Chapter 2

Heart Conditions in Felidae

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Additional information is available at the end of the chapter

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Abstract

In this chapter "Heart Conditions in Felidae," we addressed some facts of the cardiovascular system and its disorders. The disorders are: arrhythmogenic right ventricular cardiomyopathy, atrial fibrillation, atrioventricular heart block, cardiac tumors, chronic valve disease (hypertrophic cardiomyopathy), dilated cardiomyopathy, pulmonary hypertension, pulmonic stenosis, subaortic stenosis, and spongy myocardium. Veterinary clinical evaluation of the feline with heart disease is based on many aspects, and also the deviation from the normal standards suggests but does not specify structural heart disease.

Keywords: Felidae, cardiovascular system, cardiac disorders, blood parameters, therapy

1. Introduction

Feline or cats (family Felidae) consist of 37 species of cats. Thirty-six wild cats plus one domestic one are called felid. A member of this family is also called a felid.

1.1. The heart

Heart is one of the vital organs in body as it pumps oxygenated and nutrient-rich blood to different parts of the body. Heart diseases widely affect blood circulation, causing a series of problems. There are two main types of heart diseases: one affecting the heart valves and other heart muscle (**Figure 1**).

Cats with any may be efficaciously managed through nutrition, exercise, nutrition and medication. With the right food and veterinarian advice, a cat can still enjoy a happy and active life.

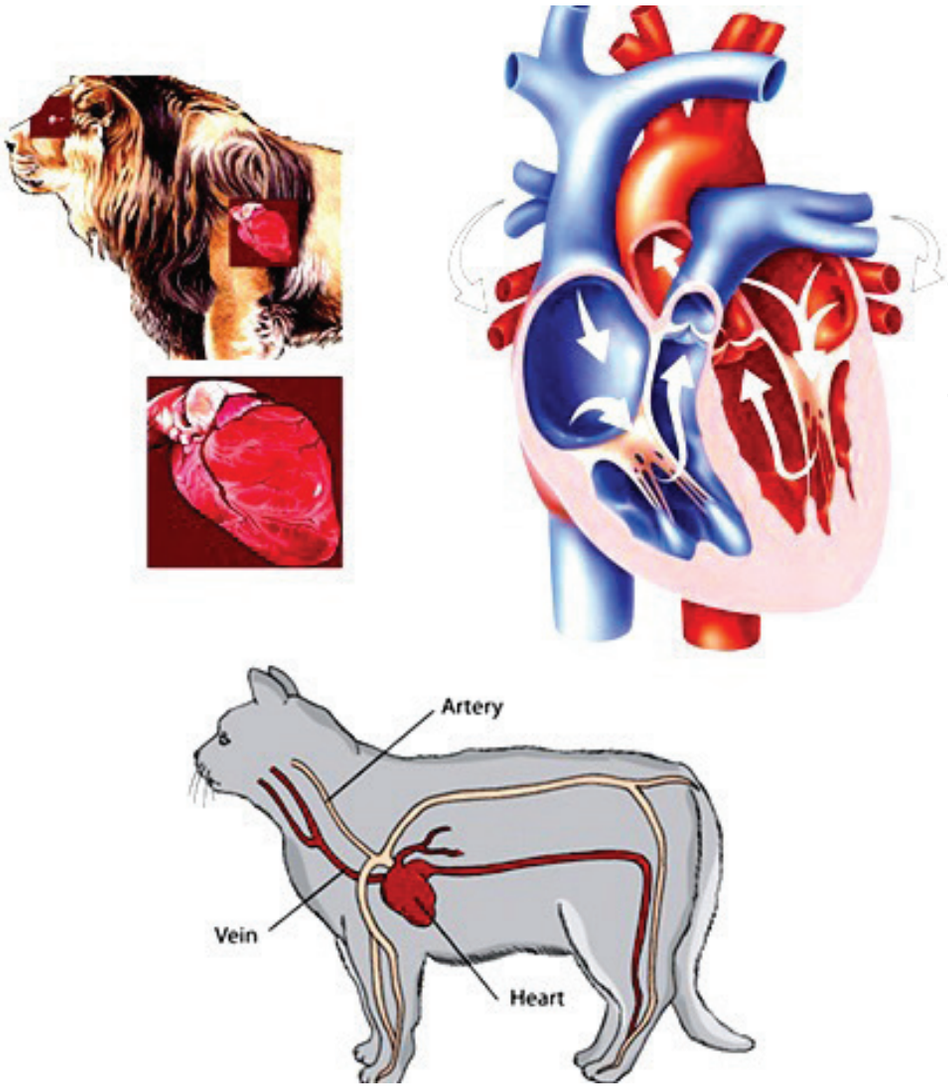

Figure 1. Heart of big and small cats.

1.2. Heart rate

The heart rate ranged between 42 and 76 beats per minute (pulse per minute). Heart charge in large felids is about 40–50 beats consistent with minute. An algometric scaling for critical signs assessment predicts coronary heart price of about 60–80 bpm for mammals weighing 100–250 kg [calculated from the equation: 241 (weight in kilograms—0.25)] [1].

Heart rate of tigers ranged from 56 to 97 bpm. In both types, the most common rhythm was detecting the normal sinus rhythm followed by arrhythmias of the sinus (**Figure 2**). The pacemaker was also observed to wander with normal sinus rhythm or sinus arrhythmias [1, 2].

Veterinary clinical evaluation of the patient with heart disease, especially heart muscle and the deviation from the normal standards, suggests but does not specify structural heart disease. Because deflections and periods of the recordings often are changed by either satisfactory or physiological factors; ECG is a useful tool to diagnose most cardiac arrhythmias and can provide information on the status of the heart muscle. It can also be used as an indicator

