Title Page

CWA Number: 1

Title: Ventricular tachycardia in an Irish Sport Horse.

Summary: Sustained monomorphic ventricular tachycardia was detected by electrocardiography in a 9-year-old Irish Sport Horse gelding presented for investigation of mild colic signs. Subsequent investigations identified increased cardiac troponin I concentrations, suggestive of myocardial damage, but did not identify an underlying cause. Treatment with magnesium sulfate was unsuccessful but a constant rate infusion of lidocaine hydrochloride resulted in conversion to a normal sinus rhythm. Follow up examination four weeks later did not identify any abnormal complexes at rest or at exercise and the horse successfully returned to previous athletic activity.

Signalment

A 665kg 9-year-old Irish Sport Horse gelding used for dressage.

History

The gelding presented for evaluation of mild colic signs (inappetence and flank watching) and lethargy noted by the owner four hours previously. Examination by the referring veterinarian had identified a resting tachycardia (80bpm), and a left dorsal displacement of the large colon was suspected based on transrectal palpation performed at the yard. The attending veterinarian had administered six liters of isotonic enteral fluids containing magnesium sulfate (1g/kg) via a nasogastric tube and flunixin meglumine (1.1mg/kg IV). The horse was promptly referred, primarily due to concern regarding the high heart rate. The horse had been in the owner's possession for three years, with no history of illness or travel abroad during this time. Vaccinations against equine influenza virus, equine herpesvirus and tetanus were up to date. The horse had been dewormed six months previously with oral moxidectin and praziquantel and a fecal worm egg count performed three months previously had not identified any oocysts. No other horses on the farm were affected and there had been no change in diet or environment over the past four weeks. The horse was stabled during the day and turned out to pasture with five other horses at night. This represented a change in management which had been initiated two weeks previously, although no change of pasture had occurred, and time spent at pasture was unchanged. Very mild loss of condition had been noted by the owner over the past 10 days. The horse was in regular work and was reported to be performing well.

Physical examination

Upon presentation, the gelding was bright, alert and in good condition weighing 665kg (body condition score 6/9).[1] Physical examination identified a heart rate of 48 beats per minute with a regular rhythm, a respiratory rate of 16 breaths per minute with normal respiratory effort, a rectal temperature of 100^{o}F and pink, slightly tacky mucous membranes with a capillary refill time of less than two seconds. Skin turgor was mildly decreased. Thoracic auscultation was unremarkable. Borborygmi were present in all abdominal quadrants and no abdominal distension was noted. Cardiac auscultation identified a regular rhythm, with no detectable murmur. Jugular refill was

bilaterally normal and peripheral pulse quality was good. Digital pulses were normal in all legs.

Initial problem list and differential diagnoses

Problems identified by the referring veterinarian during initial examination were severe tachycardia, mild colic signs and lethargy. Problems identified based on initial physical examination included dehydration, mild tachycardia and weight loss.

Lethargy is a non-specific clinical sign and can be caused by pain, any form of systemic illness or neurological disease. Lethargy in combination with mild colic signs and signs of dehydration is most commonly associated with mild to moderate gastrointestinal disease. The most likely differential diagnoses include non-strangulating lesions such as large intestinal displacements, impactions, inflammation or other non-strangulating mechanical obstruction of the gastrointestinal tract. Non-gastrointestinal causes of colic include liver disease, urogenital disease, musculoskeletal disease, cardiac disease, endocrine disease (pheochromocytoma), and respiratory disease.

