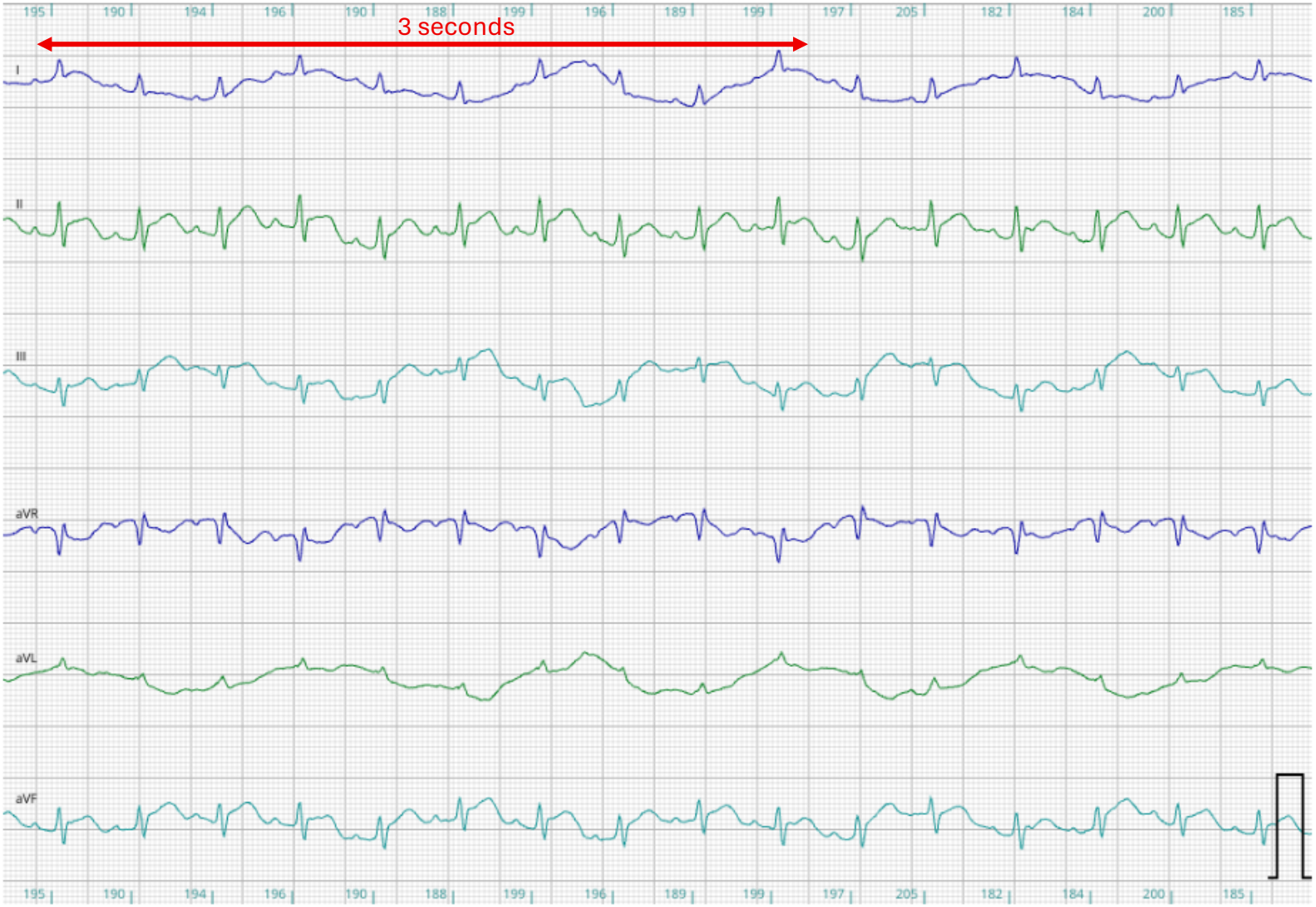
Case 1.

Signalment: 6yo MN DSH Cat, 4.4kg
History: No clinical signs, gallop sound heard on pre-anaesthesia examination.
Physical examination findings: Heart rate 160-180bpm, regular, no murmur, soft gallop sound over left sternal border, femoral pulse quality normal, abdominal palpation and thoracic auscultation unremarkable, no goitre, retinal examination unremarkable.