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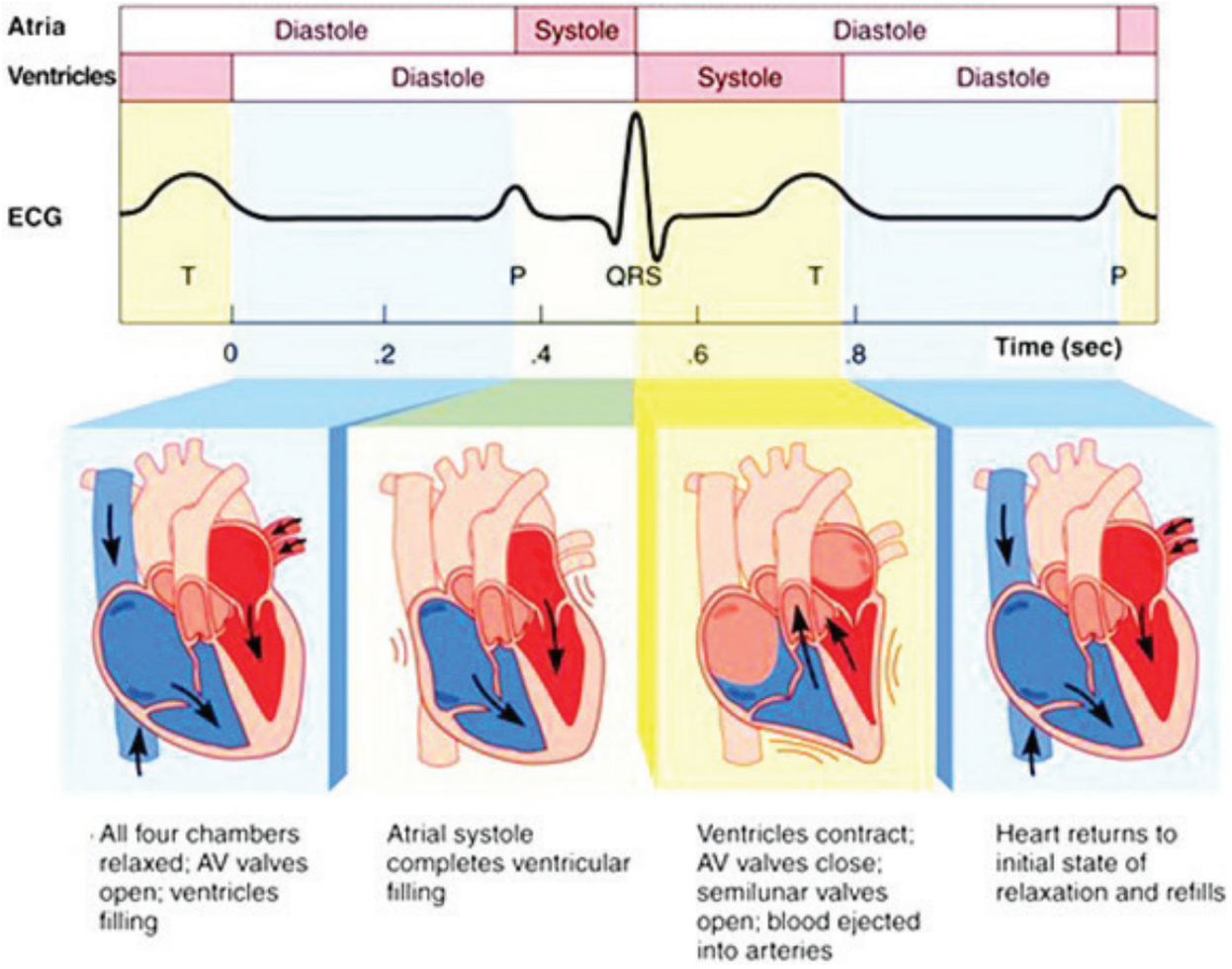

Figure 2. Cardiac cycle. *Heart Rate:* Heart rate of cat ranged from 120 to 140 beats per minute (bpm); heart rate of tigers ranged from 56 to 97 beats per minute (bpm); and heart rate in lion is about 40 to 50 beats per minute. An algometric scaling for vital signs assessment predicts heart rate of approximately 60 to 80 beats per minute (bpm) for mammals weighting 100 to 250 kg (calculated from the equation: 241 [weight in kilograms − 0 25]).

of heart that expands room and electrolyte imbalances. Currently, there is a loss of statistics on the anticipated rhythm and other parameters of electrocardiographic wild felids.

The only source for comparison is data that have been collected from domestic carnivores. Description of every tune recorded at some stage in this study is as common for puppies and cats. It can be attributed to higher values for wave durations found on the largest coronary heart muscles of large felids. A few articles describe invasive oscilloscope and Doppler ultrasound gadgets to measure arterial blood pressure of unusual felids in studies of the usage of diverse anesthetic dealers.

Electrocardiograph (ECG) recordings follow standard procedures as recommended for domestic carnivores. The animals were positioned on the right side lying down, where attached electrodes to the skin via the crocodile moistened clips alcohol in uniform places: the proper arm (RA) and left arm (la) subsequent to the involvement of the caudal aspect of the foreleg appropriate, the proper leg (RL) and left leg (LL) at the patellar ligament on the front facet of the right hind leg (**Figure 3**) [1–3].

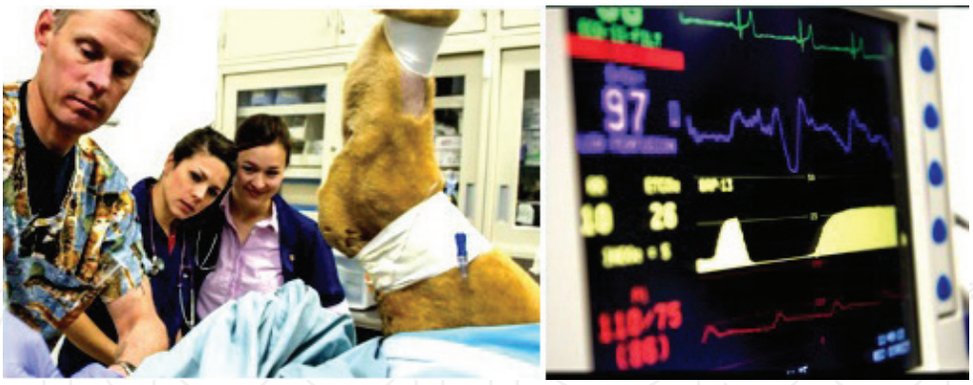

Figure 3. ECG for a lion under anesthesia.

Reports on the use of ECG in wild felines were found intermittently and are usually relevant to assess anesthetic agents. The recorded electrocardiographic parameters of three aware tigers (*Panthera tigris*) have been blanketed rhythms recorded regular rhythm sinus (48.1%), ordinary sinus rhythm with a pacemaker wandering (7.5%), abnormal heart sinus rhythm (18.5%), and cardiac arrhythmia sinus rhythm with a wandering pacemaker (26%).

If the electrical axis between +60 and +90° in 96.3% and in +90° in 3.7% of examinations [1, 2]. In some studies, tigers supplied suggest heart charge of 81 bpm; 38.4% of the animals had ordinary sinus rhythm, 15.4% had normal sinus rhythm with wandering pacemaker, 30.8% had sinus arrhythmia, and 15.4% had sinus arrhythmia with wandering tempo maker. The electrical axis ranged from +60 to +90° in 77.3% of the animals, at +90° in 15.4%, and from +90 to + 120° in 7.3%. Duration of P wave and QRS complex tended to be greater in lions than in tigers and other parameters were similar for each species. This is another wrong fallacy and false notion against the ligers, and ligers do not now have any such problems touching on a weaker coronary heart.

A heart of a liger is as normal as that of the lion and tiger, and there has been never a sign or symptom, which indicates that ligers have a weaker heart. Furthermore, in a study, on ligers where different deaths were analyzed, none of the ligers had died of heart failure. Therefore, it is far entirely incorrect to finish about the ligers that they have got very vulnerable weak spot.

The reason for the relatively low amplitude of the QRS waves, in both species, is to increase the distance between the hearts and re-ropes electrodes. This is due to the fact that the size of the chest and the thickness of the chest wall of the large felids are larger than those of the local carnivores.

2. Hematological parameters

Hematological parameters include hemoglobin (Hgb), hematocrit (Hct), red blood cells (RBC), mean corpuscular volume (MCV), mean corpuscular [8] and hematologic (**Table 1a** and **Figure 4**), and serum chemistry (**Table 1b**) reference intervals for free-ranging lions (*Panthera leo*) [4–8], and other species (**Tables 2–8b**).