Differential diagnoses for severe regular tachycardia in conjunction with colic signs often indicate more severe gastrointestinal tract lesions such as strangulating intestinal lesions. The initial diagnostic procedures were therefore directed at investigating the gastrointestinal tract first. Differential diagnoses for tachycardia with a regular rhythm include sinus tachycardia, defined as an increased rate of discharges originating in the sinoatrial node with normal conduction. This may be caused by excitation or fear, pain, relative hypovolemia, endotoxemia, endogenous or exogenous catecholamines or parasympatholytic drugs. If the degree of pain or cardiovascular compromise is minimal but the increase in heart rate is severe, a tachyarrhythmia or primary heart disease should be considered. Monomorphic ventricular tachycardia is characterized by a regular rhythm when sustained but was considered less likely due to the presenting complaint and heart rate at admission in this case; VT is typically characterized by tachycardia greater than 60bpm. Accelerated idioventricular rhythms may also sound regular on auscultation. They are caused by a subsidiary pacemaker discharging at a rate that is equivalent to or

faster than the sinus pacemaker. These arrythmias are characterized by tachycardia in the range of 60-80bpm.

Weight loss may be caused by inadequate intake due to insufficient provision or dental disease or limited access to food due to competition or musculoskeletal pain. Increased energy demand may be due to increased workload, gestation, seasonal change or hypermetabolism with chronic disease. Malabsorption of nutrients may be caused by inflammatory bowel disease, neoplasia or endoparasites. Based on further discussion with the owner and yard manager, the mild weight loss had coincided with an increase in workload and the change in turnout routine, resulting in reduced forage intake during the day. It was therefore agreed to increase the caloric intake first before further investigations would be pursued.

Initial diagnostics

To further delineate the suspected intestinal disease and to rule out involvement of other organ systems, transrectal palpation, nasogastric intubation, abdominocentesis, abdominal ultrasonography and measurement of packed cell volume, biochemistry and fecal egg count were planned. Transrectal palpation identified dry, firm fecal balls within the rectum and throughout the small colon. The nephrosplenic space was easily palpable and empty and no other abnormalities were appreciated. Nasogastric intubation yielded no reflux and abdominal ultrasonography did not identify any abnormalities. Abdominal paracentesis was attempted but did not yield any fluid. Serum biochemistry identified increased gamma glutamyl transferase (GGT) activity, increased total bilirubin concentration and hypocalcemia (table 2). Packed cell volume, total protein concentration and albumin concentration were normal.

GGT is produced by the biliary epithelium and is increased in cholestasis or biliary injury. It may also be released into plasma with hepatocellular injury. GGT is also produced in small amounts by lung, kidney, pancreas and mammary gland – however, this is released into the secretion (e.g. urine, milk), so it is considered liver specific. Increased activity might occur with acute hepatitis, biliary or cholestatic diseases, pancreatic disease or large colon displacements.[2] Mild to moderate elevations in GGT (up to 140IU/L) have also been documented in racehorses and

performance horses in training, with no specific mechanism identified.[3,4] Hyperbilirubinemia can be explained by anorexia, hemolysis, hepatic disease or cholestasis. In this case, the increased GGT activity was most likely caused by a possible displacement or previous athletic activity and the increased bilirubin concentration was most likely due to anorexia. Hypocalcemia may be caused by reduced intestinal absorption, hypoalbuminemia, sepsis, exercise, cantharidin toxicity, acute renal failure, exertional rhabdomyolysis, hypoparathyroidism, pancreatitis, oxalate toxicity or administration of tetracyclines or furosemide. Reduced intestinal absorption was thought to be the most likely mechanism in this case, given the colic signs observed by the referring veterinarian and absence of evidence of renal or muscle disease. A fecal worm egg count was performed based on the deworming history and reported weight loss; this identified 50 eggs per gram of feces.

A working diagnosis of a large intestinal displacement which had resolved during transportation to the hospital was made. Dehydration was presumed to be secondary to reduced water intake.

