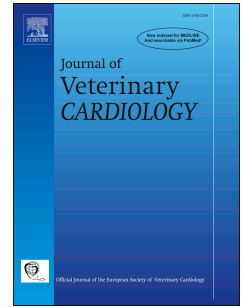


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**Supraventricular tachycardia in 23 cats; comparison with 21 cats with atrial fibrillation
(2004-2014)**

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1 **Abstract**

2

3 **Introduction:** Supraventricular tachycardia (SVT) has not been well-described in
4 cats. The aim of this study was to describe the signalment, clinical findings and
5 outcome for cats with SVT versus cats with atrial fibrillation (AF).

6

7 **Animals:** Forty-four client-owned cats; 23 cats with SVT and 21 with AF

8

9 **Methods:** Retrospective study. Clinical characteristics were compared between
10 groups using a two-sample t-test or Mann-Whitney U test. Kaplan-Meier survival
11 curves were generated to assess for impact of rhythm diagnosis, presence of
12 ventricular arrhythmia, left atrial diameter, heart rate (HR) and congestive heart
13 failure (CHF) status on cardiac death. Differences in survival between groups were
14 compared using Mantel-Cox logrank comparison of Kaplan-Meier survival curves.

15

16 **Results:** Cats with supraventricular arrhythmias most commonly presented with
17 respiratory distress (10 of 44 cats). Cats with AF had a slower median HR (220
18 [range 180-260 beats per minute (bpm)] compared to cats with SVT (300 [range 150-
19 380] bpm, $p < 0.001$). All cats with AF had structural heart disease whereas 4 cats
20 with SVT had no structural abnormalities. Left atrial diameter was significantly larger
21 in cats with AF (23.7(16.2-40.1) mm, compared to 19.1 (12.8-31.4) mm in SVT cats;
22 $p = 0.02$). Median survival was 58 days [1–780] in cats with AF, and 259 days (2 -
23 2295] in cats with SVT ($p = 0.1$). Cats with signs of CHF had a shorter survival time
24 ($p = 0.001$).

25 **Conclusions:** Most cats with AF or SVT have advanced structural heart disease.
26 Some cats with SVT had structurally normal hearts, suggesting that SVT in cats is
27 not always a consequence of atrial enlargement.

28

29 **Keywords:**

30

31 Feline

32 Arrhythmia

33 Survival

34

Abbreviation table	
AF	atrial fibrillation
ATE	arterial thromboembolism
CHF	congestive heart failure
LA	left atrium
LAD	left atrial diameter
LAE	left atrial enlargement
LV FS%	left ventricular fractional shortening
LVID	left ventricular internal diameter
SCD	sudden cardiac death
SVT	supraventricular tachycardia
VSD	ventricular septal defect

35

1 Introduction

2

3 Supraventricular tachycardia can be defined as any rapid rhythm originating from the
4 SA node, atrial myocardium, atrioventricular node/junction, or great vessels
5 connecting to the atria (venae cavae, pulmonary veins, coronary vein) [1]. The
6 arrhythmia may arise due to spontaneous depolarisation of cardiac cells as a result
7 of enhanced normal automaticity, abnormal automaticity or triggered electrical
8 activity [1,2], or due to the formation of macro or micro re-entry circuits. Atrial
9 fibrillation (AF) is a specific supraventricular arrhythmia in which a series of multiple
10 microreentrant circuits form within the atria, resulting in a chaotic ventricular rhythm
11 associated with the random selection of impulses that are conducted via the
12 atrioventricular node to the ventricles [2,3]. Other supraventricular arrhythmias may
13 be regular or irregular, depending on how the causal supraventricular impulses are
14 conducted through the atrioventricular node.

15

16 Whilst there are many studies describing the natural history, treatment and
17 prognostic significance of SVT and AF in people, our knowledge in cats is based
18 solely on individual case reports [4–8] and a single retrospective study of 50 cats
19 with AF [9]. In people [10] and large breed dogs [11,12], AF may occur in the
20 absence of structural heart disease (known as ‘lone AF’). The latter is associated
21 with reduced morbidity and mortality, compared to individuals with structural heart
22 disease [11,13,14]. Lone AF appears to be rare in cats [5]. There is a positive
23 association of AF with increases in atrial mass [15], and the majority of cats with AF
24 have myocardial disease and severe left atrial enlargement. Most cats with AF are

25 male, consistent with the reported male predominance in feline cardiomyopathy
26 [9,16].