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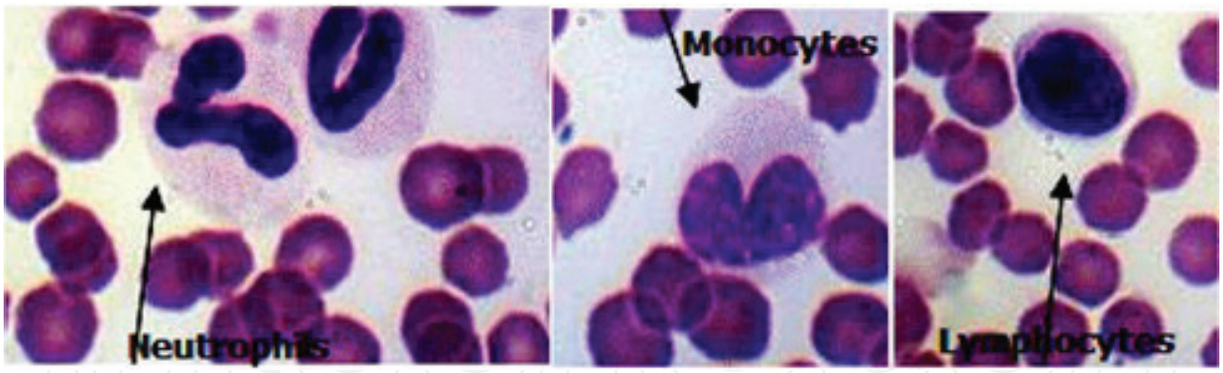

Figure 4. Blood smear of tiger *(Panthera tigris)* stained by Wright stain, 1000×.

Parameter	Range
Hgb (g\dl)	8.9–14.6
Hct (%)	26.8–44.1
RBC($\times 10^6$ cells\μl)	5.1–8.3
MCH (pg\cell):	
Male	14.8–18.5
Female	15.5–19.1
MCHC (g\dl):	
Male	29.6–35.5
Female	30.4–35.7
WBC($\times 10^3$ Cells\μl)	7.2–25.6

Table 1a. Means for the hematologic parameters of captive Siberian tigers (*Panthera tigris altaica*) [6–12].

Parameter	Range
Cholesterol (mmol\l):	1.8–5.0
Male	26.8–44.1
Female	0.9–6.0
Total proteins (g\l)	66.7–104.2
Albumin (g\l)	20.0–30.0
Globulin (g\l):	
Male	33.9–76.5
Female	40.9–75.1

Table 1b. Means for the serum parameters of captive Siberian tigers (*Panthera tigris altaica*) [6–12].

Parameter	Range
Leukocytes (×10³/ml)	5.0–12.2
Neutrophils (×10³/ml)	70.0–88.0
Lymphocytes (×10³/ml)	10.0–30.0
Monocytes (×10³/ml)	0.0–5.0
Eosinophils (×10³/ml)	0.0–2.0
Bands (×10³/ml)	0.0–2.0
Erythrocytes (×10⁶/ml)	4.3–7.8
Hemoglobin (g/dl)	8.4–14.0
Packed cell volume (%)	26.5–46.0
Mean corpuscular volume (fl)	56.0–62.0
Mean corpuscular hemoglobir concentration (g/dl)	29.0–31.8
Platelet estimate (×10³/ml)	336.0–502.0
Albumin (g/dl)	3.3–3.9
Alkaline phosphatase (IU/l)	14.0–57.0
Bilirubin (mg/dl)	0.1–0.2
Blood urea nitrogen (mg/dl)	28.0–46.0
Calcium (mg/dl)	8.8–10.1
Cholesterol (mg/dl)	92.0–122.0
Chloride (mEq/l)	19.0–126.0
Creatinine (mg/dl)	1.5–3.3
Gamma-glutamyl trans peptidase (IU/l)	0.0–1.0
Glucose (mg/dl)	79.0–136.0
Potassium (mEq/l)	3.8–4.9
Sodium (mEq/l)	55.0–158.0
Phosphorus (mg/dl)	3.9–6.5
Protein (g/dl)	6.4–7.6
Aspartate amino-transferase (IU/l)	20.0–32.0
Alanine amino-transferase (IU/l)	23.0–46.0
Thyroxine (mcg/dl)	1.8–2.9

Table 2. Hematologic and serum biochemistry values of captive lynx.

Blood constituents (cells, plasma, and chemical composition) can be used to display the fitness reputation and diagnose diseases, dietary deficiencies and the reproductive popularity (i.e., pregnancy) of animals. Techniques to perform complete blood evaluation are to be had for humans and maximum farm animals' species. The same processes can be used for wildlife species to perceive deviations from normal values in positive blood parameters (cytological or biochemical).

Parameter	Mean
PCV (%)	41.5 ± 5.1
Hb (g/dl)	12.1 ± 1.1
RBC ($\times 10^{12}$/l)	6.9 ± 1.0
MCV (fl)	63.1 ± 7.0
MCHC (g/dl)	30.5 ± 3.6
WBC ($\times 10^{9}$/l)	7.07 ± 1.76
Bands ($\times 10^{9}$/l)	0.18 ± 0.12
Segmented neutrophils ($\times 10^{9}$/l)	4.44 ± 1.44
Eosinophils ($\times 10^{9}$/l)	0.41 ± 0.23
Basophils ($\times 10^{9}$/l)	0.01 ± 0.01
Lymphocytes ($\times 10^{9}$/l)	1.53 ± 0.48
Monocytes ($\times 10^{9}$/l)	0.32 ± 0.18
Bands (%)	2.0 ± 1.5
Segmented neutrophils (%)	64.8 ± 8.8
Eosinophils (%)	6.0 ± 4.0
Basophils (%)	0.2 ± 0.2
Lymphocytes (%)	22.2 ± 5.3
Monocytes (%)	4.6 ± 3.4
Reticulocytes (%)	0.67 ± 0.48
Plasma protein (g/dl)	8.3 ± 1.2

Table 3. Hematologic values of fishing cat (*Felis viverrina*).

Hematology	Unit	Range
Red blood corpuscles	(TEC) × 106/μl	4.66–9.15
Total leukocytes count	(TLC) × 103/μl	6.2–11.05
Hemoglobin	(Hb) g/dl	7.8–13.8
Hematocrit	(PCV) Ratio	36–45
Erythrocyte sedimentation rate	(ESR) Hours	14–26
Icterus index	(II) u/l	2–5
Differential leukocyte count	%	
Neutrophils		57–75
Lymphocytes		18–35
Monocytes		2–6
Eosinophils		2–6

Hematology	Unit	Range
Basophils		0–4
Blood plasma biochemistry		
Albumin (ALB)	g/dl	2.1–4.6
Total protein (TPROT)	g/dl	3.7–8.7
Total bilirubin TBIL)	mg/dl	0.4–3.2
Creatinine (CRE)	mg/dl	1.6–4.6
Blood urea nitrogen (BUN)	mg/dl	6.5–48.2
Alanine aminotransferase (ALT)	IU/l	21.2–109.0
Aspartate aminotransferase (AST)	IU/l	14.4–84.0

Table 4. Hematological and biochemical values of Bengal tigers *(Panthera tigris tigris)*.