50mm/s, 20mm/mV



Case 1 Answers

Rhythm diagnosis: Sinus rhythm (sinus tachycardia)

Interpretation: The rhythm is sinus at a rate of ~190bpm with a regular rhythm and normally sized and associated PQRST complexes. The QRS complex is small, as is typical in cats, hence the amplitude has been increased to 20mm/mV (standard in dogs is 10mm/mV). The axis is normal, though this is difficult to tell in cats due to them often having small complexes. Axis deviations are common in cats and rarely of clinical significance. There is no ectopy, no premature complexes and no bradycardia. There is some baseline (movement) artefact, probably from movement in the left forelimb as lead II (which connects the right fore and left hind limbs) is reasonably flat.

Conclusions based on case particulars: The rhythm is normal. There is nothing on this ECG to suggest underlying disease but nor is there anything to exclude it. ECGs are poorly sensitive for detecting heart disease. In the context of a cat with a gallop sound, further investigation is warranted.

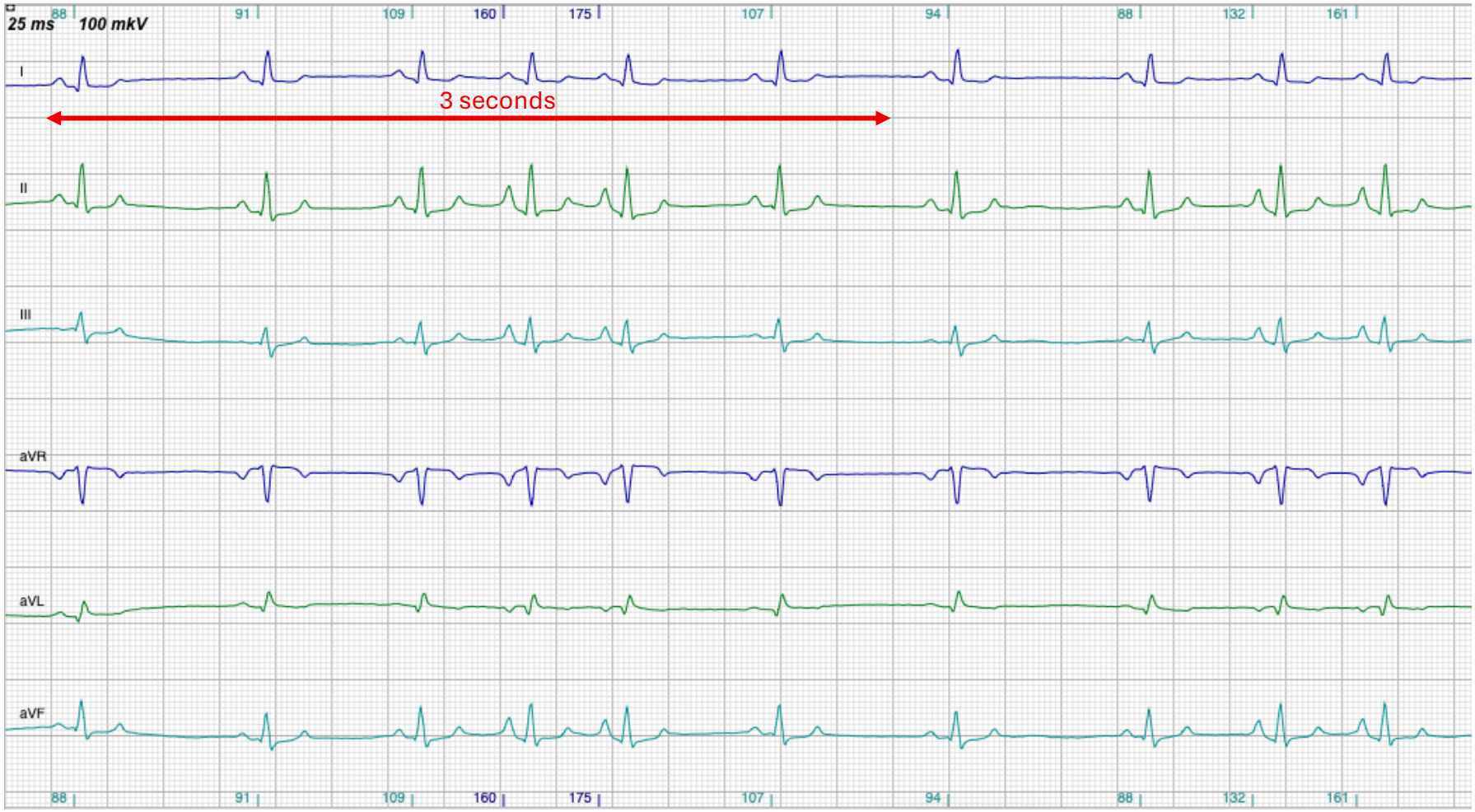
Next steps: Echocardiography is the test of choice to investigate the possibility of structural heart disease. Gallop sounds are reasonably specific for identifying heart disease and so further investigation is certainly warranted, particularly given the plan to perform general anaesthesia. If echo is not available then proBNP (either quantitative or SNAP) is a reasonable second choice test – in a cat of this age, a negative proBNP suggests significant underlying heart disease is not likely. Systemic hypertension, renal disease and hyperthyroidism are all unlikely at this age and so should only be tested for if there are appropriate clinical signs.