27 The aim of this study was to describe the signalment, presenting complaints, cardiac
28 phenotype and survival time in cats with SVT and AF. It was hypothesized that the
29 prognosis of cats with AF would be worse than that of cats with SVT, and cats with
30 AF would have a larger left atrium.

31 **Animals, materials and methods**

32 Retrospective study

33 Medical records from the Royal Veterinary College's Queen Mother Hospital for
34 Animals' database were searched for cats examined between November 2004 and
35 April 2014 using the key terms 'atrial fibrillation feline', 'AF feline', 'supraventricular
36 tachycardia feline', and 'SVT feline'. Cats were included in the study if both an ECG
37 recorded at 50mm/s at the date of diagnosis and an echocardiographic examination
38 performed within 48 hours of the ECG recording were available for review. Cats
39 without an ECG showing at least 2 leads were excluded. Information was collected
40 on patient signalment, presenting signs, radiographic, ECG and echocardiographic
41 findings, therapy and survival status.

42

43 All ECG traces from the date of examination were reviewed by a single board-
44 certified cardiologist in order to confirm the presence of SVT or AF. For the purpose
45 of this study, the average QRS depolarization rate over 3 seconds (the ventricular
46 response rate) was referred to as heart rate (HR) and was measured from all ECGs.
47 All of the cats had over 3 seconds of sustained SVT in the ECG recordings available
48 for review for calculation of the average rate. To be classified as SVT, the rhythm
49 had to demonstrate at least one of the following criteria

- 50 1. Criterion 1 for SVT: The presence of a sustained, narrow complex tachycardia
51 lasting the entire duration of the stored ECG recording, with a regular R-R
52 interval and HR greater than 260 bpm (Figure 1a supplementary data).
- 53 2. Criterion 2 for SVT: Demonstration of an abrupt onset or exit from a narrow
54 complex tachycardia, either on the paper ECG recording or on subsequent
55 telemetry (Figure 1b supplementary data).
- 56 3. Criterion 3 for SVT: Evidence of a persistent atrial depolarization at a rate
57 greater than 260 bpm, with or without variable atrioventricular conduction ratio
58 (Figure 1c supplementary data).
- 59 4. Criterion 4 for SVT: A wide complex tachycardia in which an intraventricular
60 conduction disturbance was suspected, either due to the presence of a
61 consistent atrioventricular relationship (i.e. a constant P-R interval could be
62 identified) or in which QRS morphology was identical during sinus rhythm to
63 the complex morphology documented during the tachycardia (Figure 1d
64 supplementary data).

65 For the purpose of this study, "SVT" refers to all types of supraventricular
66 arrhythmias, excluding AF [1].

67 Atrial fibrillation was diagnosed when there was a clear absence of P waves in all
68 recorded leads in association with a variable R-R interval or excluded in cats where
69 P waves could be visualized [9].

70 The ECG traces were also reviewed for the presence of ventricular tachyarrhythmia.
71 An ectopic complex was classified as ventricular in origin and premature if 1) the
72 QRS complex was wide (>40ms) and bizarre with a large T wave with opposite
73 polarity to the QRS complex, and 2) if they occurred prematurely when compared to
74 the underlying rhythm ($R-R' < R-R$) [17]. Details of how wide complex tachycardia

75 with presumed intraventricular conduction disturbance were differentiated from true
76 ventricular tachycardia have been outlined in the SVT rhythm criteria above.

77 All echocardiographic measurements were performed by a single board-certified
78 cardiologist from stored two-dimensional images. All echocardiographic studies were
79 acquired using the same ultrasound machine^a and recorded by a board-certified
80 cardiologist or supervised cardiology resident. Images were reviewed using a
81 commercial analysis platform^b. Each measured variable was calculated as an
82 average of at least 3 and 5 different cardiac cycles for cats with SVT and AF
83 respectively. A leading edge-to-leading edge technique was used to measure left
84 ventricular (LV) wall thickness (septal and LV free wall) from the long and short-axis
85 right parasternal 2-dimensional echocardiographic views at the level of the papillary
86 muscles [18]. The LV wall measurements were obtained at end-diastole, defined as
87 the first frame after mitral valve closure on the long-axis, or the frame at which left
88 ventricular internal diameter (LVID) was greatest for short-axis views [19]. The
89 maximal LV wall thickness was recorded as the highest value from averaged
90 measures of the interventricular septum (IVS) and LV free wall measurements.

