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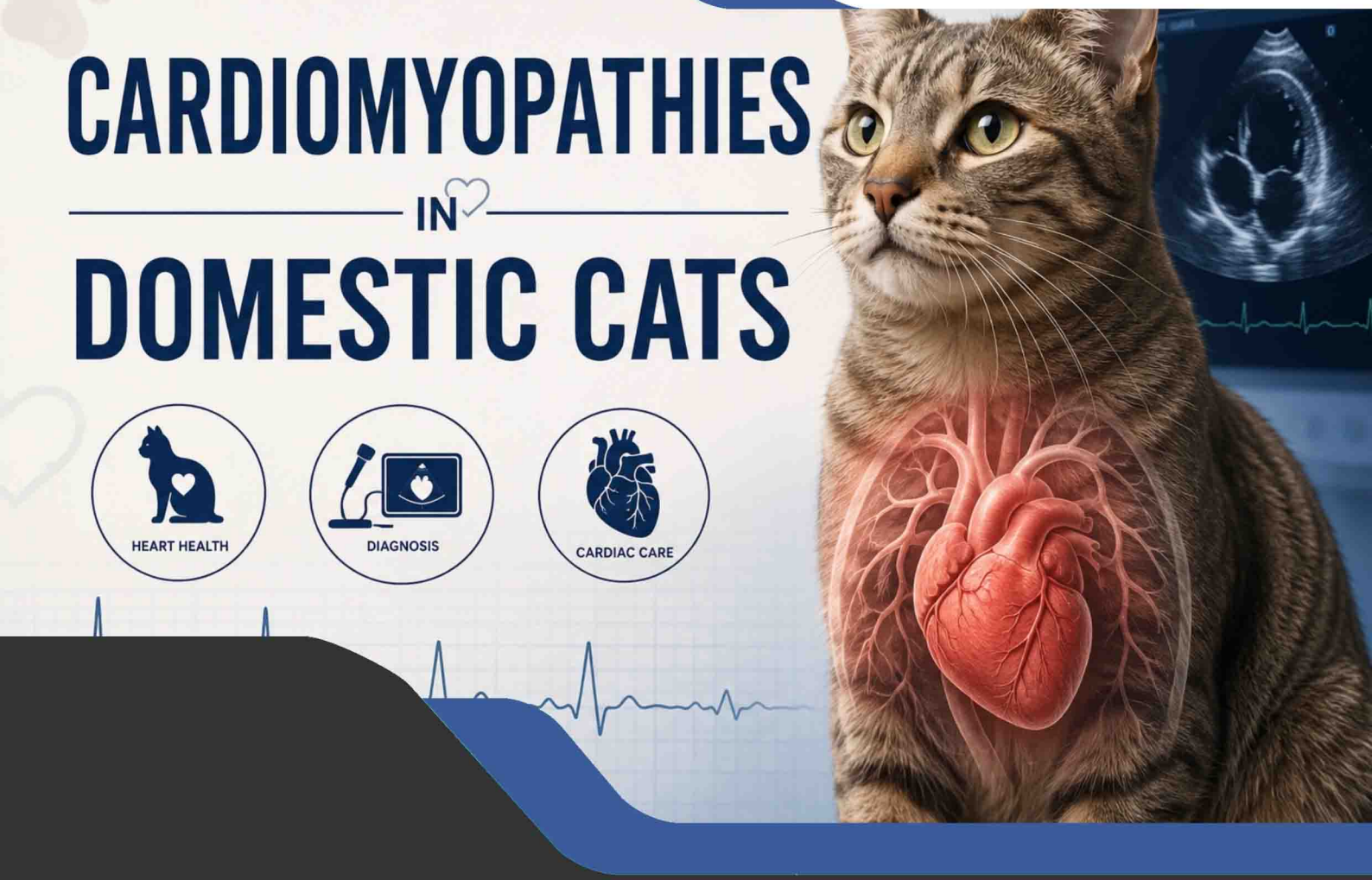

DIAGNOSIS OF CARDIOMYOPATHIES IN DOMESTIC CATS

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LIST OF ABBREVIATIONS:

HCM - hypertrophic cardiomyopathy

DCM - dilated cardiomyopathy

RCM - restrictive cardiomyopathy

ICM - intermediate cardiomyopathy

ECHO - echocardiography

ECG - electrocardiography

RAAS - renin-angiotensin-aldosterone system

AST - aspartate aminotransferase

ALT - alanine aminotransferase

ACEI - angiotensin-converting enzyme inhibitor

EDD - end-diastolic dimension of the right ventricle

EDL - end-diastolic dimension of the left ventricle

ESD - end-systolic dimension of the left ventricle

EDD - left ventricular wall thickness during diastole

LVs - left ventricular wall thickness during systole

IVSd - interventricular septal thickness during diastole

IVSs - interventricular septal thickness during systole

LA - size of the left atrium cavity during maximum filling

PP - size of the right atrium cavity during maximum filling

Ao - diameter of the aorta during maximum expansion in the systole phase

IS - contractility index

CTI - cardiothoracic index

CB - Buchanan coefficient (Viskapap).

FOREWORD

Modern veterinary medicine sets high standards for diagnosing various pathologies in animals. Today, such specialised areas of veterinary medicine as neurosurgery, oncology, orthopaedics, traumatology, cardiology, etc. are developing rapidly. Clinical research on animals in these areas requires the use of the most reliable methodological approaches, which include visual diagnostic methods. [4, 62–70, 84, 104].