Case Management

Initial Management

A 14G polyurethane catheter was placed in the left jugular vein. Dehydration was estimated at 5% based on physical examination findings and laboratory analyses. A compound sodium lactate solution was administered intravenously at an initial bolus of 30ml/kg bodyweight, followed by 4ml/kg/hour to replace the remainder of the deficit. Feed was withheld and the horse was monitored for further colic signs. Parameters were measured initially every two hours for the first eight hours and then at four-hourly intervals. Although the horse was noted to be anxious and stressed in the stable on multiple occasions, no colic signs were observed, and the heart rate remained between 44-48bpm. Twelve hours following admission, the heart rate increased to 92bpm. The horse was visibly anxious but remained alert and responsive, showing no signs of abdominal pain. Cardiac auscultation identified a regular rhythm with an occasional variation in the intensity of heart sounds noted. No pulse deficits were identified, and jugular fill and pulsation remained bilaterally

normal. Mucous membranes were pink and moist, with a capillary refill time of one second and skin turgor was normal. Borborygmi were present in all abdominal quadrants. The horse had not passed any feces since admission.

Refined problem list and differential diagnoses

Problems identified this point were marked tachycardia with variable intensity in heart sounds and reduced fecal output. Likely differential diagnoses for tachycardia at this point included pain and ventricular tachycardia. The heart rate was not consistent with the adequate hydration status and absence of signs of pain, with no evidence of systemic inflammation, making an arrhythmia more likely. Reduced fecal output may be caused by reduced food intake, gastrointestinal ileus or a large intestinal obstruction.

Diagnosis

A base-apex ECG identified sustained, uniform ventricular tachycardia with a heart rate of between 80-100bpm (figure 1). Ventricular tachycardia is defined as greater than three consecutive ventricular premature complexes (VPC) and may be sustained or paroxysmal. Monomorphic, or uniform VT is associated with a regular rhythm and is thought to be caused by a single focus in the myocardium. Polymorphic, or multiform, VT is associated with multiple ventricular foci with the resultant QRS complexes varying in amplitude, axis and duration. Causes of ventricular tachycardia in the horse include structural disease (myocardial fibrosis, neoplasia, aortic root rupture), sepsis, drug administration, autonomic imbalances, electrolyte or metabolic imbalances, anesthesia and myocarditis.

Further investigations included another serum biochemistry to assess electrolyte status, a complete blood count to screen for inflammation, and echocardiography to rule out any structural or functional changes. Ultrasonography can also identify visible myocardial lesions such as scar tissue, myocardial disease, aorto-cardiac fistula or valvular regurgitation with ventricular dilation, which can predispose to the development of VT.

Repeat serum biochemistry did not identify any electrolyte imbalances and the hypocalcemia identified on admission had resolved. Based on the history available,

toxic causes of myocardial damage such as plants (including foxglove, rhododendron, oleander and yew), ionophore antibiotics, heavy metals and cantharidin toxicosis were considered less likely. The horse was fed a well-balanced diet, with access to fresh green grass, making nutritional causes of myocarditis (vitamin E or selenium deficiency or copper toxicity) less likely. A complete blood count identified a normal total nucleated cell count with a mild lymphopenia (table 1). Causes of lymphopenia include stress, early viral infection, glucocorticoid administration, endotoxemia and malnutrition. Lymphopenia typically occurs as a concurrent finding with neutrophilia on an inflammatory leukogram; however neutrophil count and percentage were normal, and no evidence of an infectious or inflammatory process was identified on physical examination. Stress was therefore assumed to be the cause of the mild lymphopenia.

Echocardiography identified very mild regurgitation at the aortic, mitral and tricuspid valves. Mild regurgitation is not considered to be clinically significant. Interventricular septum thickness was mildly increased, and aortic root diameter was mildly reduced (table 3), likely caused by sustained VT and reduced cardiac output.[5] The remainder of the standard cardiac measurements were within published reference ranges (table 3) and no other structural abnormalities were visualized. Fractional shortening was towards the low end of the reference range, likely due to the arrythmia.[5]