Therapy: No treatment is indicated for this rhythm.

Case 2.

Signalment: 7yo FN CKCS, 8.4kg
History: Murmur auscultated on recent examination. Slowing on walks, otherwise well.
Physical examination findings: Heart rate 90-160bpm, regularly irregular, soft left-apical systolic murmur. Femoral pulse quality normal. Abdominal palpation and thoracic auscultation unremarkable. Remaining examination unremarkable.

50mm/s, 10mm/mV



Case 2 Answers

Rhythm diagnosis: Sinus arrhythmia

Interpretation: The rhythm is sinus at an average rate of ~110bpm (instantaneous rates vary between 88 and 175bpm) with a regular rhythm and normally sized and associated PQRST complexes. The RR interval varies but there are no premature complexes (nothing occurs faster than an expected sinus rate of ~200bpm) and no long pauses (nothing longer than ~2 seconds). There is a subtle change in the P-wave amplitude, known as 'wandering pacemaker' which is associated with a change in heart rate; confirming this is a normal sinus arrhythmia. There is no premature activity nor any bradycardia.

Conclusions based on case particulars: Sinus arrhythmia is normal in dogs and in some cases can sound quite extreme on examination, particularly in dogs with elevated vagal tone

Next steps: In a dog with a murmur, echocardiography is indicated to further explore the potential for heart disease. Thoracic radiographs or proBNP can be used to exclude significant heart disease but are not very sensitive nor specific.

Therapy: No treatment is indicated for this rhythm.

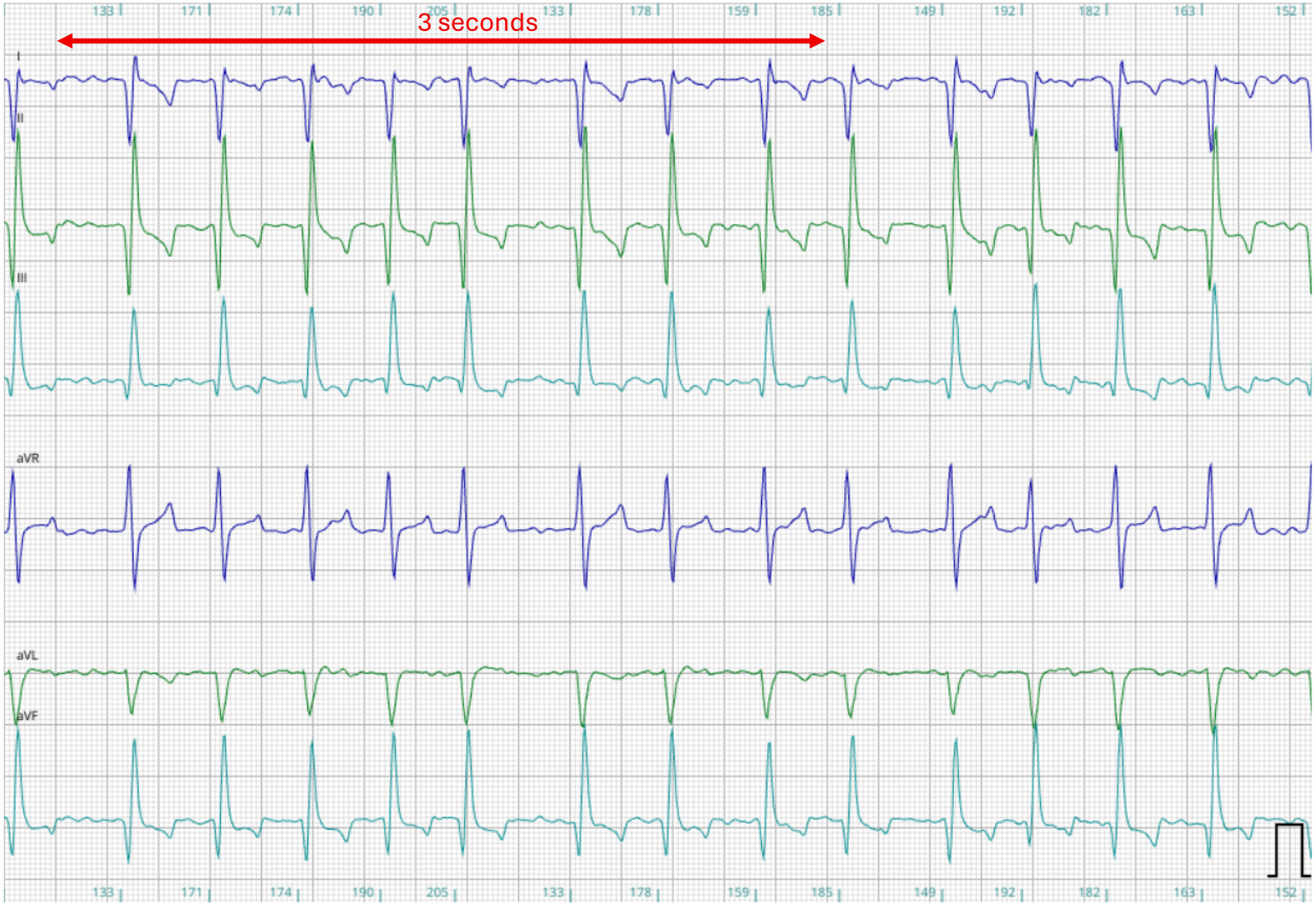
Case 3.