Modern diagnostic capabilities are constantly expanding, and scientific advances have significantly influenced our understanding of heart pathologies in domestic cats. The use of already known examination methods – electrocardiography and radiography – as well as the introduction of the latest non-invasive methods of examining the heart of animals, such as echocardiography, enable veterinarians to accurately detect abnormalities in the anatomical structure of the heart and assess its functional disorders. The main heart pathologies in domestic cats are cardiomyopathies, the diagnosis and treatment of which is one of the most pressing issues in small animal veterinary medicine. [8, 11–16, 56, 63, 106].

Cardiomyopathies in domestic cats are diseases of significant clinical importance because they are asymptomatic. The most common forms of cardiomyopathy in domestic cats are hypertrophic, restrictive and dilated. It is the asymptomatic course of cardiomyopathy in domestic cats that complicates the diagnosis of these diseases and distinguishes them from similar diseases in other animal species. Signs of cardiomyopathy can appear quite unexpectedly, such as shortness of breath and the development of pulmonary oedema, pain in the pelvic limbs, and the development of aortic thromboembolism.

Such pathological conditions often lead to the death of cats, which, in some cases, has psychological, social and economic consequences for the owners of these animals.

Currently, cardiomyopathy in domestic cats is a poorly studied pathology in Ukraine. The most pressing issues today are the establishment of aetiological factors, diagnosis and treatment of cats with various types of cardiomyopathy.

UNIT 1
REVIEW OF THE LITERATURE

1.1. Anatomical features of the heart and blood circulation in adult cats

The heart is a muscular hollow organ that moves blood through a closed and branched system of vessels. In cats, the heart is elliptical-conical in shape, covered externally by the pericardium and located in the mediastinal space of the chest cavity. The longitudinal axis of the heart in cats forms an angle of about 25-30° with the sternum, and the apex of the heart is directed towards the diaphragm. The heart is located 4/7 to the left and 3/7 to the right of the mid-sagittal line and occupies the space between the III-VI intercostal spaces, and in the frontal plane - the space between the sternum and the middle of the chest cavity. Knowing the topographical anatomy, various methods of ante-mortem examination of the heart in cats can be applied [47, 123].

The heart consists of four chambers: the right and left atria, and the right and left ventricles. The atria are separated by the interatrial septum, and the ventricles by the interventricular septum. Functionally, the heart acts as two sequential pumps - the right and left halves. The right half of the heart is a low-pressure pump, and the left half is a high-pressure pump [49, 58].

The heart rate is controlled by the nervous system, hormones, and blood pressure. The heart's primary task is to maintain adequate blood pressure. In general, the heart rate can increase in order to maintain adequate blood pressure [58, 133].

Stroke volume is characterised by cardiac contractility. Cardiac contractility is the force of myocardial contraction, which depends on preload, afterload and true myocardial contraction. Preload is determined by the resistance of the myocardium to the end-diastolic volume of the ventricle. Afterload is determined by the pressure against which the heart pushes blood. True myocardial contraction is characterised by the ability of the heart muscle to contract [133].

Under unfavourable conditions, the action of certain aetiological factors causes a disturbance in haemodynamics, which triggers compensatory or decompensatory

mechanisms of its regulation. The main diseases that cause haemodynamic disorders and can lead to irreversible consequences or death of animals include myocardial pathologies - cardiomyopathies.

1.2 Classification of cardiomyopathies

The term ‘cardiomyopathy’ was first proposed W. Bridgen in 1957. This term characterised a group of heart diseases whose common feature is selective myocardial damage [2, 155]. Over the past decades, a group of heart diseases has been formed in paediatrics, united by a clinical and anatomical principle under the name ‘cardiomyopathies’ (CM) [50, 136].

Cardiomyopathy is considered to be various structural or functional changes in the myocardium. If the only site of heart damage is the myocardium, it is generally accepted that the animal is developing a primary (idiopathic) form of cardiomyopathy. If a specific aetiological factor (systemic and metabolic abnormalities) is identified, cardiomyopathy is considered secondary [122].

In 2006, cardiologists from the American Heart Association (AHA) proposed the following definition of cardiomyopathies: a heterogeneous group of myocardial diseases manifested by mechanical and/or electrical dysfunction, which usually (but not necessarily) leads to pathological hypertrophy or ventricular dilation. These diseases are the result of many causes, most often genetic. Cardiomyopathy is an isolated heart condition or part of a generalised systemic disease. It often leads to death due to cardiovascular pathology or progressive cardiovascular failure [114, 193].

The first classification of cardiomyopathies was proposed in 1961 by J. Goodwin. According to this classification, depending on the structural and functional characteristics of the myocardium, three forms of cardiomyopathy are distinguished: congestive, obstructive, and constrictive [2, 173].

However, it is believed that any classification is incomplete and acts as a bridge between complete ignorance and absolute understanding [173].

Based on physiological, clinical, and pathological data, cardiomyopathies can be classified according to their:

— morphological phenotype (hypertrophic or dilated cardiomyopathy); — aetiology (myocardial insufficiency caused by taurine deficiency, thyrotoxic heart disease);

— myocardial function (systolic or diastolic dysfunction);

— pathology (infiltrative cardiomyopathy, neoplasia);

— pathophysiology (restrictive cardiomyopathy) [168, 169].

In 1996, the World Health Organisation (WHO) approved a new classification of cardiomyopathies, which is still in use today. According to the definitions adopted within its framework, cardiomyopathies are diseases of the myocardium of unknown aetiology that lead to cardiac dysfunction.