Serum cardiac troponin I (CTnI) measurement was also performed to investigate myocardial damage; this identified a raised concentration (0.97 pg/mL). Myocardial injury may be primary or can occur secondary to systemic disease. Infectious myocarditis may be caused by viruses, bacteria or parasites and may involve extension of a pre-existing infection. Viral causes of myocarditis include equine influenza virus (EIV), equine infectious anemia, equine viral arteritis, equine herpesvirus-1 (EHV-1), west nile virus and African horse sickness. Parasitic causes of myocarditis are rare in the horse and include migration of Strongyloides spp. and onchocerciasis. Recognized causes of bacterial myocarditis include Streptococcus spp., Staphylococcus spp., and Mycobacterium spp. Myocardial injury can also be caused by drugs, toxins, trauma, hypoxia, metabolic disease or nutritional deficiencies (vitamin E/selenium).[5]

The heart rate and rhythm were continuously monitored using a telemetric ECG. Sustained monomorphic VT with occasional normal complexes persisted. The heart rate continued to increase to 120bpm and at this point it was decided to commence treatment. Rapid sustained VT may lead to a reduction in cardiac output and tissue perfusion. Ventricular arrhythmias also have a higher potential to deteriorate into an unstable rhythm. Prompt anti-arrhythmic treatment is indicated if polymorphic complexes are present, if the heart rate is excessively high (>100bpm), if signs of cardiovascular compromise are present or if a short coupling interval, including R-on-T phenomenon is noted. Treatment was initiated in this case when the heart rate was sustained over 100bpm, although no signs of cardiovascular compromise or polymorphic complexes were observed.

Treatment

The horse was initially treated with an intravenous infusion of magnesium sulfate (1g/min). Magnesium decreases the influx of calcium ions (Ca^{2+}) into myocytes, reducing action potential duration. It also activates the Na^{+}/K^{+} ATPase in the cell membrane, increasing the threshold required for depolarization. Side effects associated with toxicity include CNS depression, weakness, trembling, bradycardia and hypotension. Very high doses may cause neuromuscular blockade with respiratory depression and cardiac arrest. Magnesium is not considered a pro-arrhythmic drug so was used as first line therapy in this case. This was continued up to a total dose of 25g. During this time, an increase in the frequency of normal complexes was noted (figure 2) but sustained ventricular tachycardia persisted following cessation of the infusion. The gelding was subsequently treated with a single bolus of lidocaine hydrochloride (0.5mg/kg slow IV). This had no effect on the arrhythmia and was repeated a further two times at 5-minute intervals to a total dose of 1.5mg/kg. VT persisted and a continuous rate infusion (CRI) of lidocaine (0.05mg/kg/min) was initiated. Continuous ECG monitoring was performed throughout treatment. The frequency of abnormal complexes gradually decreased and normal sinus rhythm, with a heart rate of 36bpm was restored within six hours of commencing lidocaine therapy (figure 3). No adverse effects were observed. Lidocaine infusion was discontinued after two hours of normal sinus rhythm. Holter ECG monitoring was performed for a further 36 hours, during which time no premature depolarizations were observed. Lidocaine is a type IB sodium channel

blocker, increasing the threshold for depolarization of myocardial cells. Adverse effects are uncommon at therapeutic doses; however, overdose can cause ataxia, CNS excitement, collapse, muscle tremors and arrhythmias.

Given the presence of acute myocarditis with no underlying cause identified, anti-inflammatory treatment with corticosteroids (prednisolone, 1mg/kg PO SID) was initiated 24 hours after conversion to normal sinus rhythm. The horse was discharged from the hospital for four weeks of stall and paddock rest at home. Oral prednisolone was continued at 1mg/kg PO SID for three weeks followed by 0.5mg/kg PO SID. Daily heart rate monitoring was performed by the owner using a stethoscope.