Parameter	Unit	Range
PCV	%	30–45
Hgb	g/dl	9.8–15.4
RBCs	$\times 10^6/\mu l$	5.0–10.0
Reticulocytes	%	0–0.6
Absolute reticulocyte count	$\times 10^3/\mu l$	<60
MCV	fl	39–55
MCH	pg	13–17
MCHC	g/dl	30–36
Platelets	$\times 10^3/\mu l$	300–800
MPV	fl	12–18
WBCs	$\times 10^3/\mu l$	5.5–19.5
Neutrophils	%	45–64
Cholesterol	mg/dl	71–156
Creatinine	mg/dl	0.9–2.2
Glucose	mg/dl	60–120
Magnesium	mg/dl	1.7–2.6
Phosphorus	mg/dl	3.0–6.1
Potassium	mEq/l	3.7–6.1
Protein	g/dl	6.0–7.9
Albumin	g/dl	2.8–3.9
Globulin	g/dl	2.6–5.1

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Parameter	Unit	Range
Sodium	mEq/l	146–156
Urea nitrogen	mg/dl	19–34
ALT	U/l	25–97
Amylase	U/l	550–1458
Alk phos	U/l	0–45
AST	U/l	7–38
CK	U/l	69–214
GGT	U/l	
LDH	U/l	58–120
SDH	U/l	
Bicarbonate	mEq/l	17–24
Bilirubin	mg/dl	0–0.1
Calcium	mg/dl	8.7–11.7
Chloride	mEq/l	115–130

Table 5. Hematologic and serum chemistry values of domestic cat (*Felis catus*).

Hematological parameter	Mean	
Erythrocytes ($\times 10^6$/μl)	8.97	
Hematocrit (%)	42.38	
Hemoglobin (g/dl)	14.11	
MCV(fl)	47.70	
MCH (pg)	15.84	
MCHC (%)	33.33	
Leukocytes ($\times 10^3$/μl)	9.37	
Differential cell count (cells/μl)		
Band neutrophils	0	
Mature neutrophils	7.748	
Eosinophils	372	
Basophils	4	
Lymphocytes	849	
Monocytes	365	
Plasma fibrinogen (g/dl)	0.23	
Serological parameters	***Range***	
Sodium (mmol/l)	133.0	161.0

Hematological parameter	Mean	
Chlorine (mmol/l)	108.8	133.3
Total Bilirubin (μmol/l)	2.0	8.0
Total carbon dioxide (mmol/l)	7.7	18.6
Gamma-glutamyl transferase (IU/l)	0.0	4.0
Uric acid (mmol/l)	0.0	0.1
Albumin (g/l)	9.0	14.0
Total Direct Bilirubin (μmol/l)	0.0	2.0
Aspartate transaminase (IU/l)	12.0	40.0

Table 6. Hematologic and serum biochemistry parameters of captive lions (*Panthera leo*).

Parameter	Units	Mean
Red blood cells (RBC)	$\times 10^6/\mu l$	7.635
Hemoglobin (Hb)	g/dl	12.21
Packed cell volume (PCV)	%	36.37
Mean cell volume (MCV)	fl	47.29
Mean cell hemoglobin (MCH)	pg	16.07
Mean cell hemoglobin conc. (MCHC)	g/dl	34.08
Red cell distribution width (RDW)	fl	22.12
Reticulocytes	% RBC's	1.04
Nucleated RBC's (NUC)	/100 RBC's	1.5
White blood cells (WBC)	$\times 10^3/\mu l$	12.19
Segmented neutrophils	$\times 10^3/\mu l$	8.0
Segmented neutrophils	% WBC's	64.3
Lymphocytes	$\times 10^3/\mu l$	3.4
Lymphocytes	% WBC's	28.8
Monocytes	$\times 10^3/\mu l$	0.39
Monocytes	% WBC's	3.2
Basophils	$\times 10^3/\mu l$	0.10
Basophils	% WBC's	0.89
Eosinophils	$\times 10^3/\mu l$	0.42
Eosinophils	% WBC's	3.4
Platelets	$\times 10^3/\mu l$	402.6

Table 7a. Hematologic parameters of jaguar.

Parameter	Units	Mean
Albumin	g/dl	3.70
Alanine aminotransferase (ALT/SGPT)	U/I	60.2
Alkaline phosphatase (ALP)	U/I	35.4
Aspartate aminotransferase (AST/SGOT)	U/I	73.4
Calcium (Ca)	mg/dl	9.92
Carbon dioxide (CO_2)	mEq/l	14.33
Cholesterol	mg/dl	147.9
Chloride (Cl)	mEq/1	115.5
Creatine phosphokinase (CPK)	U/I	515.6
Creatinine (Creat)	mg/dl	1.84
Gamma glutamine transferase (GGT)	U/I	1.6
Glucose (Gluc)	mg/dl	154.4
Inorganic phosphorus (IPhos)	mg/dl	5.77
Iron (Fe)	μg/dl	65.1
Lactate dehydrogenase (LDH)	U/I	269.7
Potassium (K)	mEq/l	4.60
Sodium (Na)	mEq/l	152.6
Total bilirubin (Tbili)	mg/dl	0.26
Total protein (TP)	g/dl	7.35
Triglycerides (Trig)	mg/dl	54.9
Urea nitrogen (UN)	mg/dl	37.7
Uric acid	mg/dl	0.55

Table 7b. Serum and biochemistry parameters of jaguar.

Parameter	Unit	Range
Hematology		
Hemoglobin	g/dl	3.6–14.8
Hematocrit	%	12.9–47.7
Red blood cells	10^6/μl	2.72–10.6
Mean cell volume	fl	43.0–54.0
Mean cell hemoglobin	pg	13.2–17.0
Mean cell hemoglobin conc.	g/dl	27.9–34.4

Table 8a. Hematologic parameters in *Puma concolor couguar*.

Parameter	Unit	Range
Serum chemistry		
Glucose	mg/dl	36.0–258.0
Urea nitrogen	mg/dl	17.0–78.0
Creatinine	mg/dl	0.5–3.4
Total protein	g/dl	5.7–8.7
Albumin	g/dl	1.1–4.1
Total bilirubin	mg/dl	0.1–0.3
Alkaline phosphatase	U/I	1.5–24.0
Alanine aminotransferase	U/I	19.0–215.0
Aspartate aminotransferase	U/I	27.0–465.0
Cholesterol	mg/dl	51.0–217.0
Calcium	mg/dl	7.8–10.9
Phosphorus	mg/dl	2.1–10.9
Sodium	mEq/l	145.0–168.0
Potassium	mEq/l	3.6–7.2
Chloride	mEq/l	96.0–124.0
Globulin	g/dl	2.7–6.1
Lipase	U/l	0.0–93.0
Amylase	U/l	87.0–612.0
Triglycerides	mg/dl	4.0–283.0
Creatine phosphokinase	U/I	133.0–1332.0
γ	Glutamyl transpeptidase GGT	U/I
Magnesium	mEq/l	1.4–9.8
Osmolality	mOsm/l	292.0–365.0

Table 8b. Serum and biochemistry in *Puma concolor couguar*.

Values of blood parameters in large cats (**Tables 1a** and **1b**) can be used by veterinarians or researchers to assess animal health or population. The blood parameter values may vary between free ranging and captive species [4, 5–12].

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3. Cardiac diseases in Felidae

3.1. Arrhythmogenic right ventricular cardiomyopathy

The cause of this disease is unknown and results from a gradual atrophy of the right ventricular myocardium replaced with fibrous and/or fatty, mirroring the disease counterpart in humans [12, 13].

Arrhythmogenic right ventricular cardiomyopathy has mentioned family inclinations for ARVC in massive cats, but it wishes in addition research.