According to the WHO classification (1996), the following cardiomyopathies are distinguished:

I. Idiopathic cardiomyopathies: dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), restrictive cardiomyopathy (RCM), arrhythmogenic right ventricular dysplasia, peripartum cardiomyopathy.

II. Specific cardiomyopathies, among which the following are distinguished:

1. Cardiomyopathies of infectious origin (viral, bacterial, fungal, rickettsial).

2. Metabolic cardiomyopathies: endocrine (occurring in acromegaly, thyrotoxicosis, diabetes mellitus); infiltrative and granulomatous processes, storage diseases (amyloidosis, leukaemia, lipidosis); deficiency of trace elements, vitamins, nutrients (selenium, potassium, magnesium, vitamin B1, kwashiorkor), anaemia.

3. Cardiomyopathies arising from systemic connective tissue diseases (rheumatoid arthritis, systemic scleroderma, lupus erythematosus).

4. Cardiomyopathies arising from systemic neuromuscular diseases (neuromuscular disorders, myotonia); due to the effects of toxic and physical factors (lead, phosphorus, hydrargyrum, anthracycline antibiotics, cyclophosphamide, uraemia, ionising radiation).

III. Unclassified CMP: fibroelastosis of the endomyocardium; non-compact (spongy) myocardium; left ventricular systolic dysfunction without dilatation or with minimal dilatation; myocardial damage at the mitochondrial level [2, 46, 114].

In 1997, the WHO Expert Committee proposed the use of the term ‘inflammatory cardiomyopathy’ for all heart diseases accompanied by an inflammatory process [114].

In 2006, the American Heart Association (AHA) proposed a classification in which DCM is included in the group of mixed cardiomyopathies, and myocarditis and inflammatory cardiomyopathy (ICM) are classified as acquired primary cardiomyopathies [140, 193].

Primary cardiomyopathies include: hypertrophic (asymmetric and symmetric; obstructive and non-obstructive), dilated (or congestive), restrictive (obliterative and diffuse) [128]. In a small number of cats, diagnosed cardiomyopathy can be classified as one of the above forms. In the vast majority of cases, the clinical manifestation of cardiomyopathy does not fit into the existing classification. Some forms of cardiomyopathy may have features of more than one functional or morphological class. Therefore, it can be assumed that myocardial pathology in these cases is an intermediate stage in the development of cardiomyopathy and, as the pathology progresses to its final stage, a myocardial impression will form that may be better suited to classification [20, 200].

In the intermediate (unclassified) form of cardiomyopathy, changes characteristic of several types of the disease may be observed, for example, hypertrophic and restrictive forms of cardiomyopathy [128].

Secondary cardiomyopathies include:

— metabolic (endocrine, nutritional);

— infiltrative (neoplasia);

— inflammatory (immune-mediated, feline coronavirus infection, feline viral immunodeficiency);

— fibrotic (post-infectious);

— toxic (use of doxorubicin, cyclophosphamide [16]);

— others (chronic renal failure, trauma, heart attack) [128].

Unfortunately, each classification may have its limitations. Therefore, some cases of cardiomyopathy may remain unrecognised and unclassified, while others may

be difficult to classify and assign to a specific form. Despite significant advances in veterinary medicine, certain difficulties remain in diagnosis and classification, as well as practical problems in the treatment of cats with cardiomyopathy. Although there are no clearly defined diagnostic criteria for these forms of cardiomyopathy, their classification and division into hypertrophic, restrictive and dilated cardiomyopathy continues to be important for clinicians [122].

Cardiomyopathy always affects the function of the heart ventricles. Myocardial damage with an established aetiology is secondary and is considered to be specific cardiomyopathy caused by the underlying disease [77, 114].

1.3. Etiology of cardiomyopathies

Cardiomyopathy (CM) accounts for more than 95% of all heart diseases in cats. In the vast majority of cats, this pathology is asymptomatic throughout their lives, so it is quite difficult to track the frequency of such cases. The most common symptomatic course of cardiomyopathy manifests itself in diastolic heart failure. The latter is characterised by diastolic dysfunction that occurs in hypertrophic and restrictive forms of cardiomyopathy [122, 165].

Almost all cardiomyopathies in cats were previously considered primary. Thus, the aetiological factors of hypertrophic cardiomyopathy are not identified in most cases. However, the situation has changed significantly after studying myocardial function in the presence of various systemic diseases. As a result, it can be assumed that studying the aetiology of cardiomyopathies in the future will lead to a situation where a large number of diseases will no longer be classified as idiopathic [49, 128].

Secondary forms of cardiomyopathy are more common in cats. Etiological factors that can lead to the development of secondary hypertrophic cardiomyopathy include hyperthyroidism, chronic anaemia, and kidney disease accompanied by arterial hypertension. Hyperthyroidism does not cause HCM, but leads to hypertrophic changes in the myocardium that may resemble HCM [76, 165]. Hyperthyroidism, in most cases, does not lead to significant cardiac dysfunction (except for cardiomegaly and tachycardia), but these do not lead to severe heart failure. In such cases, animals are not treated for cardiac pathology, except for

reducing the manifestation of tachycardia [120, 128]. Also, the development of hypertrophic changes in the wall of the left ventricle and interventricular septum may be a consequence of subaortic stenosis, which is a congenital heart defect. In this case, the developing cardiomyopathy can be classified as hypertrophic obstructive cardiomyopathy [143, 166].