Follow up

The horse was re-admitted to the hospital for repeat resting and exercising ECG after four weeks of rest. The horse was in good body condition, weighing 665kg (body condition score 6/9).[1] The owner reported that the heart rate had remained within normal limits (36-44bpm) with a regular rhythm during the rest period. Cardiac auscultation identified normal sinus rhythm with a resting heart rate of 40bpm. The horse was exercised to a heart rate of 200bpm with no abnormal complexes detected by electrocardiography at rest or during exercise (figure 4 and figure 5). Serum cardiac troponin I was normal (<0.02ng/mL). The horse was discharged with instructions to taper and discontinue oral prednisolone therapy (0.5mg/kg PO q48h for two weeks) and to gradually re-introduce exercise during this time while continuing to monitor resting heart rate and rhythm daily.

Self-Reflection

The tachycardia detected by the referring veterinarian may have reflected paroxysmal ventricular tachycardia, particularly given the mild nature of colic signs observed. Horses with VT commonly present with mild colic signs, lethargy and anxiety with an inappropriately high heart rate, as described in this case. VT can also be secondary to systemic disease such as severe colic and endotoxemia, which appears less likely in this case based on physical examination and clinicopathological analysis. Ventricular rate is typically increased (>60bpm) in VT so it is possible that the VT was not sustained at the point of admission, when mild

tachycardia (48bpm) was noted. Infrequent measurements of heart rate following admission meant that VT may have been undetected for several hours.

Ventricular tachycardia is often indicative of primary myocardial disease but can occur secondary to electrolyte or acid-base imbalances, hypoxia, systemic disease, toxicity or treatment with anti-arrhythmic drugs such as quinidine sulphate. In this case magnesium was not measured due to analyzer limitations out of hours. Hypomagnesemia has been documented in hospitalized horses and is more common in those with gastrointestinal disease.[6] In this case, the horse had received magnesium containing fluids (1g/kg PO) prior to referral, and treatment with intravenous magnesium also failed to resolve the arrhythmia, making hypomagnesemia less likely as an underlying cause. Arterial blood gas analysis could have been performed to rule out acid-base imbalance.

Myocarditis is rare in the horse. It is a focal or diffuse inflammatory reaction of the myocardium with degeneration and/or necrosis of myocytes, followed by an infiltration of inflammatory cells. Myocardial disease was thought to be primary in this case, given the absence of sepsis or severe systemic disease. No history of contact with toxins and no other co-grazing horses were reported to be affected. The horse was fed a well-balanced diet, making nutritional causes unlikely although vitamin E and selenium levels could have been measured to conclusively rule out deficiencies of these. Primary myocarditis is idiopathic in many cases, such as this, highlighting the difficulty in identifying a cause. A retrospective study identified an etiological explanation of VT in 8/21 cases, four of which were anaesthetized for exploratory celiotomy.[7] This horse did not have any specific clinical signs which may have helped narrow a diagnosis. The monomorphic nature of the complexes suggested that a single focus was likely responsible. The gold standard for diagnosing myocarditis in people is endomyocardial biopsy, which has been described in the standing horse.[8] However, this procedure samples a small area of myocardium and may not detect focal pathology. Myocardial biopsy was not performed in this case due to lack of equipment and experience with the procedure.

Elevated CTnI has been reported in horses presenting with colic and was significantly higher in horses that underwent surgery for colic compared to those

managed medically.[9] In this case, colic signs were mild and had resolved upon presentation, making this a less likely cause of elevation. CTnI could have been repeated prior to discharge as it has a short half-life (0.47h in healthy ponies) [10] and the single measured value could have been obtained during an increase. Measurement of CTnT has also been shown to be both sensitive and specific for myocardial injury;[11] however this is less widely available.

