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Flash pulmonary oedema associated with paroxysmal supraventricular tachycardia: report of two cases

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Abstract

Case summary We describe two cats that had episodic tachypnoea and increased respiratory effort during periods of paroxysmal supraventricular tachycardia (SVT). Thoracic radiographs at the time of clinical signs were consistent with cardiogenic pulmonary oedema. Echocardiography following stabilisation revealed a hypertrophic cardiomyopathy phenotype with normal left atrial size in both cats. The first cat was initially treated with diltiazem, but this did not reduce the frequency of the clinical episodes. Diltiazem was switched to atenolol and the cat remained well without further recurrence. At the time of writing, the cat was reported to be well, 3 years after the initial diagnosis of SVT. The second cat was first managed with diltiazem and was then transitioned to atenolol due to recurrent clinical episodes. The episodes were less frequent with atenolol but still present. Therefore, atenolol was changed to sotalol. The cat remained well on sotalol for 2 years with only one recurrent episode during a painful event. The patient then suffered a sudden cardiac death, 5 years after the initial diagnosis of SVT.

Relevance and novel information To our knowledge, this is the first report that describes flash pulmonary oedema developing secondary to episodic paroxysmal SVT in cats. Despite the severity and speed of respiratory compromise, prognosis may be good with an adequate arrhythmia control.

Keywords: Supraventricular tachycardia; arrhythmia; flash pulmonary oedema; respiratory compromise; dyspnoea; acute congestive heart failure; cardiomyopathy; echocardiogram; electrocardiogram; AliveCor

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Case description

Case 1

A 2-year-old female spayed domestic shorthair cat weighing 2.59 kg was presented to the Queen Mother Hospital for Animals with a 6-month history of episodic tachypnoea and dyspnoea. Each episode lasted for 24 h and occurred weekly without antecedent triggers. During one of the symptomatic episodes, the cat was diagnosed with congestive heart failure (CHF; see Figure 1) due to hypertrophic cardiomyopathy (HCM) based on thoracic radiographs and echocardiogram. Furosemide (2 mg/kg PO q8h), clopidogrel (18.75 mg/cat PO q24h) and spironolactone (2 mg/kg PO q24h) were initiated. However, no improvement in severity or frequency of episodic respiratory distress was observed. A beclometasone (100 µg) inhaler was trialled for a week without improvement. A referral was arranged.

On presentation, the cat was tachypnoeic (60 breaths/min), with normal respiratory effort. Auscultation revealed clear lung sounds, a heart rate (HR) of 248 beats/min (bpm) and a grade II/VI sternal systolic murmur. An echocardiogram showed an HCM phenotype with normal left atrial (LA) size. The LA wall was

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subjectively thickened (Figure 2; see Table S1 in the supplementary material). A six-lead electrocardiogram (ECG) was performed and showed normal sinus rhythm.

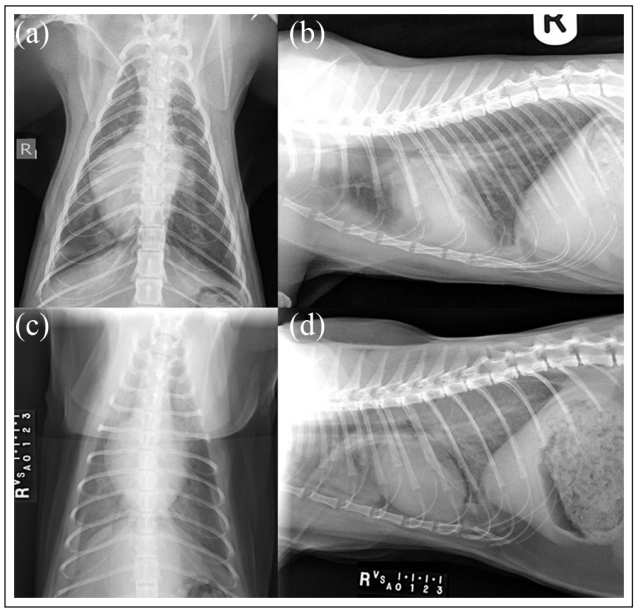

Figure 1 Radiographs during one of the episodes of tachypnoea and dyspnoea in (a,b) case 1 and (c,d) case 2. (a,c) Dorsoventral and (b,d) right lateral radiographs show pulmonary oedema consistent with congestive heart failure

Furosemide was reduced (1 mg/kg q12h), clopidogrel and spironolactone were discontinued and a 24 h Holter monitor was fitted.