91 Values $\geq 6\text{mm}$ were defined as left ventricular hypertrophy [18]. Two-dimensional
92 measures of cardiac chamber internal dimensions were made using an inner edge-
93 to-inner edge technique, at the boundary between the endocardial surface and blood
94 pool [18]. End-diastolic LVID was measured from both long and short axis views. Left
95 ventricular fractional shortening (LV FS%) was measured from M-Mode images
96 obtained from a right parasternal short-axis view of the LV, obtained at the level of
97 the papillary muscles. LV FS% was calculated using the following equation: LV FS%
98 = $[\text{LVIDd} - \text{LVIDs}] / \text{LVIDd}$ (where d=diastole and s=systole) [20]. The LVIDs was
99 measured at the end of the T wave on the ECG [20]. Cats were considered to have

100 LV systolic dysfunction when LV FS% was $\leq 30\%$ [19]. Assessment of right-sided
101 cardiac dimensions was subjective; right atrial dilation was assessed by comparing
102 right and left atrial areas from the right-parasternal long-axis view [20].

103

104 Left atrial (LA) size was assessed using two methods: LA diameter to aortic root
105 diameter ratio (LA:Ao) taken from a two-dimensional image from the right parasternal
106 short axis view, measured on the first frame after aortic valve closure and LA
107 diameter (LAD), measured as the diameter of the left atrium parallel to the mitral
108 annulus at the last frame before mitral valve opening, using a right parasternal 4
109 chamber long-axis view [21,22]. Left atrial enlargement (LAE) was present when
110 LA:Ao > 1.6 [18] and/or LAD $\geq 16\text{mm}$. The presence of spontaneous echo contrast
111 and/or an intra-cardiac thrombus was recorded. Each cat was assessed for the
112 presence or absence of congenital cardiac disease, based on the opinion of the
113 cardiologist reviewing the entire study.

114

115 Thoracic radiographs, when obtained, were evaluated by a board-certified radiologist
116 at the time of presentation. Congestive heart failure status was determined by the
117 presence of pulmonary infiltrates consistent with pulmonary edema on thoracic
118 radiography and/or pleural effusion or pericardial effusion on echocardiography as
119 determined by a board-certified cardiologist.

120

121 Survival data were obtained from clinical records, or where date or cause of death
122 was not documented, referring veterinary practices were contacted between August
123 and December 2014 to establish the outcome of each cat. Cardiac death was
124 defined as animals that had been euthanized or died because of congestive heart

125 failure (CHF) or arterial thromboembolism (ATE) or died as a result of sudden
126 cardiac death (SCD). Definitions used for these events were as follows: SCD was
127 defined as being found dead without an obvious cause at home where the cat had
128 been apparently well during the preceding 24 hours or as a witnessed event. Death
129 due to CHF was defined as dying with dyspnea, crackles, cyanosis, fluid pouring out
130 of the mouth and/or euthanasia due to becoming refractory to CHF medication.
131 Death due to ATE was defined as death or euthanasia following a new episode of
132 ATE or worsening of a current ATE episode [23].

133 Statistical analysis was performed using commercially available software^c.
134 Continuous data were assessed for normality using the Shapiro-Wilk test. Normally
135 distributed data are presented as mean (\pm standard deviation) and non-normally
136 distributed data are reported as median [range]. Differences between population
137 characteristics of cats with SVT vs AF were compared using the two-sample t-test
138 and Mann-Whitney U test for normally and non-normally distributed data
139 respectively. Categorical variables were compared using Chi-squared test or
140 Fischer's exact test as appropriate. A statistically significant result was defined as a
141 p-value <0.05 . Kaplan-Meier survival curves were generated to assess for impact of
142 rhythm diagnosis, presence of ventricular arrhythmia, left atrial size (using LAD), HR
143 and congestive heart failure status on cardiac death. Differences between groups
144 were analysed using the Logrank (Mantel-Cox) test. Data was censored if death was
145 due to unknown or non-cardiac reasons, or if they were still alive at the end of the
146 study. Survival times are reported as medians (range).