Other congenital diseases include endocardial fibroelastosis. This disease has been described as a congenital anomaly: enlargement of the left atrium and ventricle is observed, accompanied by fibroelastic thickening of the endocardium. Endocardial fibroelastosis, like endocardial fibrosis, is the result of the formation of a very large amount of fibrous tissue in the endocardium, myocardium or subendocardial tissues, which is already a definition of restrictive cardiomyopathy [169, 177].

The aetiological factors that cause the development of restrictive cardiomyopathy in cats also remain unknown. However, it is assumed that the development of restrictive cardiomyopathy may be the result of inflammatory processes in the myocardium, which may have a long asymptomatic latent period and end with the manifestation of symptoms of diastolic failure [221].

At the same time, there is some certainty regarding the aetiology of dilated cardiomyopathy in cats. In 1987, Pion, Kittelson and their colleagues discovered that dilated cardiomyopathy (DCM) in cats is caused by a taurine deficiency in the diet [185, 205]. Taking these data into account, scientists at the New York Veterinary Centre (USA) conducted a study to determine the significance of taurine deficiency in dogs with DCM (n = 76). The study revealed a taurine deficiency in American Cocker Spaniels diagnosed with DCM. For the purpose of treatment, animals in this group were prescribed a taurine supplement to normalise myocardial function, similar to cats with DCM. During the treatment of the experimental groups, it was found that myocardial function did not return to normal, but the improvement achieved made it possible to discontinue the use of pharmacological agents [53]. However, it should also be taken into account that the development of dilated cardiomyopathy may be due to the course of late stages of intermediate cardiomyopathy or congenital valve diseases [90].

According to some authors, the most popular theory among humans is the viral aetiology of DCM, mainly enteroviruses, which is considered to be the result of viral myocarditis [2, 193]. Other factors that can cause DCM include pregnancy and childbirth [62], hereditary predisposition, exposure to toxic factors, nutritional deficiency of macro- and microelements and, possibly, some other substances [2]. An example of cardiomyopathy whose development may be related to nutrition is L-carnitine deficiency, which has been described in humans and dogs (boxer breed) [121, 181].

Currently, the link between myocardial damage and selenium deficiency has been fully established. Thus, dietary selenium deficiency leads to the development of a disease that morphologically and clinically resembles dilated cardiomyopathy. This disease is called 'Keshan disease'. The name of this disease comes from a region in China where the soil has a low selenium content and the population often experiences non-coronary myocardial damage, manifested by dilation of the heart chambers and symptoms of congestive heart failure [2, 46].

A large number of practising doctors in Ukraine tend to consider hypertrophic and restrictive forms of cardiomyopathy to be genetically inherited [89].

The first genetic studies of cats with HCM were conducted by Dr. Kittelson, who in 1999 claimed that HCM in Maine Coon cats was inherited in an autosomal dominant pattern with 100% penetrance (the indicator of phenotypic expression of the allele in the population). In turn, American cardiologists from the University of Washington, Dr. Meurs and Dr. Kittleson found the A31P mutation (gene I) in the cardiac myosin-binding protein-C3 (MYBPC3) gene in Maine Coon cats in 2005 [180]. And in 2007, Dr. Nuberg and colleagues discovered a point mutation A74T (gene II) in codon 74 of the cardiac MYBPC3 gene. Also, in 2007, Dr. Mers K.M. discovered a mutation in codon 820 of the cardiac MYBPC3 gene in Ragdoll cats [165, 187].

Taking into account the data from genetic studies of cats with HCM in recent years, the following conclusions were made:

— autosomal dominant inheritance is considered or suspected in such cat

breeds as Maine Coon, American Shorthair and British Shorthair;

— cat breeds that may be prone to HCM include Maine Coon, American Shorthair, British Shorthair, Norwegian Forest Cat, Siberian Forest Cat, Persian, Ragdoll, Burmese, Turquoise Van, Scottish Fold, and Sphynx;

— Only three mutations of the MYBPC3 heart gene have been described in cats: A31p, A74T, and R820W [187].

In 2010, German scientists from Ludwig Maximilian University in Munich, led by Dr. Wes (OegBaggi ^e88), conducted a study of the German cat population (n=86 Maine Coon cats and n=68 cats of various breeds). The studies were conducted using ultrasound examination of the heart (phenotype) and genetic analysis (genotype). The results of the studies were very interesting: in 21.7% of cats, the genetic test was positive, and in 78.3%, it was negative. In 83.3% of cats with a positive genetic test, the phenotypic ultrasound test was negative. Only 16.7% of cats with a positive genetic test had a positive ultrasound result, and 13.9% of Maine Coon cats with a genetically negative HCM test were diagnosed phenotypically (by echocardiography). These data indicate that there may be other causes of mutations or other aetiological factors of HCM [219].

Hypertrophic cardiomyopathy in humans is a primary genetically determined myocardial disease with an autosomal dominant pattern of inheritance [103, 130]. Currently, more than 100 different gene mutations are known [114]. For example, a mutation in the cardiac troponin T and I gene can cause the development of both hypertrophic and restrictive forms of cardiomyopathy [142, 185].