More recent publications have recommended magnesium sulphate doses for treatment of VT as high as 100mg/kg bodyweight, equating to a total dose range of

66.5g in this horse.[10] A lower dose of magnesium sulphate (total dose 25g) was administered in this case based on previously published doses[13] and senior clinician recommendation. Although CRI administration of lidocaine to treat arrythmias has been described in horses,[14,15] lidocaine infusion is not included in many standard treatment protocols for VT. In this case, the constant rate infusion may have maintained plasma concentrations necessary to achieve conversion compared to bolus treatment. A higher bolus dose of lidocaine is used in small animal medicine (2mg/kg, up to 8mg/kg total dose).[16] However, there are no published studies documenting the use of this dose range in the horse, and there are significant differences between species with regard to toxic threshold. [17] Levels of 1.5–5.0µg/ml have been associated with successful conversion of VT [18] while levels of 3.24 ± 0.74 µg/ml have been associated with clinical signs of toxicity in horses.[17] Monitoring of plasma lidocaine concentrations could have been performed to ensure adequate levels were reached but was not utilized in this case due to lack of equipment. Other treatment options that would have been considered should the arrhythmia have been refractory to both magnesium and lidocaine, were sotalol, propafenone, and procainamide. Quinidine gluconate, propranolol and amiodarone are also described, however, availability of these is limited in the author's region.

In the absence of any obvious infectious etiology, corticosteroids were used to treat myocardial inflammation in this case. The use of corticosteroids may be considered controversial, particularly if viral infection is suspected. Viral infection was not conclusively ruled out in this case, however physical examination was not consistent with this, and the neutrophil count was within normal reference range. Viral infection was also considered unlikely as the horse had been vaccinated against EIV and

EHV-1 and the remainder of known causes of viral myocarditis are not endemic to the author's region. Ideally, a blood smear would have been performed to examine neutrophil morphology and help to rule out an infectious process. Viral infection could have been further investigated by submitting a nasal swab for EIV and EHV PCR. Measurement of acute phase proteins could also have been performed to assess for inflammation and infection.

Should VPCs have been noted at the time of discharge, treatment with oral sotalol or propranolol could have been considered. For completeness, a further ECG could have been performed once the horse was back in full work and off all medication.[19] The owner continued to monitor heart rate on a daily basis, although this is unsuitable for the detection of isolated premature complexes. A second ECG would have been even more important should the horse be required to perform at maximal intensity e.g. eventing or racing. A single lead (II, RA → LL) was recorded and was sufficient for diagnosis and monitoring response to treatment. A three lead ECG should have ideally been displayed at re-examination, as lead III (LA → LL) produces an alternative QRS complex, which can aid in the detection of ectopic complexes. [20,21]

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Table 1: Hematology Report

	Normal Reference Range	Day 1	Day 2
PCV % (manual)	30-50	44	36
RBC x 10^6/ul	5.50-11.00	7.59	
MCV fl	37-55	42	
MCH pg	13.0-19.0	15.4	
MCHC %	31-39	36.4	
Reticulocytes/ul			
RBC Morphology			
Nucleated Cells/ul			
Nucleated RBC's/ul			
White Blood Cells10^3/mm^3	5 -10	8	
Metamyelocytes/ul			
Band Neutrophils/ul			
Seg. Neutrophils/ul			
Lymphocytes 10^3/mm^3	1.4-4.0	1.2	
Monocytes 10^3/mm^3	0.0 - 0.4	0.10	
Eosinophils 10^3/mm^3	0.09		
Basophils/ul			
Granulocytes 10^3/mm^3	3-8	6.7	
Leukocyte Morphology			
Platelets 10^3/mm^3	100-400	114	

Table 2: Biochemistry Report

Parameter	Units	Normal Reference Ranges	Day 1	Day 2	Day 34
Creatinine	umol/L	53-194	102		
ALP	IU/L				
AST	U/L	175-340	309	317	
LDH	IU/L				
SDH	IU/L				
GLDH	IU/L				
GGT	U/L	5-40	41	41	
CK	U/L	120-470	431	270	
Total bilirubin	umol/L	9-39	54	121	
Direct bilirubin	mg/dl				
Amylase					
Lipase					
Urea	mmol/L	2.5 - 8.9	6.8	4.1	
Glucose	mmol/L	3.6-6.1	5.5	6.1	
Na	mmol/L	126-146	140	140	
K	mmol/L	2.5-5.2	3.2	2.9	
Cl	mEq/L				
Ca	mmol/L	2.88-3.55	2.62	2.90	
P	mg/dl				
Mg	mg/dl				
Osmolality	mOsm/kg				
Total protein (refractometer)	g/L	52-74	52		
Total protein (analyzer)	g/L	57-80	60	59	
Albumin	g/L	22-37	32	32	
Globulin	g/L	27-50	27	28	
Uric acid	mg/dl				
Ammonia	ug/dl				
Bile Acids					
pH					
PO_2	mm Hg				
PCO_2	mm Hg				