147

148 **Results**

149 One-hundred-and-eight cats were identified from the clinical records database that
150 had been diagnosed with SVT or AF. Fifty-four cats were excluded due to
151 unavailability of a recorded ECG for review, while 10 cats were excluded due to
152 corruption of the stored echocardiographic data, leaving 44 cats that were eligible for
153 inclusion. There was no significant difference in sex ($p=0.7$), age ($p=0.1$), or breed
154 ($p=0.4$) between cats with SVT or cats with AF. The majority (32/44) were male (see
155 table 1). The breeds represented were domestic short hair ($n=29$), domestic long
156 hair ($n=5$), Maine Coon ($n=2$), British Shorthair ($n=2$), Birman ($n=2$), Persian ($n=1$),
157 Ragdoll ($n=1$), Devon Rex ($n=1$) and Sphynx ($n=1$).

158

159 Initial presenting signs were recorded for all 44 cats and are summarized in table 2.
160 The most frequent presenting signs across both groups were respiratory distress and
161 lethargy. All cats with AF had clinical signs. Only 2 cats were subclinical on
162 presentation, both of which had SVT.

163

164 As expected, all cats with AF had a chaotic rhythm on auscultation, compared to 12
165 cats with SVT ($p=0.01$), likely due to the presence of sinus rhythm with numerous
166 supraventricular ectopic complexes, numerous paroxysms of SVT or due to the
167 presence of SVT with variable atrioventricular conduction at the time of auscultation.
168 The group of cats with AF had a significantly slower HR documented on their ECG
169 (220 beats per minute [180-260]) when compared to the group of cats with SVT (300
170 beats per minute [150-380], $p<0.001$).

171

172 Thoracic radiographs were available in 27 of the cats (13 cats with AF and 14 cats
173 with SVT). Congestive heart failure was documented in 18 cats (11 cats with AF and

174 7 cats with SVT): 3 cats had pulmonary edema (1 cat with AF, 2 cats with SVT), 12
175 had pleural effusion (8 cats with AF, 4 cats with SVT) and 3 cats had both pleural
176 effusion and edema (2 cats with AF and 1 with SVT), with no significant differences
177 between groups ($p=0.1$). The presence of pericardial effusion was documented via
178 ultrasound in 7 cats (1 with SVT and 6 with AF).

179

180 All cats with AF had echocardiographic evidence of left or right atrial enlargement,
181 whereas 4 cats with SVT had no evidence of underlying cardiac disease. Of the
182 latter, one cat (an 11-year-old MN DLH) was subsequently diagnosed with splenic
183 haemangiosarcoma with metastases to both liver and omentum. Another cat (9-year-
184 old MN DSH) had a history of chronic diarrhea and recent onset vomiting of unknown
185 origin. Investigations revealed no significant biochemical or hematological
186 abnormalities and urinalysis and abdominal ultrasound were unremarkable. No
187 identifiable co-morbidities were found in the 2 remaining cats. The ECGs from these
188 4 cats are available for review as supplementary data online, (Figures II to V). When
189 cats were classified according to severity of LAE using LAD, 7 cats had mild LAE (5
190 with SVT, 2 with AF), 15 cats had moderate LAE (7 with SVT and 8 with AF) and 12
191 cats had severe LAE (5 with SVT and 7 with AF). The following values were used for
192 mild, moderate and severe LAE respectively; 16-18mm, 18-24mm and >24mm.[20]
193 Only two cats had congenital heart disease, including a cat with SVT that had left
194 ventricular hypertrophy, LAE and a ventricular septal defect (VSD) identified
195 incidentally on echocardiographic examination and a cat with AF that had a double-
196 chambered right ventricle, VSD and severe right atrial enlargement.

197

198 The proportion of cats with left ventricular hypertrophy was similar between groups
199 which was documented in 14 cats with AF and 11 with SVT ($p=0.2$). Subjective right-
200 sided eccentric +/- concentric right ventricular hypertrophy was more commonly
201 identified in cats with AF ($p=0.04$), and of the 7 cats with right-sided cardiac changes
202 (1 cat with SVT and 6 cats with AF), 6 had concurrent LAE. Left ventricular systolic
203 dysfunction was identified in 8 cats, with equal numbers in both groups.