The cat had two episodes of short-lasting tachypnoea and dyspnoea during the Holter recording, which were managed with oxygen supplementation and additional furosemide treatment. The Holter recording revealed paroxysmal supraventricular tachycardia (SVT) at 270–300 bpm during both episodes. Furosemide was discontinued, and diltiazem (1.8 mg/kg PO q8h) was initiated. The cat was discharged and a repeat 24 h Holter assessment 2 weeks later was recommended but declined due to cost and the cat's nervous temperament. Instead, the owner agreed to start using a mobile ECG device (KardiaMobile; AliveCor) and to administer a 1 mg/kg emergency furosemide dose during another respiratory episode. Two weeks later, the cat had a short-lasting tachypnoea and paroxysmal SVT episode (Figure 3). The owner reported that the cat was stressed by children before developing clinical signs, so decided to attempt preventing stressful events without antiarrhythmic therapy adjustment.

Another SVT episode occurred 1 month later, persisting for >3 h. The cat was admitted to hospital and treated with a single dose of esmolol 0.1 mg/kg IV (4 h after the diltiazem dose). Immediately after esmolol administration, the cat developed severe bradycardia with sinus arrest/block and ventricular escape complexes at 80 bpm

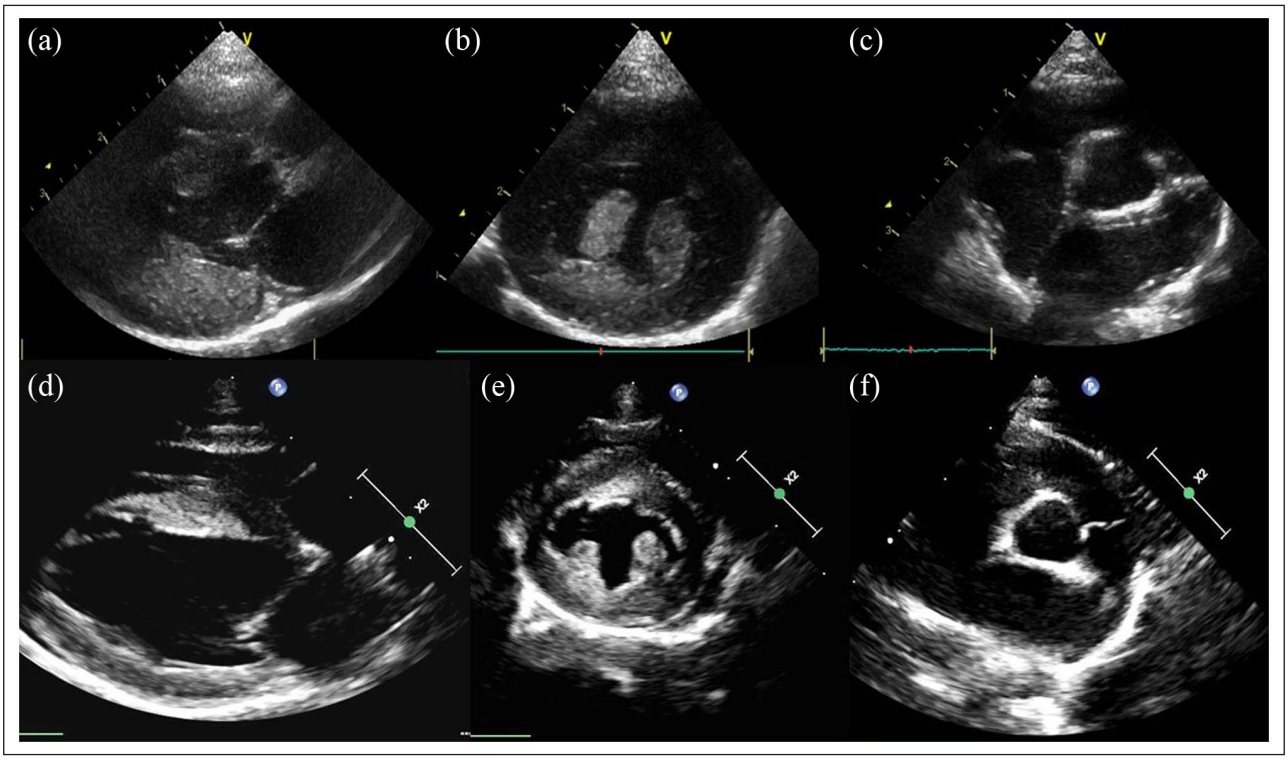

Figure 2 Still images taken at end-diastole from the echocardiographic evaluation of (a–c) case 1 and (d–f) case 2: (a,d) four-chamber right-sided parasternal long-axis view; (b,e) right-sided parasternal short-axis view at the level of the papillary muscles; and (c,f) right-sided short-axis view at the level of the left atrium and aorta