HCO_3	mEq/L				
Total CO2	mmol/L	20-33	31	28	
L- Lactate	mmol/L	< 2	1.8	1.7	
Cardiac Troponin I	ng/mL	0.01-0.2	-	0.97	<0.02

Table 3: Cardiac Measurements (Day 2)

Measurement	Day 2	Reference Range (Warmbloods)
PA	5.12cm	4.9-6.1 cm
PAD	5.97cm	5.6-7.7 cm
AoV	6.06cm	6.1-8.0 cm
AoD	7.98cm	7.1-9.0 cm
IVSs	5.54cm	3.6-5.2 cm
IVSd	3.97cm	2.6-4.1 cm
LVIDd	12.65cm	9.9-13.4 cm
LVIDs	8.43cm	5.9-9.2 cm
LVFWD	2.48cm	1.6-3.0 cm
LVFWS	4.55cm	3.0-5.7 cm
LAD	11.71cm	11.6-14.6 cm
AoD:PAD	1.36	1.0-1.3 cm
LAD: Ao	1.47	1.2-1.7 cm
FS	33%	32-49%

Reference: Reed S, Bayly W, Sellon D. Equine Internal Medicine. 4th ed. Missouri: Elsevier; 2018:421-438.

Figure 1: Electrocardiogram displaying sustained Monomorphic Ventricular Tachycardia (gain 10mm/mV, paper speed 25mm/s)

Figure 2: Electrocardiogram during treatment with magnesium sulphate (gain 10mm/mV, paper speed 25mm/s)

Figure 3: Electrocardiogram showing normal sinus rhythm following treatment with lidocaine infusion (gain 10mm/mV, paper speed 25mm/s)