204 The majority of cats had LAE (see table 4), the proportion of which was similar
205 between groups. Cats with AF had larger LAD (23.7mm [16.1-40.1] vs 19.1mm
206 [12.8-31.4], $p=0.02$). There was no significant difference in LA:Ao between groups
207 ($p=0.08$). The 4 cats with SVT and no LAE were the same cats detailed previously
208 as having no evidence of structural cardiac disease. The only cat with AF and no
209 LAE was the cat with a double-chambered right ventricle, VSD, and severe right
210 atrial enlargement. Spontaneous echo-contrast was common, identified in 22% of
211 cats ($n=10$). Six of these cats also had visible thrombi within the left atrial
212 appendage.

213
214 Cardiac medications were administered to 43 of the 44 cats. The antiarrhythmic
215 medications administered were diltiazem ($n=15$), atenolol ($n=10$) and sotalol ($n=1$)
216 Antiarrhythmic medication was administered more commonly in cats with SVT than
217 AF (16 cats vs 6 cats respectively, $p=0.01$). Antithrombotic medication (aspirin
218 and/or clopidogrel) was administered in 23 cats and 29 cats were treated with
219 furosemide.

220

221 Survival information was available for 40 cats (21 SVT and 19 AF), and the cause of
222 death was known for 32 cats (15 SVT and 17 AF). The most common cause of death
223 across both groups was refractory CHF, occurring in 47% of cats with AF and 43% of

224 cats with SVT (10 cats with AF and 10 cats with SVT). This was followed by ATE
225 reported in 23% of cats with AF (n=5) and 13% of cats with SVT (n=3). Sudden
226 cardiac death was reported in 2 cats (1 with AF and 1 with SVT). Median survival
227 was 58 days [1 – 780] in cats with AF, and 259 days (2 – 2295] in cats with SVT
228 (Figure 1, p=0.1). Cats with a lower HR did not survive longer until cardiac death
229 than cats with a higher HR with either tachyarrhythmia, or when the population of
230 cats was considered as a single group (Figure VI in supplementary data). Increased
231 LA size (according to median LAD for cats with SVT, AF and all cats) did not predict
232 worse survival (Figure VII in supplementary data). The presence of congestive heart
233 failure at presentation was associated with a worse prognosis, (p=0.001, Figure 2)
234 whilst the presence or absence of ventricular arrhythmia on resting ECG had no
235 association with cardiac death (Figure VIII in supplementary data).

236

237 **Discussion**

238 The causal mechanisms, predisposing factors and natural course of SVT and AF are
239 well described in people and to a lesser extent in dogs. Cats with SVT and AF have
240 not been well-represented in the literature, and our study provides information on the
241 characteristics and outcome in cats with these rhythm disturbances. Cats with AF
242 are generally recognised as having a poor prognosis [9], so we have provided similar
243 information in a contemporary cohort of cats with SVT as a comparison. This is the
244 largest study to describe cats with SVT.

245

246 The signalment of this population was reflective of the high prevalence of
247 cardiomyopathy, demonstrating that cats presenting with SVT or AF have a male
248 predisposition and first present at a wide range of ages [9,16,24]. The most common

249 presenting sign across both groups was respiratory distress, most likely a sign of
250 congestive heart failure, which is in agreement with previously published studies
251 demonstrating that the primary presenting sign of cats with AF is decompensated
252 cardiac disease [9].

253 The contribution and relevance of arrhythmia-induced remodeling to cardiac disease
254 progression is poorly understood in cats. In dogs, SVT appears to be frequently
255 associated with structural heart disease (65% of cases in one study) [25]. It is still not
256 clear whether the structural changes seen in these cats are a consequence of pre-
257 existing myocardial disease or due to the presence of chronic arrhythmias resulting
258 in cardiac remodeling. Information regarding thyroid status and blood pressure were
259 inconsistently recorded and therefore secondary cardiomyopathy could not be
260 excluded in these cats. Hyperthyroidism is a known risk factor for the development of
261 atrial fibrillation and supraventricular tachycardias in people (prevalence varies from
262 2-20%) [26]. In people, achievement of a euthyroid state is typically associated with
263 restoration of sinus rhythm, especially in young patients and where duration of
264 disease is not long [26]. Thyroid status was variably determined in our population of
265 cats. Consequently, any association between hyperthyroidism and the presence of
266 supraventricular arrhythmias could not be assessed and is considered a limitation of
267 the study.