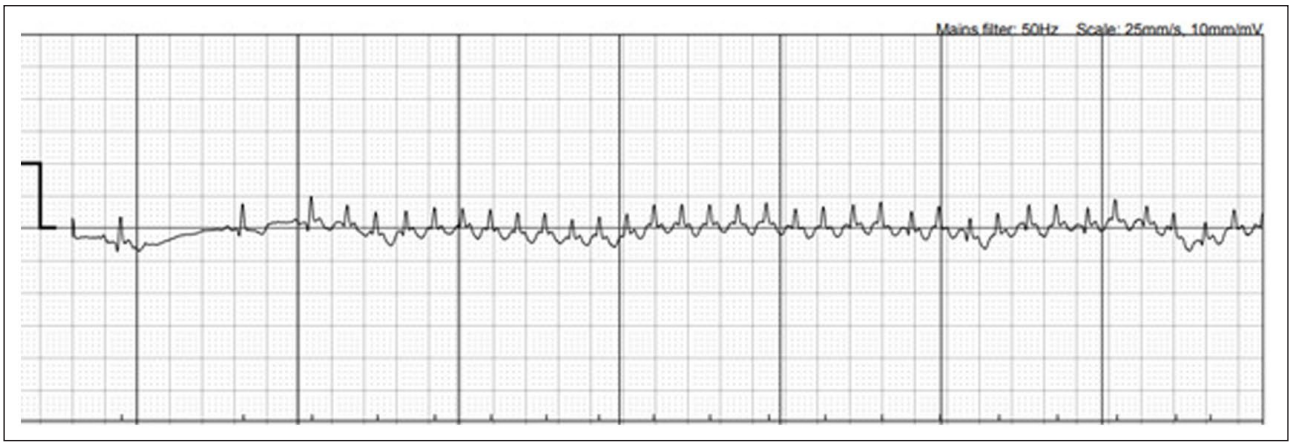

Figure 3 A single-lead ECG tracing obtained by the owner using a mobile ECG (AliveCor) device. Note the sinus rhythm converting into paroxysmal supraventricular tachycardia. Paper speed = 25 mm/s; sensitivity 10 mm/mV

for 10 s before resuming sinus rhythm at 120–140 bpm. Diltiazem was discontinued and atenolol was initiated (2.2 mg/kg PO q12h). The cat was monitored for 12 h and discharged. At a 6-month follow-up, LA enlargement was observed. Clopidogrel (18.75 mg/cat PO q24h) was reinitiated. The patient remained well, 3 years after initiating atenolol.

Case 2

A 3-year-old male neutered Ragdoll cat weighing 5.5 kg was presented to the Animal Referral Centre for episodic tachypnoea and dyspnoea (see Video 1 in the supplementary material). The cat developed the first respiratory episode at 8 months of age and was diagnosed via radiographs with CHF (Figure 1). Furosemide (0.5 mg/kg PO q12h) was initiated. One week later, echocardiography showed an HCM phenotype with normal LA size. Furosemide was discontinued, and within 36 h, the cat developed a recurrent respiratory episode. This time, severe tachycardia (HR >300 bpm) was detected. An ECG revealed SVT. Diltiazem (1 mg/kg PO q8h) and furosemide (1 mg/kg PO q12h) were initiated; however, these did not prevent respiratory episodes, necessitating weekly hospitalisation. Diltiazem was changed to atenolol (6.25 mg/cat PO q12h) which reduced episode frequency, but the tachypnoea and dyspnoea were of progressive severity. Serum cardiac troponin I concentration was increased (2.11 ng/ml; reference interval <0.2–0.25). The cat was negative for feline immunodeficiency virus antibody and feline leukaemia virus antigen negative on a SNAP test (IDEXX). A single toxoplasma antibody titre returned as a weak positive (1:64), which was not suggestive of current infection given the high prevalence of toxoplasmosis in New Zealand. A cardiac referral was arranged.