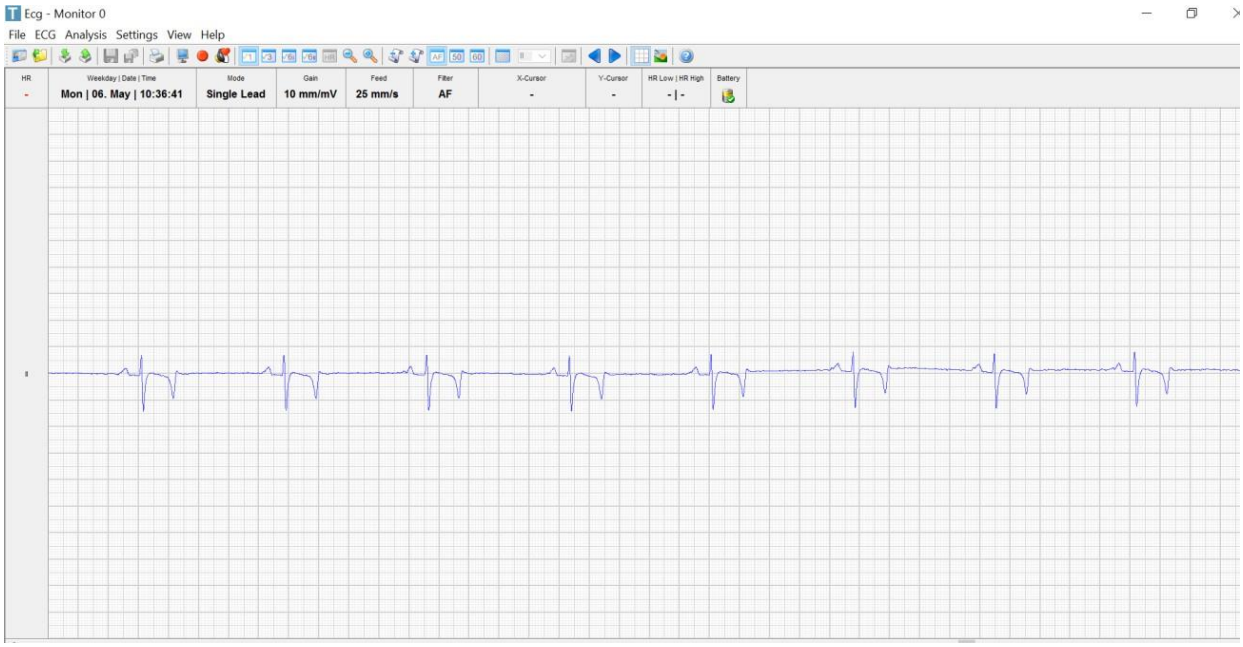

Figure 4: Electrocardiogram showing normal sinus rhythm during canter exercise at re- examination four weeks post-treatment (gain 10mm/mV, paper speed 25mm/s)

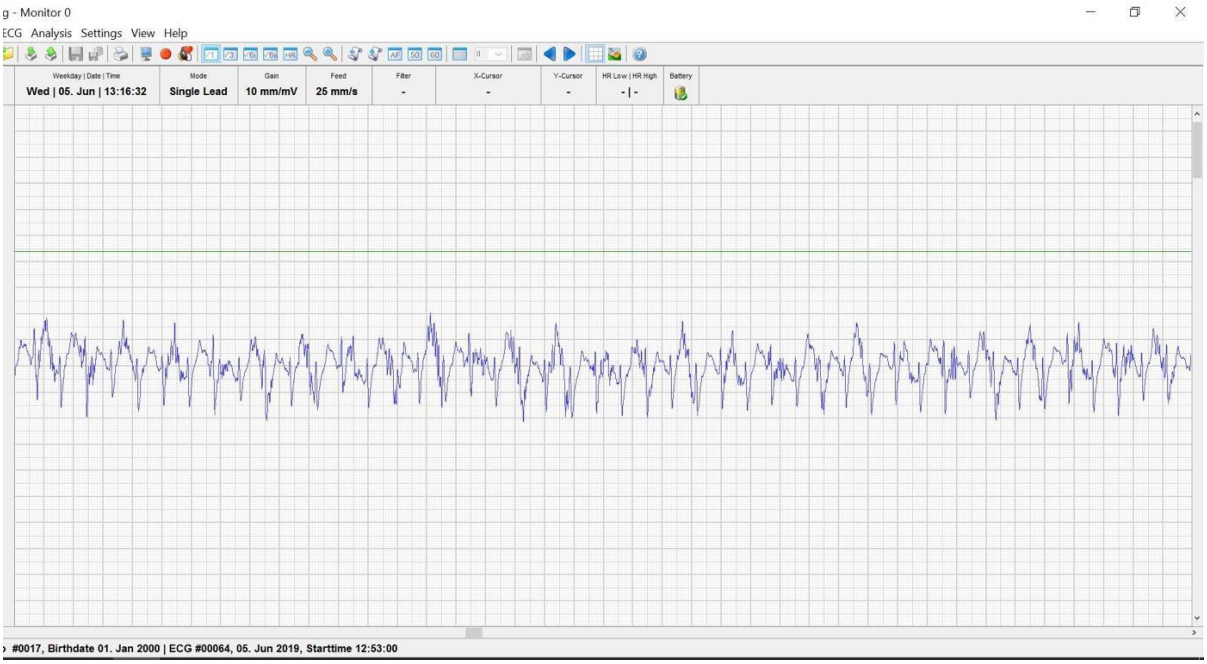

Figure 5: Graph showing heart rate during exercise at re-examination (0.5cm/min)