268 Left atrial diameter was used to assess LA size (in addition to LA:Ao) and was
269 significantly larger in cats with AF than cats with SVT. A critical mass of atrial tissue
270 is required to sustain the minimal number of circuits necessary to perpetuate AF [15],
271 and consequently, this arrhythmia is frequently associated with conditions (e.g.,
272 hypertrophic, dilated or restrictive cardiomyopathy) that cause left or right atrial

273 dilation. This finding is also reported in the Côté *et al.* (2004) study that identified
274 LAE in cats with concurrent AF [9]. Left atrial enlargement is associated with a poor
275 prognosis in cats with acquired heart disease, and consequently, AF has been
276 considered an end-stage event in cats with myocardial disease [27]. Left atrial size
277 did not have an impact on overall survival in our study. This may be due to the small
278 numbers of cats included in this population, as a larger study demonstrated a
279 measurable effect of LA size on outcome [16]. There is one report of lone AF in a cat
280 [5]. One cat with AF had normal LA size in our study, but in this cat, development of
281 AF was attributed to severe right atrial enlargement (the cat had a double-
282 chambered right ventricle and VSD).

283 The majority of cats with right-sided remodelling also had LAE. These changes could
284 represent primary bilateral ventricular pathology (e.g. hypertrophic cardiomyopathy
285 or arrhythmogenic right ventricular cardiomyopathy affecting both ventricles), or
286 might reflect remodelling consistent with a tachycardia-induced cardiomyopathy.
287 Tachycardia-induced remodeling is a well-established pathological sequela to rapid-
288 pacing in experimental models of cardiac failure in dogs and has also been
289 documented secondary to naturally-acquired tachyarrhythmia in dogs [28]. Sustained
290 tachycardia or paroxysms of any type of tachyarrhythmia affecting more than 15% of
291 the daily heart beats may result in tachycardia-induced cardiomyopathy in people
292 [29–31]. A single case report exists describing feline tachycardia-induced
293 cardiomyopathy [5], in which follow-up longitudinal echocardiographic assessment
294 showed progressive reduction in cardiac size in response to oral antiarrhythmic
295 therapy. Unfortunately, few cats in this study had echocardiographic assessment
296 prior to onset of the arrhythmia, longitudinal echocardiographic follow up and/or
297 Holter ECGs to demonstrate whether or not rate control was adequate.

298 The data presented here show that SVT can be documented in cats without
299 structural heart disease. In 2 of the 4 cats with no evidence of heart disease,
300 concurrent systemic disease was also documented (metastatic splenic
301 hemangiosarcoma, and chronic gastrointestinal disease of unknown origin
302 respectively.) Gastrointestinal signs are frequently reported in dogs with SVT [25],
303 however, it is not possible for us establish whether there is any link between the
304 rhythm disturbances in these cats and their concurrent systemic signs. Two cats
305 presented with paroxysmal supraventricular tachycardia and had no known co-
306 morbidities, though further characterisation of the SVT could not be achieved. A
307 potential mechanism for SVT in a young animal without structural heart disease is
308 macroreentrant tachycardia involving an accessory pathway, but the
309 electrocardiographic features for this condition have not been well described in cats.
310 Ventricular pre-excitation was not identified in any cat in this study and the current
311 practical limitations in performing diagnostic electrophysiological studies makes it
312 challenging to achieve a definitive diagnosis in cats with SVT [3].

313 The median HR obtained from the ECG recordings was found to be higher in cats
314 with SVT than AF (300 bpm and 220 bpm respectively), which is perhaps
315 unsurprising given the diagnostic criteria for this study for cats with SVT (i.e. the
316 presence of an sustained narrow complex tachycardia, with a regular R-R interval
317 and HR greater than 260 bpm). The median HR of 220 bpm in cats with AF was
318 similar to the rate that was published from a larger group of cats with this arrhythmia
319 [9]. It is interesting to note that medications aimed at reducing HR were uncommonly
320 prescribed to cats with AF in this population despite the fact that control of average
321 HR is considered to be an important therapeutic target in both people and dogs. It is
322 also impossible for us to conclude whether the administration of medication to these