The physical examination was unremarkable. An echocardiogram showed an HCM phenotype with normal LA size (Figure 2; see Table S1 in the supplementary material). During the echocardiogram, the cat developed paroxysmal SVT at 300 bpm. A vagal manoeuvre (pressing the nose) was ineffective. Intravenous antiarrhythmics were unavailable at the time and the cat was treated with oxygen and single doses of furosemide (2 mg/kg IV), sotalol (3.5 mg/kg PO) and butorphanol (0.5 mg/kg IV). The cat's respiratory signs and SVT resolved within 1 h. A 24 h Holter monitor and smartphone-based ECG (KardiaMobile; AliveCor) device were recommended but not elected. The cat was discharged on sotalol (3.5 mg/kg PO q12h).

The owner reported no further episodes until 1 year later. The cat re-presented with an HR of 320 bpm and recurrent SVT (Figure 4), tachypnoea, signs of cardiogenic shock (subdued mentation, pale-pink mucous membranes and absent femoral pulses with cold extremities) and severely matted fur covering approximately 80% of the abdomen. The cat was administered oxygen, butorphanol (0.2 mg/kg IM) and midazolam (0.2 mg/kg IM). Following a brief echocardiogram, which showed a normal LA and left ventricular (LV) size, two doses of IV metoprolol were administered (0.02 mg/kg and 0.04 mg/kg, respectively, 5 mins apart). The first metoprolol dose did not change the ECG. The second dose successfully converted back to sinus rhythm at 120 bpm following a brief period of ventricular ectopics. The cat quickly made a dramatic improvement in mentation and perfusion parameters. Continuous ECG monitoring for the following 20 mins showed sinus rhythm without arrhythmia. A complete echocardiogram was then performed, which showed mild dilation of the left and right ventricles secondary to bradycardia, without other significant changes. As pain and stress from the matted fur were perceived to have caused the episode, the fur was clipped and medications were unchanged.

The cat presented for a routine recheck 5 months later (5 years after the initial SVT diagnosis) without