It detected apoptosis in a high percentage of cat's hearts ARVC, with mean thoughts similar to those reported in patients with human ARVC index. This conclusion is supported by the hypothesis that causes the pathogenesis of ARVC, may depend in part on inflammation as well as apoptosis. Coexistence between the cells and the inflammation of the heart muscle in cats with ARVC indicates that these mechanisms contribute to muscle injury and repair in felines likely indicate that the heart muscle inflammation may represent a starting point for apoptosis. In most cats, death is a common sequel by the time clinical signs become apparent due to congestive heart failure right side gradually. And it is often misdiagnosed as cats ARVC tricuspid dysplasia of honor, but the latter always happens disease in cats as young congenital abnormality, which is not true deep cardiac hypertrophy treatment. There is no justification for the mechanical removal of pleural effusion by thoracentesis or closed thoracostomy tube when hoarseness exists. Stabilization of cat cases includes a scientific technique to use an angiotensin-converting enzyme inhibitors, diuretics, and digoxin, with variable achievement.

Antioxidants such as furosemide (2–4 mg/kg/day), spironolactone (5–10 mg/Eg fortekor 2.5 mg/cat daily), and digoxin (1/4 0.125 mg oral tablet P 24 hours).

3.2. Atrial fibrillation

Cat's heart consists of four chambers [13]. Higher chambers are called atria (one: the atrium), and the lower chambers are called ventricles.

Valves are present between each pair of atria and ventricles, each on the left and right hand sides. The coronary valve is a triplex valve found between the left atrium and the left ventricle. Constant rhythmic pattern is a result of an exceptional synchronization and harmony found between numerous atrial and ventral structures. Loss of this synchronization results in several disorders like atrial flutter and atrial fibrillation. Atrial fibrillation is a condition that delays electrical conduction from the atria to the ventricles, or for a prolonged period on ECG, this suggests that the PR period is prolonged time between the principle electric impulses, referred to as the P wave, the QRS complex, which is identified because the coronary heart beats.

Atrial fibrillation (arrhythmia) often arises from atrial flutter. Atrial flutter is characterized by a premature electrical impulse originates in the atria, causing a rapid heartbeat with either regular or irregular frequency.

Arrhythmia or atrial fibrillation results in irregular rhythms of the ventricle as well. On the electrocardiogram, which measures the electrical activity of the heart, a clear pattern can be discerned in atrial fibrillation and atrial flutter.

Atrial fibrillation is classified by:

- Primary atrial fibrillation
- No underlying heart disease involved—caused has not been identified
- Secondary atrial fibrillation
- Severe underlying heart disease such as CHF is involved
- Atrial fibrillation
- Frequent cyclic attacks persist for a short period of time (less than seven days) as the heart returns to normal rhythm on its own
- Continuous atrial fibrillation
- Irregular heartbeat continues (arhythmia) for more than 48 hours, and only responds to treatment
- Permanent atrial fibrillation
- Irregular, nontreatable arrhythmia symptoms are generally associated with underlying disease such as congestive heart failure (CHF).

The symptoms related to atrial fibrillation are (**Figure 5**):

- Heart stimulation(galloping heart)
- Vulnerability
- Coughing
- Shortness of breath (difficulty breathing)
- Tachypnea (rapid breathing rate)

Figure 5. Symptoms of cardiac affections. Weakness, fainting, and syncope/loss of consciousness.

- Idle (lethargy)
- Chronic heart disease which involves valves
- Syncope/loss of consciousness (rare)
- Myocardial infarction (heart muscle disease)
- Expanding the heart(enlargement)
- Congenital heart disease
- Tumors digoxin (drugs commonly used to treat various heart disease (toxicity). As a sequel congestive heart failure, cause may remain unknown.

3.2.1. Diagnosis

- Taking a detailed history of the health of the cat, and the onset of symptoms and potential accidents.
- Veterinarian conducts a full physical examination.
- Lab tests will include full blood tests, a glimpse of biochemistry, and urinalysis.
- It is possible that the results of these tests may not reveal a lot of information related to this disease, but it may be useful to get a comprehensive picture of the health of the cat and the detection of other diseases.
- Additional diagnostic tools echocardiography, X-ray imaging, and color Doppler. The treatment will be directed toward normalizing heart rhythm and get a sinusoidal atrioventricular node back into sync with the atrioventricular node (AV) node. If fibrillation is a chronic problem, the success rate drops accordingly. ECT can be used to normalize the rhythm in some cases. If coronary heart disease at the back of, including the Swiss franc gift, it will also be directed closer to the treatment of treatment, at the side of the success of stability in a heartbeat.

3.3. Atrioventricular heart block

Usually, the reason is the contraction of the heart through electrical impulses that arise from the node, the sino-atrial, and stimulate the atria, and travel to the atrioventricular node, and finally to the ventricles [12, 13]. This electrical conduction system is responsible for controlling heart rate, generating electrical pulses (waves), which was published through the heart muscles, and stimulates the heart muscles to contract and push blood through the interior of the arteries in and out of the body (**Figure 6**).

3.3.1. First-degree atrioventricular block

First degree AV block is common in young felines and healthy cats as a result of high vagal tone (impulses generated by the vagus nerve causing inhibition of heart beat) or accompanied

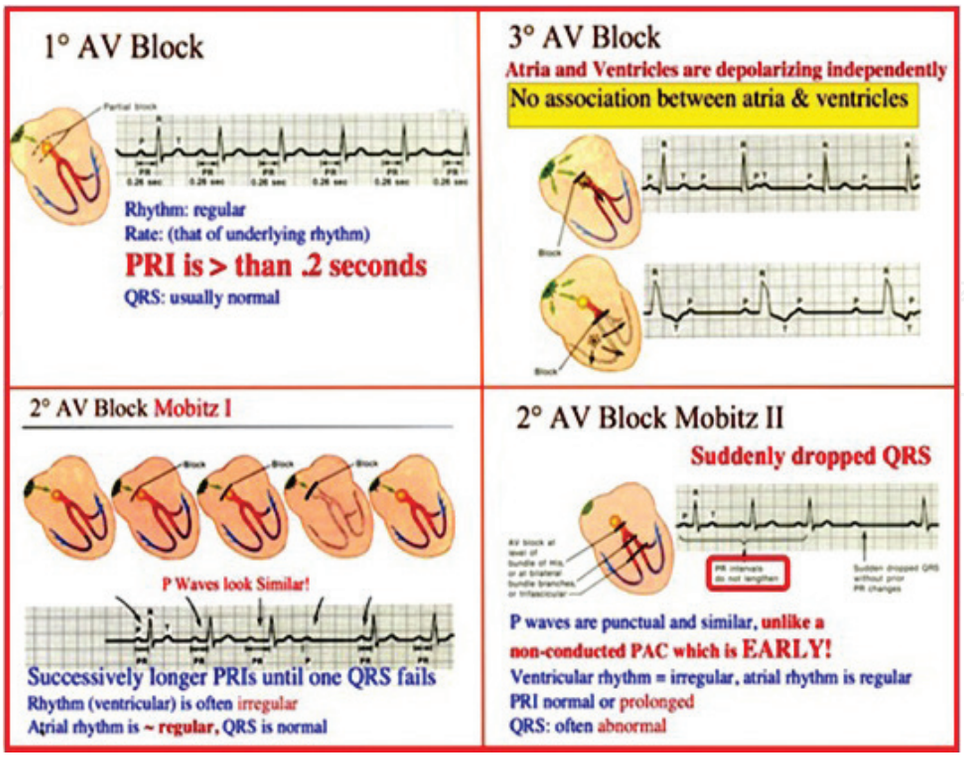

Figure 6. Degrees of atrioventricular heart block in Felidae.

with degenerative conduction system disorder, which by order activates the AV node, conducting regular electrical impulses from the atria to the ventricles with mechanical activity to force blood constantly into the ventricles just before the ventricles are activated to contract and push blood to the body using the pulmonary artery and the aorta?