Signalment: 10yo ME Irish setter, 35kg

History: arrhythmia picked up on routine examination. No clinical signs of heart disease. No coughing or collapse reported.

Physical examination findings: Heart rate 164bpm with irregularly irregular (chaotic) rhythm, no audible murmur, slightly weak and variable femoral pulse quality with some deficits. Basic echo performed- DCM phenotype with mild mitral valve regurgitation. LA/Ao 1.45 (normal <1.65), LVDDN 1.7 (normal <1.9).

50mm/s, 10mm/mV



Case 3 Answers

Rhythm diagnosis: Atrial fibrillation with a ventricular response rate of 160bpm.

Interpretation: This ECG shows a narrow complex irregular tachycardia with absent P waves and variable RR interval, consistent with atrial fibrillation. In some places there appear to be more coarse flutter waves present, but this is not sufficiently consistent to suspect an atrial flutter with intermittent conduction. R wave amplitude is variable but within normal limits. There are no ventricular premature beats. The QRS is mildly prolonged.

Conclusions based on case particulars: This heart rate and rhythm in association with the clinical history would most likely be associated with underlying structural heart disease causing significant left atrial enlargement. In this breed degenerative mitral valve disease is most likely, though DCM is also possible, differentiating the two on echo (particularly at elevated heart rates) is tricky. It is rare to have a fractional shortening of 25% in a primary DCM case, unless there is torrential mitral regurgitation. Hypothyroidism or elevated vagal tone from extra-cardiac disease can rarely be associated with development of AF but this would be considered unlikely given the history and signalment.

Next steps: Thoracic radiography may be useful to assess for cardiomegaly and presence of pulmonary venous congestion/ pulmonary oedema. However, sleeping respiratory rate at home is a good predictor of the presence of pulmonary oedema which can help to determine if congestive failure is likely to be present or imminent, and help to guide therapy.

Ambulatory ECG (Holter monitoring) is useful to assess heart rate away from the clinic and screen for ventricular tachyarrhythmia. I would ideally do this rather than add rate control in this case where heart rate is higher than ideal in the clinic but not severely elevated. Holter monitoring gives us a baseline heart rate from which to guide treatment. Typically, we start treatment once the in-clinic rate is above 160bpm and aim for an in-clinic rate of <160 on treatment, but in a stressed/nervous dog it's difficult to know what the true resting rate is without Holter.