The following data are available on genetic research in human medicine. Genetic testing to differentiate hypertrophic cardiomyopathy in humans should be performed in cases where clinical examination data or the results of visual examination methods do not allow a diagnosis to be established. Genetic tests for several genes related to cardiomyopathy are currently available, but they have their limitations and problems. The sensitivity of genetic tests is low: a particular patient or family may have not one but several genetic variants of the pathology. Other problems include: the evaluation of genetic tests in children, which may include age-

dependent expression of the gene in the phenotype; the treatment of children who do not yet have clinical manifestations of the disease; the psychological consequences of testing minors with asymptomatic disease; and the high cost of genetic testing [207]

1.4. Pathogenesis and clinical manifestations of cardiomyopathies

In hypertrophic cardiomyopathy, symmetrical or asymmetrical hypertrophy of the left ventricle, papillary muscles, and interventricular septum is observed, which can significantly reduce ventricular filling with blood and cause diastolic heart failure. In most cases, systolic heart function does not change in hypertrophic cardiomyopathy, and the ejection fraction (contractility index) may increase. This significantly distinguishes hypertrophic cardiomyopathy from restrictive cardiomyopathy (fibrotic changes reduce myocardial contractility). To fill the left ventricular cavity with blood, the left atrium may enlarge and pressure in the pulmonary veins may increase, which can lead to pulmonary oedema and pleural and/or pericardial effusion [128, 165, 166]. In the vast majority of cases, the degree and area of left ventricular myocardial hypertrophy may vary, while the size of the left ventricular cavity remains normal [154]. With existing obstruction of blood flow from the left ventricle, concentric hypertrophy of the left ventricle, mitral valve insufficiency, and enlargement of the left atrium develop. The cause of obstruction of the left ventricular outflow tract may be aortic stenosis, which in more than 95% of cases is subvalvular (subaortic). Valvular stenosis is rare, and supravalvular stenosis is even rarer. As a rule, this pathology is more characteristic of young animals.

Differential diagnosis of the consequences of stenosis from HCM may be difficult [73, 165, 166]. Due to the changes observed in HCM, pressure overload is noted. This, in turn, increases intramyocardial tension, which causes the development of hypertrophic changes in the myocardium without ventricular dilatation [1, 73].

The histological picture of hypertrophic changes in the myocardium may vary, but most often hypertrophy and disorganisation of cardiomyocytes and fibrosis in the interstitial space are observed [128]. Fibrotic changes in the myocardium play a major role in the pathogenesis of severe arrhythmias, diastolic dysfunction, and progression of heart failure in HCM and other cardiovascular diseases. Hypertrophic changes in

cardiomyocyte myofibrils may be compensatory in nature against the background of fibrotic changes that form as a result of cardiomyocyte apoptosis. The results of studies of fibrotic changes in the myocardium in recent years increasingly emphasise that myocardial fibrosis is a possible primary factor in the development of HCM [38, 104, 127].

Fibrous changes in restrictive cardiomyopathy during histological examination are local or diffuse in nature to varying degrees in the myocardium and under the endocardium. Hypertrophic changes in cardiomyocytes, destruction of muscle fibres and inflammatory infiltration of myocardial tissue may also be observed. The development of restrictive cardiomyopathy begins when too much fibrous tissue forms in the myocardium, endocardium, or subendocardial tissues [46, 136]. Moderate hypertrophic changes in the left ventricular wall, similar to those seen in hypertrophic cardiomyopathy, and fibrotic changes can limit its filling, leading to diastolic heart failure, mitral valve insufficiency, and enlargement of the left atrium. Depending on the area of the myocardium affected by fibrotic changes, the systolic function of the heart may be significantly weakened. The ventricular walls become significantly rigid, which prevents the ventricles from filling completely with blood from the atria [128].

Myocardial infarctions, especially of the free wall of the left ventricle, can be a common occurrence. Ischemic myocardial necrosis is the result of an acute disturbance of coronary blood flow that does not meet the needs of the myocardium. Myocardial infarction may be suspected in cases where echocardiographic examination of the heart reveals regional or general myocardial hypokinesia or impaired contractility of the free wall of the left ventricle [122]. In cases of congestive cardiomyopathy, hypertrophic myocardial changes, and atrioventricular valve defects, microinfarcts of the myocardium can also occur in dogs [70], but this pathology is most common in humans [2].

A common feature of restrictive and hypertrophic cardiomyopathies is impaired adequate filling of the ventricles with blood, which is further exacerbated by the development of tachycardia. This, in turn, contributes to the development of

ischaemic phenomena in the myocardium and reduces its oxygen demand [122].

The main characteristic of the dilated form of cardiomyopathy is the pronounced development of systolic insufficiency, which occurs as a result of a decrease in the contractility (ionotropic function) of cardiomyocytes. The left ventricle and left atrium are enlarged (dilated), which is clearly visible during an ultrasound examination of the heart, and a decrease in the myocardial contractility index indicates systolic insufficiency. Due to the dilation of the heart chambers, the atrioventricular valve is stretched, which causes its insufficiency, leading to the appearance of systolic murmur.

In the dilated form of cardiomyopathy, histological changes in the myocardium may include degenerative changes in cardiomyocytes, foci of fibrosis, which may not cause severe myocardial dysfunction [128], as well as hypertrophic and atrophic changes in cardiomyocytes and their chaotic arrangement [136].

Chronic heart failure develops as a result of systolic or diastolic heart dysfunction. The systolic form of heart failure occurs as a result of pressure overload, volume overload, and/or myocardial damage. Pressure overload is the result of changes associated with narrowing of the heart valve structures, namely aortic stenosis, pulmonary trunk stenosis, bicuspid or tricuspid atrioventricular stenosis, as well as the onset of arterial hypertension. Volume overload is the result of blood regurgitation due to insufficiency of the atrioventricular valves, semilunar valves of the aorta and pulmonary trunk. Heart failure resulting from myocardial damage will be observed in the presence of myocardial infarction, myocarditis, and idiopathic cardiomyopathies. The development of diastolic heart failure is associated with increased ventricular wall rigidity and impaired ventricular relaxation. A decrease in the rate of ventricular relaxation is a consequence of a decrease in their filling with blood during diastole, and an increase in ventricular wall rigidity leads to an increase in end-diastolic pressure, which, accordingly, increases the load on the atria during ventricular systole and increases pressure in the latter. Such haemodynamic disturbances lead to atrial dilatation, congestion in the lungs and/or in the systemic circulation [1, 4, 49].