Affected cases may present variable symptoms

- Congestive heart failure manifestations conjoined with hypertrophic cardiomyopathy
- Shortcoming and overall weakness
- Healthy cats are also prone
- Disorders indirectly related to the heart
- Excessive production of thyroid hormone (hyperthyroidism)
- Cardiomyopathy
- Heart neoplasia
- Drug interference with the function of the AV node

3.3.1.1. Treatment

Treatment will vary depending on the underlying disease causing the second-degree Mobitz Type 1 atrioventricular block. A pacemaker may be necessary.

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3.3.2. Second-degree AV block

A disease in which the previously stated electrical conduction system goes off track, where part of impulses is conducted down from the atria to the ventricles, causing failure of the heart muscle to contract and pump blood efficiently.

3.3.2.1. Symptoms and types

- Weakness
- Idle
- Sudden collapse
- Fainting (fainting) causes involving noncardiac diseases
- Age-related degenerative changes within the cardiac delivery system side effects of drugs (e.g., digoxin, a drug used to treat many heart diseases)
- Heart tumors
- Heart-related infections (e.g., bacterial, viral, parasitic)
- Myocardial infarction (myocardial infarction)
- Shock or trauma

3.3.3. Third degrees of atrioventricular heart block

3.3.3.1. Diagnosis

The precise date for the health of the cat is important, and the onset of symptoms, and possible incidents that preceded this condition. After a full physical examination, the cat arterial blood pressure measurement makes sure that high blood pressure (hypertension) is associated with heart disease. The laboratory test includes standard full blood count, personal biochemistry and urine analysis. These tests are important in diagnosing this problem as there are some biochemical changes that can create a cat to prevent AV block, for example, the presence of an infectious disease or parasitism tests. The culture/sensitivity of the blood test guide will be shown for the type of organisms involved in the infection and the sensitivity of various antibiotics. Other diagnostic tools which are important for comparing structural and useful cardiac parameters are encompass ECG (EKG) and echocardiography for measuring electrical pulses.

3.3.3.2. Treatment

Do not treat the disease aggressively in cats. If it is to maintain the heart rate at the level at which the heart can pump enough blood to the body's natural functions, and generally will be required to treatment. If the primary disease is responsible for AV block, and the veterinarian to deal with them accordingly.

3.4. Cardiac tumors

Body aorta and carotid tumors, recognized as chemodectomas, mostly are benign tumors (**Figure 7**) that arise from chemical receptor tissues in the body. Oxygen content and PH levels in the blood are highly labile to body's chemical changes. Chemical receptors are present over a wide range in the body; however, chemical receptors in the aorta and carotid artery systems are the main ones affected by chemodectomas.

Chemodectomas are rare in cats, but when they occur, older cats tend to be more predisposed. However, it does not seem to be sex or breed a mile of chemodectomas. Given that this is a rare condition in cats, and aortic tumors are more common than carotid tumors, but a malignant tumor in the other organs seem to be more common in cats when it does not happen.

Symptoms and types associated with aortic body tumors include:

1. Coughing
2. Trouble breathing
3. Symptoms of right-sided congestive heart failure
4. Weakness, lethargy carotid body tumors, near the bifurcation point on the carotid artery, where arteries of internal and external carotid originate. The main function of these arteries is to transport oxygenated blood to upper organs like head and neck. Owing to complicity of this area and the function of theses arteries, carotid tumors are often inoperable.

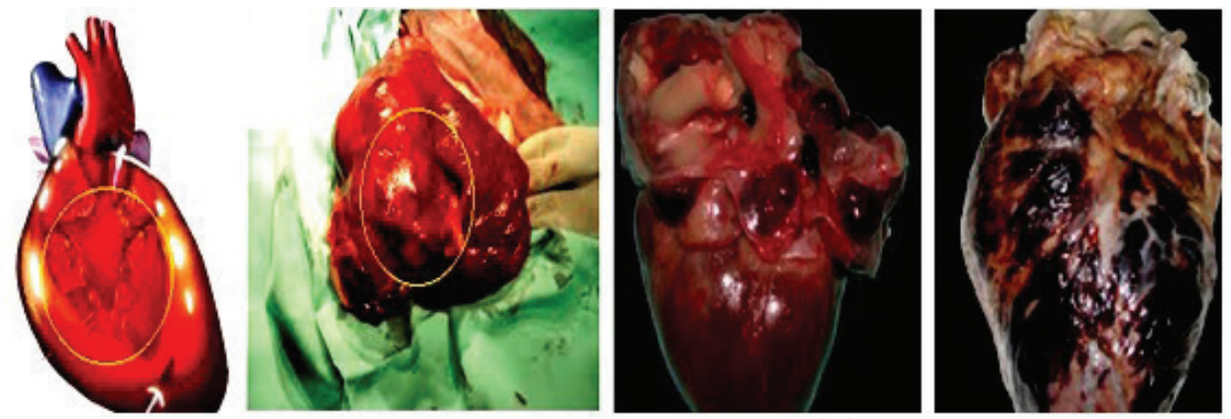

Figure 7. Aortic and carotid body tumors, classified as chemodectomas, are generally benign tumors that grow from the chemoreceptor tissue of the body.

In maximum cases, these tumors nonetheless gradually grow, but benign, as is the case with aortic tumors, they end up a health hassle after they invade the spaces of neighboring blood vessels and lymph vessels. In an expected 30% of the instances, a malignant tumor within the surrounding organs, along with lungs, bronchia or lymph nodes, or more in the liver or pancreas, may occur:

1. Gastroesophageal reflux
2. Vomiting
3. Eating disorders (anorexia)
4. Neck lumps
5. Severe hemorrhaging due to presence of tumors in blood vessels (sudden death may occur)
6. Up to 50% of the cases show metastasis to local blood vessels.
7. Up to 20% of the cases show organ failure due to cancerous growths.

3.5. Chronic valve disease hypertrophic cardiomyopathy (HCM)

It is a disease that is maximized (enlarged) part of the heart muscle (myocardium) without any apparent reason and the creation of a functional disorder of the heart [12, 13]. This is the main cause of sudden death. The prevalence of hypertensive cardiomyopathy is the main cause of sudden cardiac demise in any age organization and the reason of heart attack signs. HCM is often asymptomatic until sudden death. This is frequently one point missense mutations inside the genes of beta-myosin heavy chain (MHC), the myosin-binding protein C, troponinT coronary heart, or tropomyosin. These mutations reason muscle and muscle structural malformations and ability defects in power generation.

Myocardial infarction is a disease that affects my heart muscle. With them, myosites (heart-limiting cells) increase the heart's volume, leading to cardiomyopathy. In addition, the natural alignment of muscle cells is disrupted, a phenomenon known as myocardial disarray and dysfunction (**Figure 8**).

Manifestations of the clinical course of HCM are fluctuating. A number of cases are asymptomatic or showing mild symptoms. Symptoms incorporate dyspnea (short breath) owing to stiffening and reduced blood filling of the heart ventricles, chest pain with effort and exertion (angina) as a result of decreased or restrained flow of the blood (referred to as ischemia) to the coronary arteries, palpitations due to the preceded ischemia, in addition to the interruption of the electrical system running across the defected heart muscle, light headedness, lethargy, passing out (Syncope) and sudden cardiac arrest. Respiratory distress basically attributed to accelerated left ventricular rigidity, which weakens the process of filling the ventricles, and however, also results in better strain within the left ventricle and atrium, resulting in posterior stress and interstitial congestion of the pleura.