323 'treated' cats had an influence on survival due to inconsistencies in treatment
324 recording in relation to ECG traces in the hospital and lack of subsequent re-
325 examinations following the introduction of treatment. Recently published
326 retrospective data in dogs has suggested that a lower average HR in dogs with AF is
327 associated with improved survival [32] and it is not known whether the same would
328 be true in cats. This study did not document a statistically significant association
329 between HR and survival in cats with AF or SVT, or when the population was
330 considered as a whole, although it is possible that this study was under-powered to
331 achieve statistical significance. Furthermore, the impact of medication on survival
332 remains unknown due to incomplete information available from the clinical records;
333 specifically, regarding the timing of medication administration in relation to
334 acquisition of the recorded ECG traces, doses of medications, lack of 24-hour Holter
335 ECG analysis and infrequency of re-examination following the introduction of
336 treatment.

337

338 In this group of cats, the documentation of transient ventricular arrhythmia on 6-lead
339 paper trace ECGs was not associated with worse survival. In recent studies
340 evaluating clinical risk markers for HCM in people, non-sustained ventricular
341 tachycardia proved to be a significant independent risk factor for SCD, especially in
342 the young [33]. We know from previous studies that cats with myocardial disease
343 have more frequent and complex ventricular arrhythmia than normal cats [34],
344 however further research, ideally with Holter ECG data and a larger number of cats,
345 would be required to determine whether these arrhythmias are associated with
346 increased risk of SCD or whether they influence long-term survival.

347 There were many limitations of this study, some of which relate to the inherent
348 challenges associated with the recording and interpretation of feline ECGs, including
349 low amplitude voltages, and motion/purring artifacts. At a HR > 260 bpm, short R-R
350 intervals can make it challenging to differentiate truly irregular rhythms from regular
351 rhythms [9], and some of the ECGs may have been incorrectly classified. In
352 particular, small p' (supraventricular depolarizations not arising from the sinoatrial
353 node) wave amplitudes made further classification of the SVT impossible in many
354 cases and so was not attempted in this current study. In some cases, irregular SVT
355 (e.g. due to multifocal atrial tachycardias or atrial flutter) may have been
356 misclassified as AF due to an inability to identify p' or flutter waves. Furthermore,
357 criteria to define SVT were extrapolated from those used in dogs due to lack of well-
358 established criteria in cats. An arbitrary rate of 260 bpm was used to define SVT;
359 however, it is possible that some of these cats may have had a physiological sinus
360 tachycardia. Conversely, some cats with a true SVT but a HR less than 260 bpm
361 may have been mistakenly excluded. Comparing cats with SVT to those with AF
362 carries with it a number of inherent limitations. Atrial fibrillation was presumed to be
363 sustained in all cats presenting with this arrhythmia. Holter ECGs were not
364 performed in any cat and therefore the frequency and duration of SVT is unknown.
365 Consequently, it is challenging for us to draw conclusions regarding the impact of
366 this arrhythmia on myocardial remodeling and possible CHF, for example, and
367 comparing survival data without Holter data therefore may be inappropriate.

368 Identification of ventricular arrhythmia from a 6-lead paper trace ECG is inferior to
369 Holter ECG assessment, and therefore underreporting of ventricular arrhythmia is
370 likely to be present in this group of cats. Further studies regarding the prognostic
371 significance of HR in the hospital and home environment of cats with SVT and AF

372 are warranted as this may provide additional information with regards to optimizing
373 therapy. Thoracic radiographs were not performed in all cats and therefore some
374 cats may have been misclassified regarding their CHF status.

375

376 Almost half of the cats diagnosed with SVT or AF according to the clinical records
377 system had to be excluded due to missing ECG records from the patient file. A
378 further 10 cats were removed as the storage discs containing the echocardiographic
379 images had become corrupted. This could affect the application of findings from this
380 study to a wider population of cats and importantly the small group sizes may have
381 limited the statistical power of the analyses. Further studies involving a larger
382 number of cats are therefore warranted.

383

384 **Conclusions**

385 Supraventricular tachycardia was as common as AF in our hospital population,
386 despite the paucity of case reports in the literature. All cats with AF had underlying
387 structural heart disease, whereas some cats with SVT had normal cardiac chamber
388 dimensions.

389

390 **Conflicts of interest statement:**

391 The authors do not have any conflicts of interest to disclose.

392

393 **Footnotes:**

394

395 a) Vivid 7, General Electric Medical Systems Ultrasound, 71 Great North Road,
396 Hatfield, AL9 5EN, United Kingdom.

