial fibrillation in animals cannot be applied without reservation to man (7, 9 and 10).

In conclusion, it can be stated that the ventricular rhythm in true uncomplicated atrial fibrillation is random and that autochthonous atrial fibrillation in man and dog seems to be identical. The pattern of ventricular dysrhythmia in dogs with artificial atrial fibrillation is dependent on atrial stimulation rate.

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