Manifestations are distantly related to the existence and severity of the outermost gradient. Symptoms often imitate those of congestive heart failure (especially in activity and short of breath), but with different therapeutic regimen. Beta-blockers are used in each case but, using diuretics as a major pillar in therapy, will aggravate the signs and manifestations of obstructive hypertensive myocardial infarction through reducing the scale of the ventricular load and accordingly growing in the glide resistance (less blood to push aside thick blocking tissue).

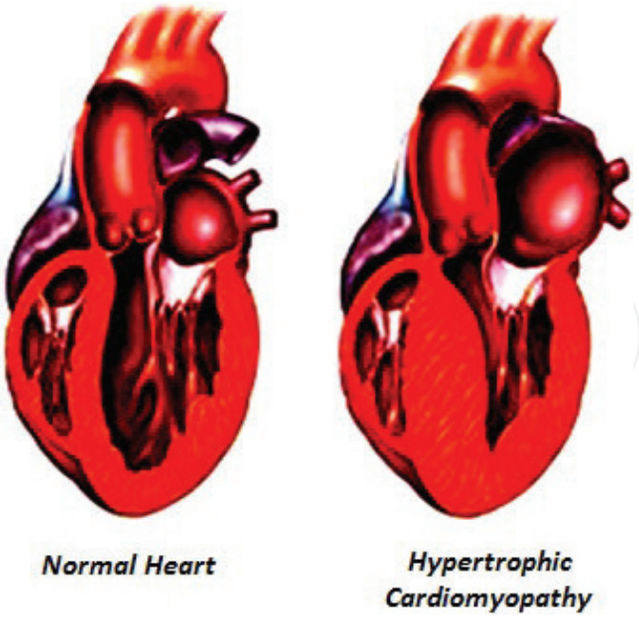

Figure 8. HCM also causes disruptions of the electrical functions of the heart. The cardiomyopathy is a disease that affects the heart muscle, with HCM and myocytes (the contractile cells of the heart) to increase the heart rate in size, leading to a thickening of the heart muscle. In addition, a phenomenon, the natural alignment of the cells of muscle breakdown, is known as chaos of the heart muscle. HCM also causes disruption of electrical functions of the heart.

The main risk of sudden death factors in individuals with an earlier history of cardiac arrest or ventricular fibrillation, spontaneous arrhythmia constant heart beat and ventricle, in exercise, blood pressure tachycardia and irregular heart rate and ventricle.

3.6. Dilated cardiomyopathy

Enlarged coronary heart (dilated cardiomyopathy) in cats is a heart disorder that impacts the ventricular muscle [12, 13].

It is characterized by dilated heart chambers, or enlarged, reduced the ability of deflation, that is, the lower the ability to push blood from the ventricle of them. It is suspected that this may have been related to the food shortages of the amino acid taurine.

Manifestations of reduced cardiac blood flow, caused by DCM:

- Physical and mental suffering (distress and depression), anorexia, and lethargy.
- Reduced blood flow as a result of blocked blood vessels, thromboembolism can be readily seen as an abrupt onset of ache and paraparesis (referred to as partial paralysis).
- Further examinations can show low or high coronary heart charge or regular, soft coronary heart puff, drop rhythm, hypothermia, left heart weak point, and quiet lung sounds.

3.6.1. Diagnosis

In addition to a comprehensive physical examination of the heart, some medical tests are needed for the diagnosis of DCM and the exclusion of other diseases. To record the electrical (or EKG) can be used to study electrical currents in the heart muscle, can reveal any defect

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in the electrical conduction of the heart (which lies behind the ability of the heart to contract/pulse), and can also help the veterinarian to determine the origin of irregular heartbeat, if they are present. X-ray imaging of the chest (pectoral rays) may reveal enlarged heart and fluid accumulated in the chest. Required echocardiography (ultrasound) imaging is carried to confirm the diagnosis of DCM. This test enables the veterinarian to check the eyesight of heart size and capacity of the ventricular muscle to contract. And echocardiography may reveal the thin walls of the ventricle, which is an enlarged left ventricle and the left atrium, and a reduced ability of deflation, which confirms the presence of the diagnosis of DCM. Treatment DCM varies with the state of the cat. If cat is suffering from severe symptoms, hospitalization may be necessary. It may include treatment for DCM drugs to control irregular heartbeat, and the Department of Health to prevent renal failure, treatment of low blood pressure, and treatment of complications from blood clots (i.e., blood thinning drug). And treatment in the hospital for congestive heart failure usually includes supplemental oxygen therapy, diuretics to reduce fluid retention, nitroglycerin to improve blood flow, and low doses of dobutamine to stimulate cardiac contractility and cardiac output. Other drugs, such as anticoagulants (blood) liquidity, and beta-blockers to control the rhythm can be used for the treatment of DCM, but their use depends on the specific problems that are secondary to the disease. Cats suffering from DCM will typically have loss of appetite, but they also need to give a low-sodium diet, to reduce the tension of fluid on the heart, and you need a diet that would raise the interesting cat eating plan, in order to assist in its recovery.

3.7. Pulmonary hypertension

Pulmonary hypertension happens in feline due to constriction of the pulmonary arteries and capillaries, blockage, or gain superfluous blood flow. Blood capillaries in the lung are minute branches of blood vessels, approximately thick as one cell. Linking smaller veins into smaller arteries for oxygen and carbon dioxide swapping between blood and surrounding tissues. Arteries transport oxygenated blood from the heart to the lungs, and pressure exerted by the blood on the left atrium can result in high pressure in the capillaries in the lungs.

3.7.1. Symptoms and types

1. Exercise intolerance
2. Trouble breathing
3. Bluish-purplish tinged skin
4. Coughing
5. Coughing or vomiting up blood
6. Enlarged abdomen
7. Weight Loss
8. Fatigue
9. Fainting

3.8. Pulmonic stenosis

Pulmonic stenosis is rarely seen in cats. Pulmonary stenosis is a congenital heart defect (the present one since birth), which causes a narrowing of the pulmonary valve to the heart (**Figure 9**). Pulmonary valve connects the right ventricle heart chamber to the pulmonary artery that carries blood from the heart to the lungs. When the pulmonary valve stenosis, in blocking the flow of blood from the heart to the lungs, right ventricle must work harder than usual to push the blood through. In many cases, the pressure builds up behind the obstructed pulmonary valve, leading to accumulation of fluid in the abdomen or chest that leads to congestive heart failure. Overworking the heart can also cause a heart murmur (abnormal heart sound) and cardiac arrhythmia (abnormal heart rhythm).