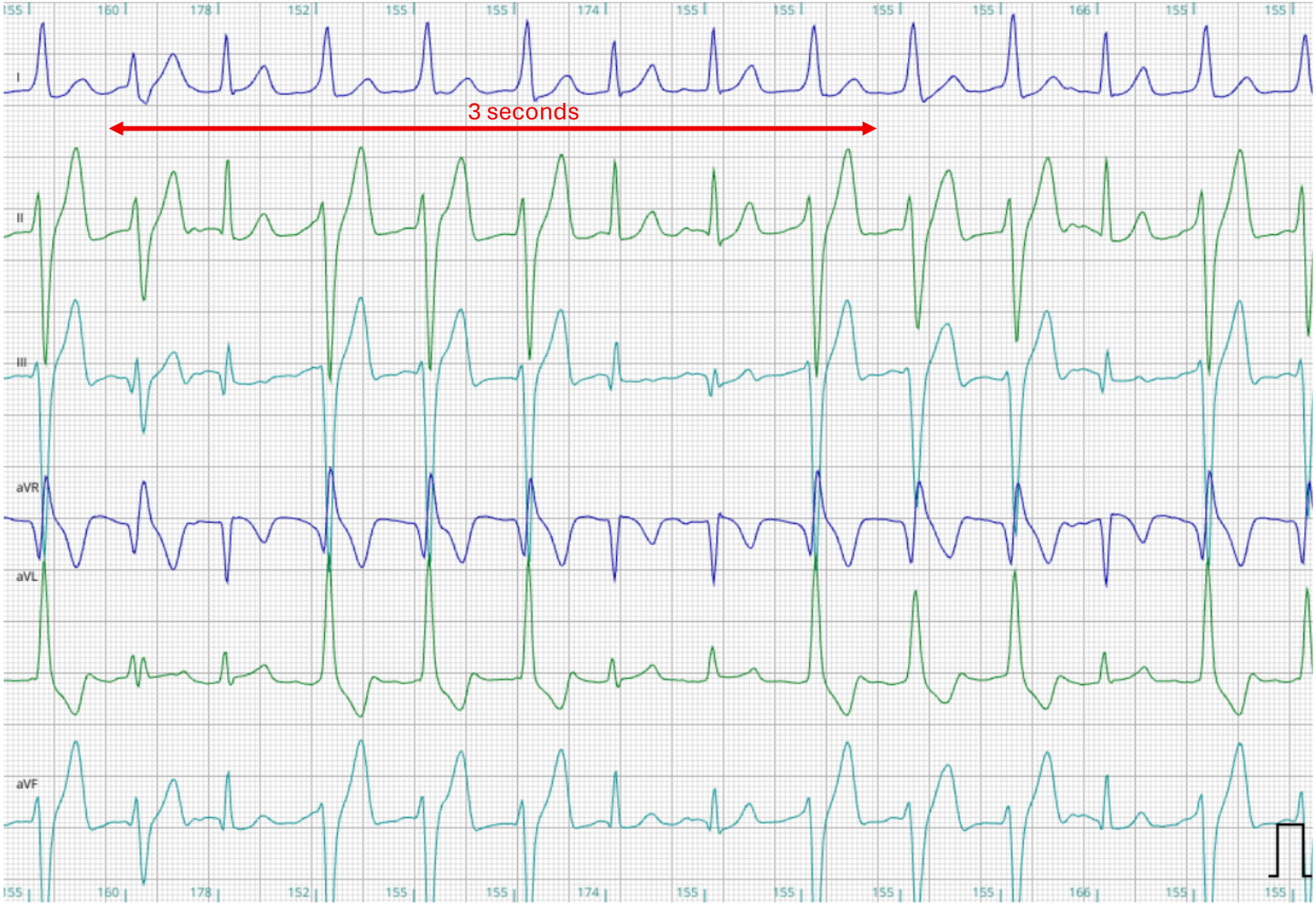
Therapy: If further investigation is not financially viable then I would advise dilitazem at 2mg/kg (modified release) every 12 hours. I wouldn't use digoxin yet - pending a Holter - but would add this in for secondary rate control at 3-5ug/kg every 12 hours if diltiazem is insufficient on it's own to achieve rate control. Monitoring SRR can guide whether or not to introduce frusemide (<30breaths per minute is normal), with truly normal dogs being in the teens, and those nearing onset of heart failure in the high 20s. >30-35br/min suggests an increased risk for the presence of pulmonary oedema.

If you treatment is started, I would Holter 3 weeks after starting to see how well the heart has responded to rate control and to screen for pro-arrhythmia.

Case 4.

Signalment: 10yo MN Crossbreed dog, 38.5kg
History: Surgical splenectomy following splenic rupture. Complicated anaesthesia. Blood transfusion given. Lidocaine CRI currently being given.
Physical examination findings: ECG performed on recovery, examination findings not available.

50mm/s, 10mm/mV



Case 4 Answers

Rhythm diagnosis: Accelerated idioventricular rhythm

Interpretation: A regular wide-complex rhythm predominates with absent P waves. There are occasional sinus capture beats are present (the narrow complex beats preceded by P waves). This an accelerated idioventricular rhythm (also sometimes called a "slow ventricular tachycardia").