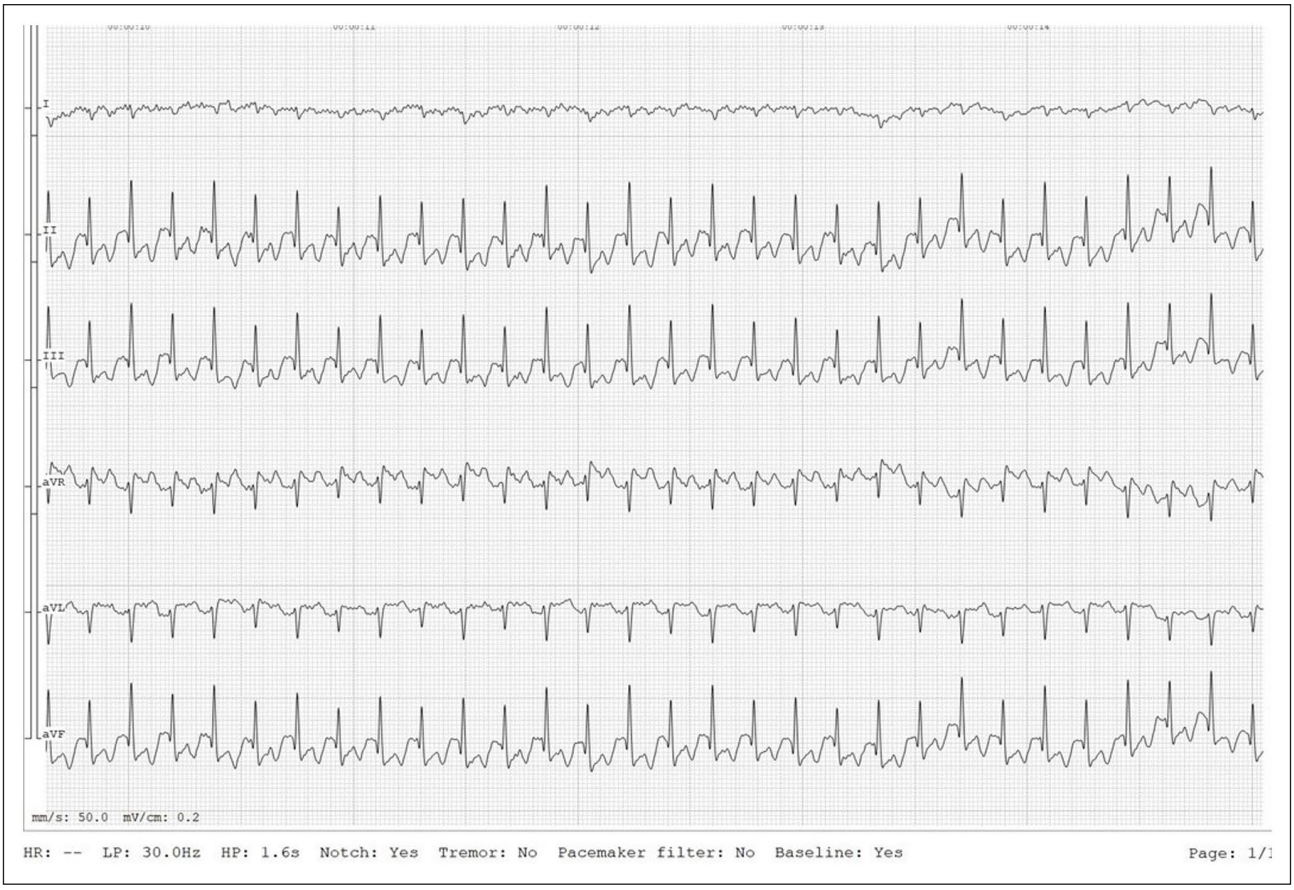

Figure 4 Six-lead electrocardiogram tracings obtained from case 2 shortly after presentation at the referral hospital displaying continued narrow complex tachycardia (supraventricular tachycardia) with a heart rate of 320 bpm. Paper speed 50 mm/s; sensitivity 0.5 cm/mV

further SVT episodes. On examination, the cat had an HR of 100 bpm with sinus bradycardia on an ECG trace, and a systolic blood pressure of 140 mmHg. Serum creatinine and lactate were normal. Echocardiogram showed a stable HCM phenotype, compared with previous assessments. Pacemaker implantation was discussed as the long-term effects of bradycardia are unknown, but this was not pursued. Continuing sotalol was advised. The cat remained well but ultimately suffered a sudden cardiac death 5 months later. Postmortem examination was not performed.

Discussion

To our knowledge, flash pulmonary oedema (FPO) due to paroxysmal SVT has not been previously reported in cats. The term FPO is better described in human medicine, where it is characterised by a sudden onset of increased LV filling pressure, leading to severe pulmonary oedema within minutes.[1,2] In people, cardiac causes of FPO range from arryhthmias, hypertensive crises, myocarditis, acute coronary syndrome, acute aortic regurgitation and stress (Takotsubo) cardiomyopathy.[2] In cats, the term FPO is not well utilised, but cardiac differentials include cardiomyopathies,[3,4] transient myocardial thickening,[5,6] supra/ventricular arrythmias,[3] hypertension,[7,8] myocardial ischaemia or infarction.[4,5]

The fast HR owing to SVT is considered the primary mechanism of FPO in both cats. The resolution and lack of recurrent FPO following adequate SVT management supports this explanation. However, not every cat with an HR of 270–320 bpm will develop similar severity of FPO as the cats in this report. Both cats had an underlying HCM phenotype, and it is possible that the impaired LV function contributed to the rapid increase in the LV filling pressure and hydrostatic capillary pressure following SVT onset. Whether cats with a normal echocardiogram can develop FPO secondarily to a similar SVT rate to this report is unknown.

The prognosis of FPO in people depends on the underlying cause.[1,2,8] Delayed FPO diagnosis is common in people with episodic respiratory difficulties[8] and, based on our report, is likely similar in cats. Our cases demonstrate that diagnosing paroxysmal arryhthmias can prove difficult, as ECG recording during symptomatic episodes is necessary. For example, our first case had weekly respiratory episodes over a 6-month period,

and the second case had 14 episodes over 4 years before being diagnosed with SVT. We diagnosed SVT because both cats had persistent SVT during hospitalisation. In those with shorter durations of SVT, either a longer duration of Holter monitoring, loop recorder[9] or a smartphone-based ECG device (KardiaMobile; AliveCor)[10,11] may be required. In our case, a smartphone-based ECG was effective, as it provided a long-term, at-home monitoring tool.