The defect or the illness is generally observed, while a heart murmur is detected all through a routine checkup, warranting further investigation. In moderate or severe cases, the animal may show symptoms of congestive heart failure. It includes:

- Difficulty breathing/shortness of breath
- Weakness
- Exercise intolerance
- Collapse treatment for pulmonic stenosis will ultimately depend on the severity of the defect. Cats with light or mild cases may live normally without the need for intervention. Cats with light or moderate cases may additionally live normally without the need for intervention. Cats with slight-to-severe defects, however, are normally handled with a minimal surgery called a vulvoplasty valve balloon. Balloon valvuloplasty is a catheterization

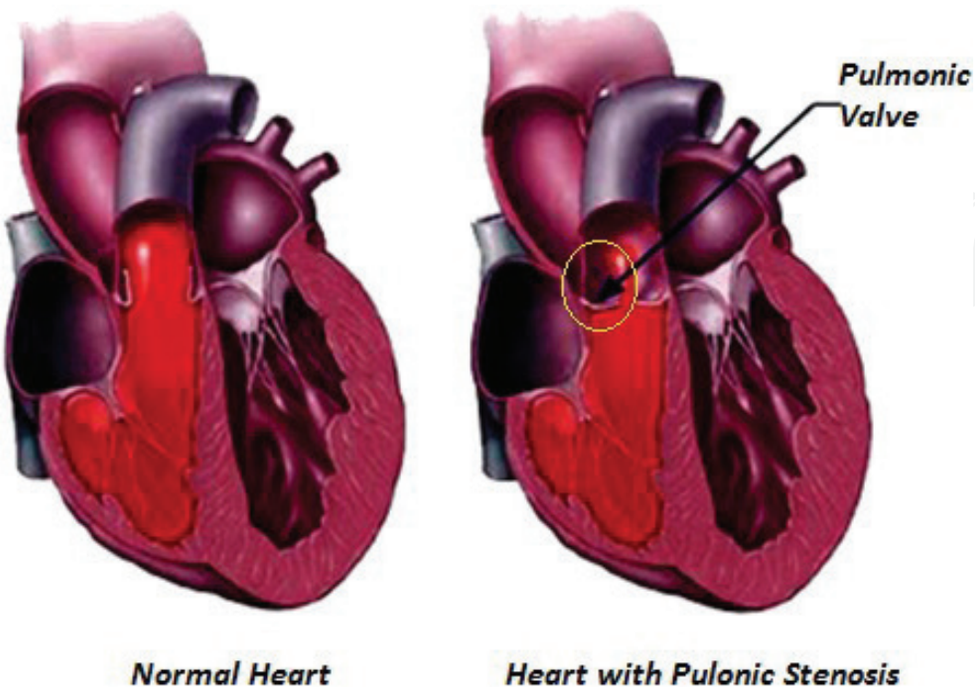

Figure 9. Pulmonic stenosis can range from mild to severe.

procedure, wherein a balloon is guided to the narrowed a part of the valve after which inflated, causing the valve to stretch and blood flow to improve.

3.9. Subaortic stenosis

- A rare defect in the cat [12, 13].
- Made of abnormal tissue placed under the aortic valve causing a blockage to the coronary artery. The cardiac muscle must exert more effort to pump the blood toward the frame.
- As a result of exerting more effort, the cardiac muscle starts to thicken (hypertrophy). Blood pumped at an elevated speed and pressure than normal through the stenosis into the aorta and creates heart murmur.

3.9.1. Symptoms

1. Lethargy.
2. Weakness following exercise or excitement.
3. Fainting.
4. In some advanced cases, coughing and difficulty breathing secondary to congestive heart failure.

Diagnosis of SAS echocardiogram with Doppler under supervision of board-certified veterinary specialized in heart disorders is a powerful tool helping to visualize the atria and ventricles, in addition to the subaortic area. Using Doppler helps in estimating the strain present in the heart by blockage (stenosis).

There is a correlation between elevated pressure and the degree of SAS. Cases with irregular cardiac rhythm need further examinations using ECG (**Figure 10**).

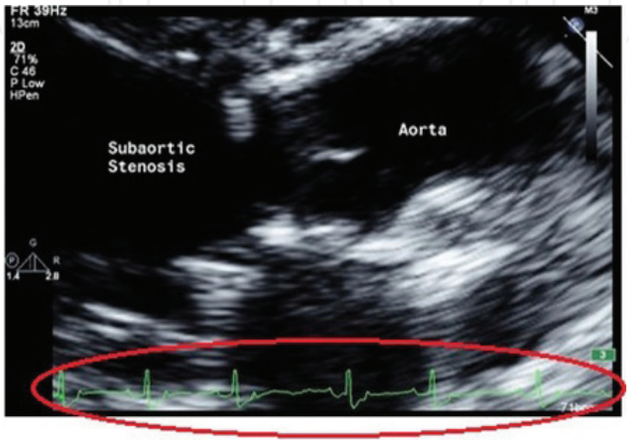

Figure 10. Echocardiogram with Doppler.

Treatment:

1. Prophylactic antibiotics
2. Limited exercise
3. Cardiac medications:

- Beta-blockers are often recommended for moderate-to-severely-affected SAS
- Additional therapy to treat specific arrhythmias and heart failure may also be required.

3.10. Left ventricular noncompaction

Left ventricular noncompaction or "spongy myocardium" is a rare congenital cardiomyopathy that can be diagnosed at any age. Eventually, this condition can potentially lead to chronic heart failure, life-threatening ventricular arrhythmias, and systemic embolic events. LVNC is a condition of the heart where the walls of the left ventricle (the bottom chamber of the left side of the heart) are noncompacted. This causes channels to form in the heart muscle, called trabeculations (**Figure 11**).

This offers the left ventricle a feature 'sponge' feature (a touch like honey). Even though it commonly impacts the left ventricle, it can additionally affect the proper ventricle.

Symptoms of left ventricular noncompaction (LVNC):

- Breathlessness
- Fatigue (extreme tiredness)
- Feeling dizzy
- Fainting or passing out (Syncope).

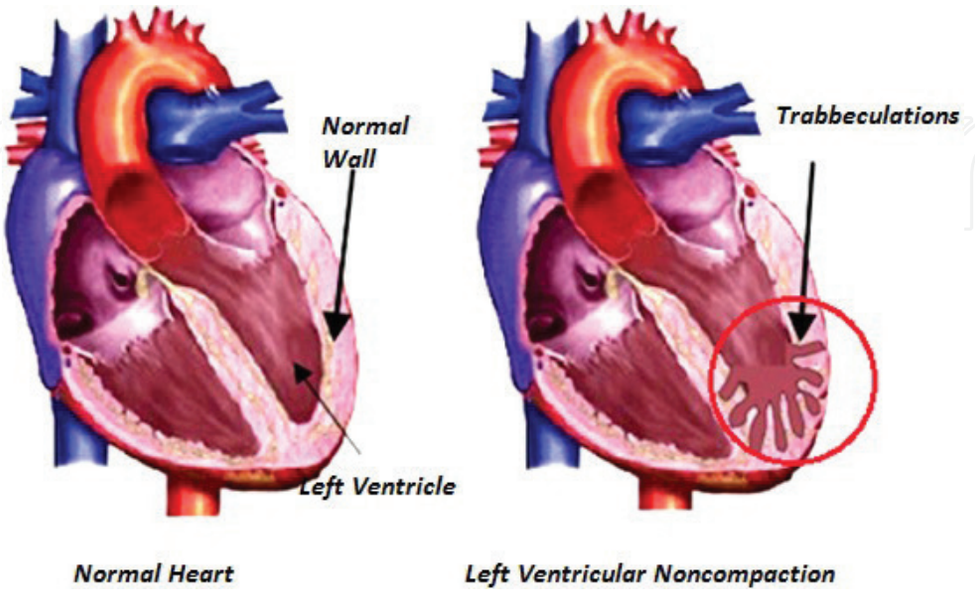

Figure 11. Left ventricular noncompaction or "spongy myocardium," is a rare congenital cardiomyopathy that can be diagnosed at any age.

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