Conclusions based on case particulars: Although this rhythm is "wide and bizarre", the instantaneous rate of the ventricular beats varies between 155-160bpm, which is not a tachycardia therefore this can't be termed a 'ventricular tachycardia'.

Accelerated idioventricular rhythm (AIVR) is not usually dangerous or unstable as such, however it usually denotes significant underlying disease. The most common cause of this arrhythmia is often non-cardiac disease, most often abdominal disease eg splenic or hepatic neoplasia, haemorrhage, pancreatitis etc. Anaemia, hypokalaemia and trauma are other potential causes. Cardiac causes include primary myocardial disease (dilated cardiomyopathy), pericardial effusion, or rarely myocardial inflammation/infection (myocarditis). The splenectomy and surgical complications are certainly the underlying cause here.

To help differentiate AIVR from VT (aside from using the heart rate, which is typically around 160bpm and always under 200bpm in AIVR and is usually over 200bpm in VT), blood pressure is helpful as AIVR rarely causes hypotension.

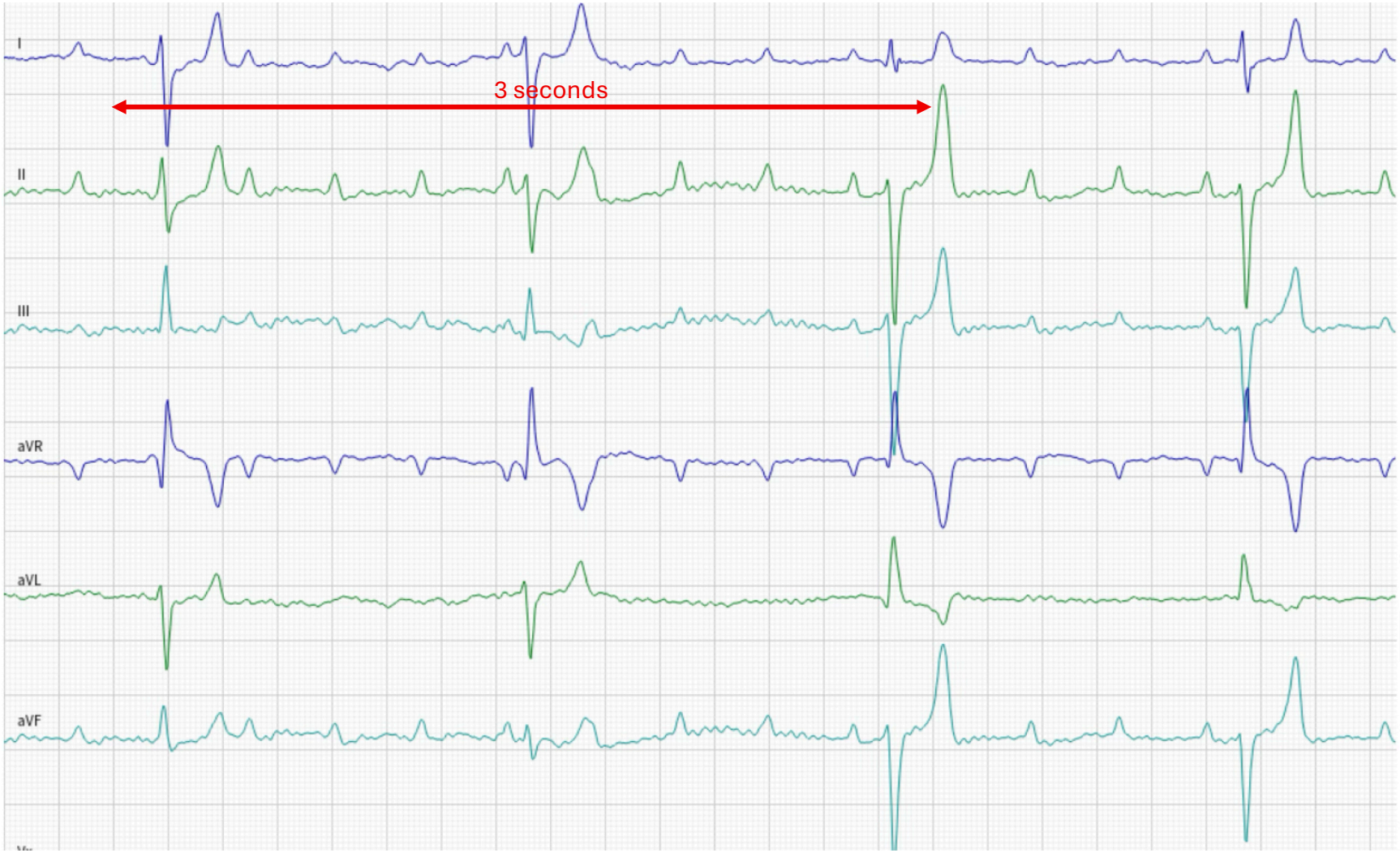
Next steps: If the patient is clinically stable, no further investigation is needed and supportive care should be provided as appropriate. In most cases this rhythm with self-resolve within 24-48h. If it still persists 10-14 days post surgery, or should the patient develop signs of hypotension, shock or cardiac crisis then further investigation is warranted at that point.