The symptoms of heart failure that occur in various heart diseases in domestic animals and cardiomyopathy in cats are no exception in this case. The causes and consequences of pathophysiological changes during each heart disease are different, but the response of the heart itself remains the same. Thus, the neurohormonal response to heart failure is a short- and medium-term compensatory process that maintains cardiac output and blood pressure. However, if the compensatory process continues for a long time, it leads to a decrease in heart function, which has adverse effects on the body. The neurohormonal response is the primary activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system (RAAS) [19, 38]. Understanding the compensatory process in heart failure allows us to develop the right therapeutic tactics that can increase the animal's lifespan.

The development of heart failure leads to increased sensitivity of baroreceptors, which enhances sympathetic innervation of the heart, kidneys, and peripheral vessels. Activation of the neurohormonal response leads to an increase in the concentration of noradrenaline in the blood plasma, which stimulates the β1-receptors of the heart and causes vasoconstriction, tachycardia, and sodium and water retention. Catecholamines with β1-adrenergic activity activate cyclic AMP, which leads to the binding of more calcium to the sarcoplasmic reticulum during diastole and then its release during systole. The main ion involved in myocardial contraction is calcium. The catecholamine noradrenaline is a factor in this process that increases the rate of binding and release of calcium ions. The response to increased sympathetic innervation of the heart is an increase in the synthesis of the proteolytic enzyme renin in the kidneys. Under the action of renin, angiotensinogen, a blood protein, is converted to angiotensin I, which, in turn, is converted to angiotensin II under the action of angiotensin-converting factor (ACF, secreted by the cells of the pulmonary vessel endothelium and renal epithelium). Angiotensin II has a vasoconstrictor effect and stimulates the secretion of aldosterone from the adrenal cortex. Aldosterone increases the reabsorption of sodium ions and water in the renal tubules, which increases the volume of circulating blood (VBC), and this, in turn, maintains mean arterial blood pressure [1, 133]. Activation of the renin-angiotensin-aldosterone

system is associated with long-term compensatory processes of heart failure and influences the mechanisms of myocardial hypertrophy [38]. According to some authors (Drapkin O. M., Chekunov E. V., Drapkin Yu. S., 2012, 2014), RAAS hyperactivation is the cause of chronic heart failure. In recent years, views on RAAS have changed: there are separate, fairly autonomous, intraorgan renin-angiotensin-aldosterone systems in various organs - the liver, pancreas, heart, and kidneys. In these organs, RAAS is involved in the processes of cell growth and death, inflammation and fibrosis.

The clinical manifestation of cardiomyopathies will depend on whether diastolic or systolic heart failure is compensated or decompensated. The main symptoms include fatigue, shortness of breath, arrhythmia, and arterial thromboembolism. Fatigue is not so noticeable, but in dogs it is the earliest sign of heart failure [22]

. Given that even clinically healthy cats can feel anxious, stressed, and worried during a visit to the vet, symptoms such as shortness of breath and arrhythmia may go unnoticed. With a rather limited range of clinical signs, a large number of cardiomyopathies in sick cats remain undiagnosed [20]. Most often, cardiomyopathies are asymptomatic and end in sudden death [46].

Arterial thromboembolism is one of the most serious consequences of cardiomyopathy. Sometimes it is the primary reason for animal owners to visit a veterinary clinic. Thromboembolism can occur in any form of cardiomyopathy. Risk factors that can lead to thrombus formation include enlargement of the left atrium by more than 20 mm, blood stasis (especially in myocardial failure or atrial fibrillation) and endocardial fibrosis [122]. The clinical manifestation of thromboembolism depends on the degree of heart failure and damage to tissues and organs affected by embolism. Most often (more than 90%), the embolus obstructs the aorta at the level of the bifurcation into the femoral arteries. The symptoms of this pathology are paresis or paraparesis of the pelvic limbs (depending on the size of the thrombus), pain in the muscles of the pelvic limbs (ischemic myopathy develops), and weakening or absence of the pulse in the femoral arteries. Rarely, thromboembolism can occur in other

places: the chest (limb, which can cause lameness), renal arteries (which can develop severe azotaemia), mesenteric, coronary and other arteries. Coronary, renal, mesenteric or pulmonary embolism can lead to rapid death. Thromboembolism results in the development of ischaemia, gangrene [210], DIC syndrome, metabolic acidosis and hyperkalaemia. The prognosis for thromboembolism depends on the size of the ischaemic area and the severity of cardiomyopathy. During pathological autopsy, thromboembolism can be detected in 25-48% of cats with cardiomyopathy [74, 128].

Cardiomyopathies can also cause depletion of the animal's adaptive systems, namely hyperstimulation of the adrenal cortex. Data on the decrease in the level of adrenal cortex hormones in blood plasma may confirm this assumption [78].

1.5. Diagnosis of cardiomyopathies

The diagnostic approach in cases of cardiomyopathy is primarily based on medical history and basic physical examination methods. Physical examination methods provide the doctor with basic information that allows them to suspect the presence of cardiac disease in a cat. Attention is paid to the breed and age of the cat, taking into account the hereditary predisposition of cats of certain breeds to specific forms of cardiomyopathy. Congenital heart defects are more common in young animals, while acquired pathologies are more common in older animals. Due to general weakness, some animals are inactive, while others adopt a forced body position with their elbows extended to improve lung ventilation [49].