The term SVT describes a tachycardic rhythm disturbance originating from structures within or above the atrioventricular (AV) node.[12] Arrhythmias under this term include multi/focal atrial tachycardias, atrial flutter, sinus and AV nodal re-entrant tachycardias, AV accessory pathway tachycardias and junctional tachycardia.[12] In humans, SVT is generally idiopathic, whereas in dogs, the majority (65%) have structural heart disease.[12,13] In contrast, there is paucity of feline SVT studies, with descriptions limited to case reports[9,13–20] and one retrospective study.[21] In the study, the most common presenting clinical sign of SVT was respiratory distress, and the diagnosis of CHF was associated with shorter survival times, in a univariate analysis.[21] However, our report suggests there may be a subgroup of cats where CHF is secondary to SVT, and can be treated by adequate SVT management. Indeed, the retrospective study documented that a small number of cats with SVT had structurally normal hearts.[21] Whether these cats followed a similar clinical course to our cases is unknown.

Treatments provided in our cases can be divided into FPO (CHF) and SVT management. Management of FPO was achieved through supplemental oxygen and furosemide. The FPO was not persistent in either case, as SVT was the triggering factor, which became well-managed on antiarrhythmic medications. Therefore, furosemide therapy was not required in the long term. Treatment of SVT considered the underlying cardiac size and function, antecedent events, likely SVT mechanism/type, drug availability and clinicians' experience. Beta blockers were more successful than diltiazem, perhaps because of the high sympathetic tone that was a triggering, modulating SVT factor and that the SVT mechanism was not AV node-dependent. For example, in case 1, children induced stress; in case 2, stress resulted from severely matted fur. Sympathetic activation from stress can cause significant effects on cardiac myocyte electrophysiology.[22] Nevertheless, diltiazem was quickly switched to atenolol in both cases and whether a higher dose of diltiazem, or an alternative calcium channel blocker such as verapamil, would have achieved a similar outcome is unknown.

Sotalol was more effective in preventing SVT than atenolol in case 2, suggesting that the additive potassium channel blocking effect is useful in certain SVT types, as in people and dogs.[9,23–25] However, this could also be attributable to the high dose used (owing to New Zealand's limited availability of antiarrhythmics). While the resultant HR of 100 bpm did not cause changes in perfusion or clinical signs, the long-term effect of feline bradycardia is unknown. Therefore, a pacemaker implantation was discussed, although not elected by the family as the cat seemed well. This cat ultimately suffered a sudden death. Possible explanations include arrhythmias that quickly degenerated into asystole or ventricular fibrillation, or other HCM-related complications, including myocardial infarction or a thromboembolic event.

Other SVT treatment options, including electrical cardioversion, alternative calcium channel blockers and lidocaine, may have been considered by different clinicians and their country's drug availability. The authors caution readers from generalising SVT management with this case report. Clinicians must be aware of adverse effects, especially when combining drugs. For example, case 1 developed severe bradycardia when esmolol (beta blocker) was administered after the cat received diltiazem (calcium channel blocker). The aim of this case report is to highlight the association of SVT and FPO, and not to provide a guideline for feline SVT management.

Both cats were diagnosed with an HCM phenotype, with case 1 also having a thickened LA (signifying a possible atrial myopathy). An obvious LA thickening was not observed in case 2. As previously mentioned, the underlying HCM phenotype may have contributed to a faster development of FPO. The exact cause of SVT, whether it was previous infection, inflammation or underlying HCM phenotype could not be established in this report based on available information. However, SVT control did not prevent the HCM phenotype progression in case 1.

Conclusions

We describe the clinical management of two cats that developed FPO due to paroxysmal SVT. Despite the severity and speed of respiratory compromise, successful SVT management can provide a good long-term prognosis.

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Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards

('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*. Although not required, where ethical approval was still obtained, it is stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers) for all procedure(s) undertaken (prospective or retrospective studies). For any animals or people individually identifiable within this publication, informed consent (verbal or written) for their use in the publication was obtained from the people involved.

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Supplementary material The following files are available online:
Supplementary Video 1: Presentation of case 2.
Table S1: Echocardiographic measurements from cases 1 and 2.

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