Therapy: None needed. Lidocaine is usually unsuccessful at treating this arrhythmia and so the CRI should only be continued if indicated for other causes (e.g. pain relief).

Case 5.

Signalment: 9yo FN Miniature schnauzer, 6.9kg
History: Collapse episodes, unresponsive to anti-epileptic medication.
Physical examination findings: Heart rate 44bpm, soft left apical systolic murmur, strong and synchronous femoral pulse quality. . Normal abdominal palpation, normal lung auscultation, remaining examination unremarkable.

50mm/s, 10mm/mV



Case 5 Answers

Rhythm diagnosis: Third-degree atrioventricular block (complete heart block)

Interpretation: The P waves and QRS complexes are unrelated – the P rate is 185bpm and this continues throughout recording although in some cases the P waves are buried in QRS complexes or T waves. The QRS complexes are wide and bizarre and also occur at a steady rate of around 44bpm due to a subsidiary pacemaker, most likely of ventricular origin with this morphology. There is no evidence of conduction between the atria and ventricles, hence this is 3rd degree AV block.

Conclusions based on case particulars: This rhythm is certainly likely to be associated with the reported clinical signs. It is most likely that this ventricular escape rate (44bpm) is tolerated at rest / gentle exercise but does not allow increased cardiac output during exercise.

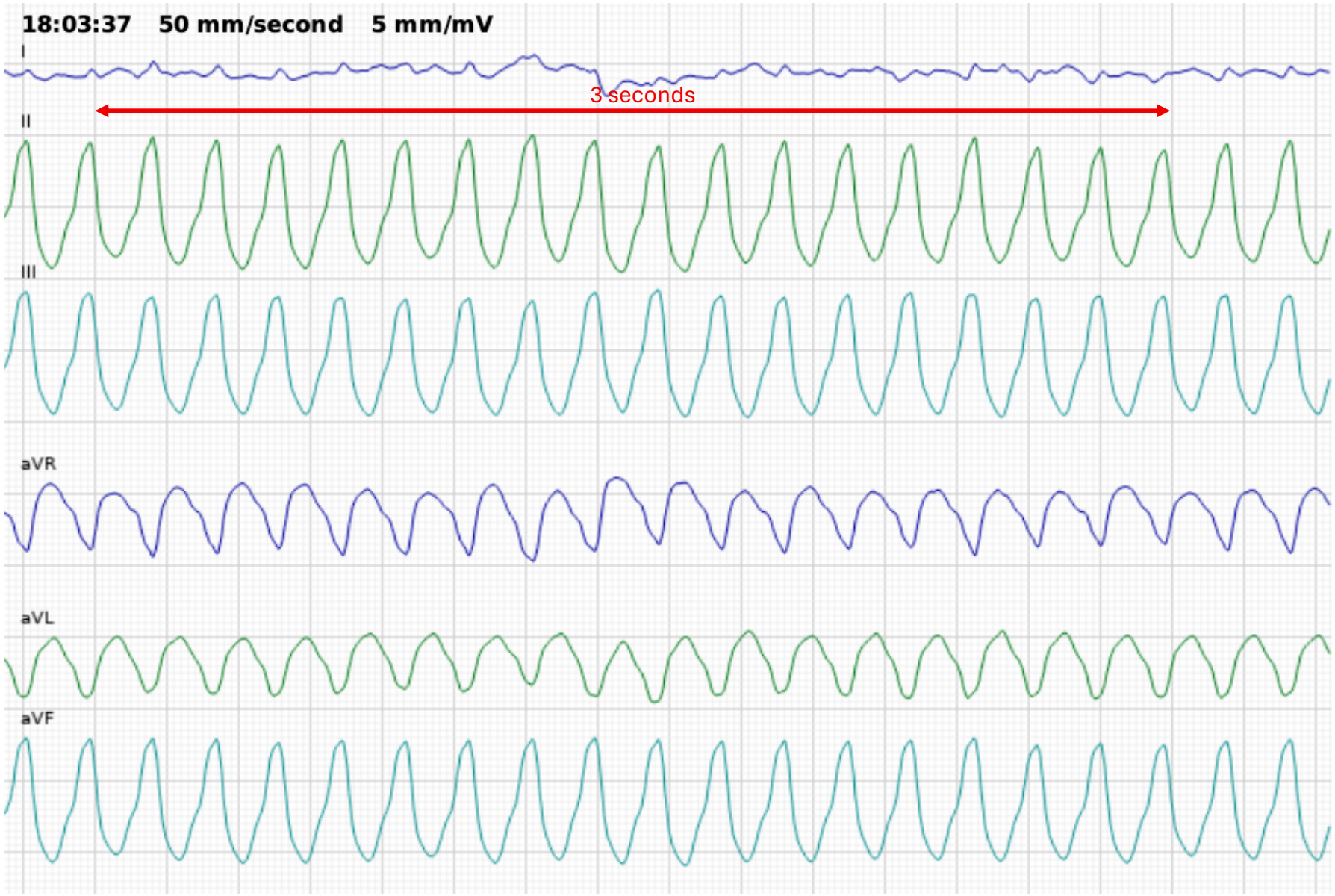
Next steps: 3rd degree AV block in dogs is usually secondary to age-related degeneration and fibrosis disrupting the conduction pathways. Rare causes of 3rd degree AV block include infectious/inflammatory myocarditis, or neoplastic infiltration. If echocardiography has not been performed then this would be a good idea, to look for primary myocardial or inflammatory/infiltrative disease. Serum cardiac troponin-I may be useful to look for an inflammatory basis (usually severely raised in cases of myocarditis). Infectious causes are very unusual although reported in association with Lyme's disease (Borrelia) infection. If troponin is severely elevated then Borrelia, toxoplasma +/- neospora serology should be considered. We routinely advise excluding hypothyroidism and hypoadrenocorticism as both endocrinopathies can cause AV block.