Cats suffering from cardiomyopathy most often come to the veterinary clinic in a state of dyspnoea (rapid and labored breathing) and behave restlessly. Auscultation of the heart reveals tachycardia, and systolic murmur is noted in cases of bicuspid valve insufficiency. However, even healthy cats may experience tachycardia when they are anxious. Cardiomyopathy cannot be ruled out in cats with shortness of breath in the absence of heart murmurs [20]. The presence of a gallop rhythm is a specific sign of cardiac pathology in general and cardiomyopathy in particular [22, 49]. Auscultation can reveal the presence of rales in the lungs, which is a sign of developing pulmonary oedema.

The vast majority of doctors recognise that the possibility of detecting HCM

using physical examination methods is quite low [83, 212]. None of the detected symptoms is a reliable sign of any form of cardiomyopathy in a sick animal. Confirmation of the diagnosis of a specific type of cardiomyopathy requires instrumental and hardware diagnostic methods.

Laboratory methods that can be used for cardiovascular diseases include general and biochemical blood tests. However, these blood test methods do not provide diagnostic information about the pathology in the myocardium itself [86]. Anaemia, especially prolonged anaemia, can cause heart failure. The presence of anaemia is confirmed by such indicators of a complete blood count as a decrease in haemoglobin concentration and a decrease in the number of red blood cells. The kidneys are the first to respond to circulatory failure, so the concentration of creatinine and urea in the blood serum can confirm the manifestation of renal failure. Hemodynamic disturbances in congestive heart failure lead to blood stasis in the liver vessels. This, in turn, can lead to cholestasis and impaired liver detoxification function, which is recorded in the results of biochemical blood tests (increased activity of transaminases, alkaline phosphatase, gamma-glutamyltransferase, cholesterol content) [22].

The first biomarkers (substances produced by certain cells and detectable in the blood) of heart condition include aspartate aminotransferase, lactate dehydrogenase, and creatine phosphokinase. However, due to the large number of isoenzymes in skeletal muscles, these indicators have no diagnostic value. Currently, cardiac troponin and natriuretic peptides are reliable indicators for detecting cardiomyocyte damage [98, 115]. Troponins are protein molecules formed into a complex of three subunits (Troponin I, Troponin C, Troponin T) and located on actin filaments in striated and cardiac muscle tissue. It is the troponin complex that is involved in the processes of myocardial contraction and relaxation. One of the most sensitive markers of myocardial damage is cardiac troponin I, which does not reflect damage to skeletal muscles [100, 115, 203, 213, 220].

The electrocardiography method for confirming the diagnosis of cardiomyopathy is not very informative. In cats, normal ECG values in the lateral

position of the animal may not be recorded. Each cat is unique, so the veterinarian should determine which body position is most comfortable for performing electrocardiography in a particular animal. Sedatives can have different effects on the heart and/or autonomic tone. Such drugs can directly affect heart rate and rhythm, and indirectly through their effect on autonomic tone. For more reliable electrocardiography data, it is advisable to discontinue all medications used in the sick animal to exclude their side effects on the electrocardiogram recording [75]. In practice, this is often impossible because animals are brought to the doctor with severe cardiovascular failure and require immediate intensive care.

The main abnormalities that can be recorded on an ECG are heart rhythm disturbances and impulse conduction. However, such changes are not specific to a particular form of cardiomyopathy. In various cardiomyopathies, ECG can detect high or wide P waves, which may indicate atrial enlargement; various tachyarrhythmias or conduction disturbances [128].

ECG has significant diagnostic sensitivity and specific irregular signs, but does not provide reliable information, as abnormal data can also be obtained in clinically healthy cats [40]. To avoid contradictions, it is better to obtain additional information from X-ray and echocardiographic examinations of animals [177].

The X-ray method allows visualisation of the consequences of congestive heart failure resulting from the development of cardiomyopathies. These consequences include pulmonary oedema, pleural effusion, or venous congestion. Differential diagnosis of dilated, hypertrophic, and restrictive forms of cardiomyopathy can be performed based on certain qualitative features obtained by radiography. However, to confirm the diagnosis of any of the three forms of cardiomyopathy listed above, none of these signs should be considered reliable. In hypertrophic cardiomyopathy, the atria may be enlarged, so the heart may appear ‘valentine-shaped’ in the dorsoventral projection. However, the atria may also be enlarged in restrictive cardiomyopathy. In dilated cardiomyopathy, generalised cardiomegaly may be observed, or the heart shadow may be normal [74].

The enlargement of the atria results in a constant increase in venous pressure,

leading to ascites and pericardial effusion. In the presence of pericardial effusion, the apex of the heart may appear rounded on X-ray images [128].

Chest X-ray data should not be used for the differential diagnosis of various forms of cardiomyopathy [74], as chest X-rays provide only indirect information about the overall condition of the heart [20].

One type of X-ray is non-selective angiography. This method is based on the use of contrast agents in peripheral veins, followed by X-ray imaging. However, this method is invasive and has a number of disadvantages (the possibility of an allergic reaction and anaphylactic shock to contrast agents, the need to perform X-ray imaging very quickly after the administration of contrast agents, etc.).

Therefore, the use of non-selective radiography in general practice is not advisable [74, 128].