397 b) Echopac, General Electric Medical Systems Ultrasound, 71 Great North
398 Road, Hatfield, AL9 5EN, United Kingdom.

399 c) BM SPSS Statistics 21.0 for Windows 7, IBM (UK) Ltd, Portsmouth, UK;
400 GraphPad Prism 6, GraphPad Software Inc, San Diego, CA

401

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522 **Figure 1.**

523 Kaplan-Meier curves to explore the difference in median survival time between cats
524 with supraventricular tachycardia (SVT) and cats with atrial fibrillation (AF)

525

526

527

528 **Figure 2.**

529 Kaplan-Meier curves to explore the difference in median survival time between cats
530 with and without signs of congestive heart failure (CHF)

531

Demographic findings			
Variable	AF (n=21)	SVT (n=23)	p-value
Male	16	16	0.7
Pedigree	4	6	0.7
Median age (months)	121 (40-174)	84 (6-196)	0.1
Median HR (bpm) established from paper trace ECG	220 (180-260)	300 (150- 380)	<0.001

Table 1. Demographic data for cats with supraventricular tachycardia (SVT) and atrial fibrillation (AF) grouped according to presenting rhythm diagnosis. HR: Heart rate

Presenting complaint	AF (n=21)	SVT (n=23)	p-value
No clinical signs	0 (0.0%)	2 (4.5%)	0.5
Respiratory distress	6 (13.6%)	4 (9.1%)	0.5
Lethargy	6 (13.6%)	3 (6.8%)	0.3
Collapse	2 (4.5%)	6 (13.6%)	0.2
Hindlimb paresis	3 (6.8 %)	2 (4.5%)	0.7
Ascites	2 (4.5%)	1 (2.3%)	0.6
Weight loss	1 (2.3%)	2 (4.5%)	>0.9
Cough	1 (2.3%)	0 (0.0%)	0.5
Inappetence	0 (0.0%)	1 (2.3%)	>0.9
Vomiting	0 (0.0%)	1 (2.3%)	>0.9
Weakness	0 (0.0%)	1 (2.3%)	>0.9

Table 2. Presenting signs of cats with supraventricular tachycardia (SVT) and atrial fibrillation (AF) grouped according to presenting rhythm diagnosis.

Echocardiographic findings			
Variable	AF (n=21)	SVT (n=23)	p-value
Normal heart	0	4	0.1
Congenital disease	1	1	>0.9
LVH	14	11	0.2
LAE	20	19	0.1
SEC/thrombus	5	5	>0.9
Right heart disease	6	1	0.04
Systolic dysfunction	4	4	>0.9

Table 3. Cats with supraventricular tachycardia (SVT) and atrial fibrillation (AF) grouped according to phenotypic findings on echocardiography. LAE: left atrial enlargement (LA:Ao >1.6 and/or LAD>16mm); LVH: left ventricular hypertrophy; SEC: spontaneous echo contrast

Echocardiographic findings			
Variable	AF	SVT	p-value
LVPWDd LAX (mm)	7.1 (3.6-10.5)	5.35 (3.9-12.1)	0.2
IVSd SAX (mm)	5.55 (3.1-8.4)	5 (3.7-8.5)	0.1
IVSd LAX (mm)	6 (3.9-9.0)	5.3 (4.1-9.9)	0.6
LVPWd SAX (mm)	6.16 +- 0.49	6.1 +- 0.40	0.9
LVID LAX (mm)	15.79 +- 0.72	14.98 +- 0.87	0.5
LA/Ao ratio	2.63 +- 0.16	2.22 +- 0.15	0.08
LAD (mm)	23.7 (16.1-40.1)	19.1 (12.8-31.4)	0.02
FS %	37.29 +- 4.92	38.88 +- 3.88	0.8

Table 4. Specific echocardiographic measures of left ventricular wall thickness, left atrial size and systolic function. FS%: fractional shortening; IVSd SAX: Interventricular septum in diastole, short-axis; LA/Ao: left atrial to aortic ratio; LVID LAX: left ventricular internal diameter, long-axis; LVPWDd LAX: left ventricular posterior wall diameter in diastole, long-axis; LVPWd SAX: left ventricular posterior wall diameter in diastole, short-axis.

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