Therapy: In symptomatic patients with no evidence of underlying inflammation/infection then pacemaker implantation is usually very effective in palliating clinical signs. Where cost precludes this medical therapy with pimobendan (0.25-0.3mg/kg every 12 hours) can help – the positive chronotropic and inotropic qualities can improve cardiac output. Theophylline (positive chronotrope) can also be considered to try and increase ventricular rate, using standard doses. Sadly ,this is usually a progressive condition and does carry a risk of sudden cardiac death, though medical therapy helps some patients for a period of time. Low-output congestive heart failure is an uncommon but possible sequel hence any breathlessness or cough should be reported and investigated with echocardiography/thoracic radiography.

Given the slow ventricular escape rate, referral for pacemaker assessment, if feasible, should be done on an urgent basis.

Case 6.

Signalment: 4yo ME English bulldog, 23kg
History: Known history of pulmonic stenosis, presented yesterday with lethargy and tachypnoea.
Physical examination findings: HR 340bpm with regular rhythm. Weal femoral pulses. Pale membranes, tachypnoea (RR 44).



Case 6 Answers

Rhythm diagnosis: Wide complex tachycardia at 340bpm

Interpretation: There is a saw-toothed waveform at a rate of 340bpm (matching with the clinical examination), wide-complexes, with no return to baseline.

Conclusions based on case particulars: The differentials for this rhythm are an aberrantly conducted supraventricular tachycardia (SVT) or a ventricular tachycardia. This is a life-threatening emergency and needs urgent treatment. Unless the rhythm is interrupted by sinus complexes and the onset/offset of the tachycardia can be identified, exact determination of the primary rhythm is not possible, but in this breed with known heart disease, assuming it is a ventricular tachycardia is reasonable.

Next steps: Place an intravenous cannula and give lidocaine, 2mg/kg bolus, with simultaneous ECG monitoring. If there is no response to repeated boluses (maximum of 4 boluses, 8mg/kg total, spaced 5 minutes apart) consider alternative therapy (intravenous esmolol, procainamide, precordial thump).

Therapy: See comments above. Urgent cardiologist advice should be sought as soon as possible.

This case responded well to a single bolus of intravenous lidocaine and was then given oral mexiletine. Cardiac investigation found right ventricular failure secondary to pulmonic stenosis.