Echocardiography (ultrasound examination of the heart) is the most informative and non-invasive method of examining the heart [22]. The examination is performed by transthoracic ultrasonography using a sectoral (phased) ultrasound sensor, which is currently multi-frequency, improving the visualisation of the heart in animals of different body weights. The structures of the heart are visualised in two-dimensional B-mode, which provides a complete overview of the anatomical structures of the heart, and in one-dimensional M-mode, which provides images of the phases of the cardiac cycle. The standard is to visualise a 'virtual' dissection of the heart along the long axis (longitudinal dissection - from the apex to the base of the heart) and the short axis (transverse dissection) of the heart (Fig. 1.1 and Fig. 1.2).

Accurate quantitative data can be obtained in the standard one-dimensional M-mode, in which the examination is performed in depth and in real time (Fig. 1.3). This mode allows you to measure cardiac structures, time intervals, and obtain a graphical image of cardiac structures by phases. Echocardiography can be used to obtain information about the internal structure of the heart and its function, and to compare heart parameters with normative data (Table 1.1).

The functional parameter of the heart is the contractility index, which is the percentage of contraction of the small axis of the heart during systole and is calculated

using the formula: CI = (LVd - LVs) / LVd x 100%.

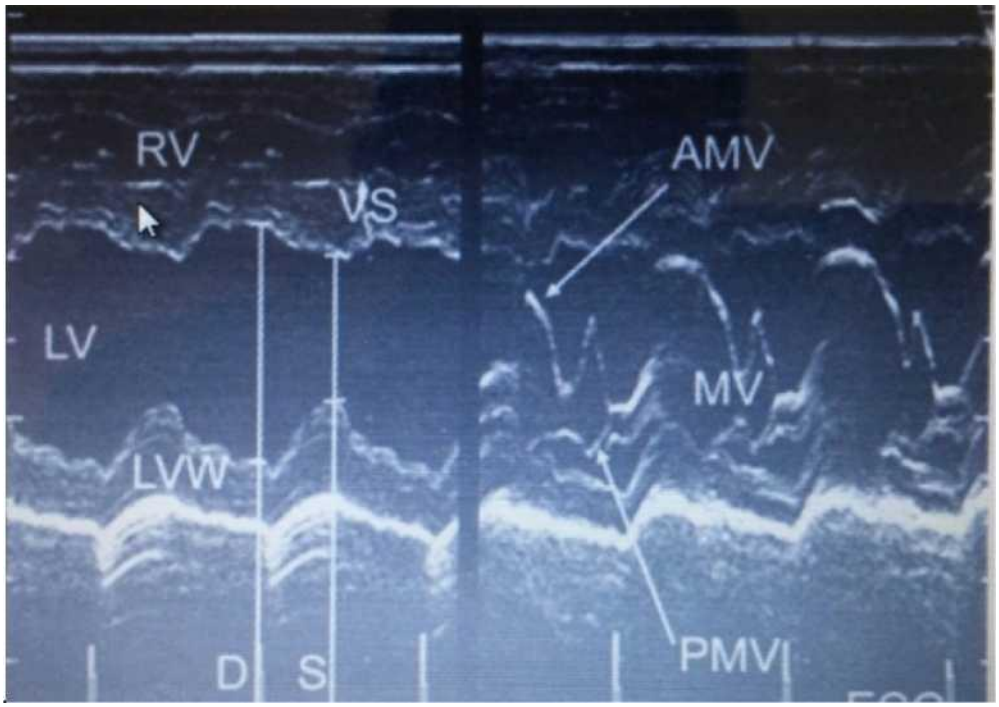

Fig. 1.1. Ultrasonogram of an animal's heart in M-mode in the right parasternal projection. The vertical axis represents the depth of the scan, while the horizontal axis represents time. On the right side of the ultrasonogram, a 'virtual' dissection of the heart is performed between the papillary muscles, and on the right side of the ultrasonogram, at the level of the mitral valve. [73]

— IC values below normal indicate a decrease in myocardial contractility (hypokinesis), while values above normal indicate hyperkinesis [48, 129].

— Echocardiography is the most sensitive and specific method for diagnosing and classifying cardiomyopathies by phenotype [187].

— Let us consider the information that can be obtained by echocardiography, which would characterise the restrictive form of cardiomyopathy:

— moderate hypertrophy of the left ventricular wall and, less commonly, the interventricular septum may be observed;

— enlargement of the left atrium (more than 16 mm, and more often more than 20 mm) in the absence of significant volume overload, for example, in mitral valve insufficiency;

— the internal size of the left ventricle is normal or slightly enlarged, the lumen of the left ventricle may be uneven due to varying wall thickness and fibrosis;

— possible presence of pericardial effusion;

— left ventricular myocardial contractility index is normal or reduced to less than 26-29%;

— localised (focal) thinning of the left ventricular wall associated with myocardial infarction or scar formation is sometimes observed;

— bright hyperechoic endocardium is a sign of endocardial fibrosis.

Table 1.1

Normal echocardiography parameters in cats [165, 178].

Parameter	Unit of measurement	Variation range (n - 30)
End-diastolic size of the right ventricle cavity	mm	2,7 - 9,4
End-diastolic size of the left ventricle cavity	mm	12,0 - 19,8
End-systolic left ventricular cavity size	mm	5,2 - 10,8
Left ventricular wall thickness during diastole	mm	2,2 - 4,4
Left ventricular wall thickness during systole	mm	5,8 - 8,1
Interventricular septal thickness during diastole	mm	2,2 - 4,0
Interventricular septal thickness during systole	mm	4,7 - 7,0
Left atrial cavity size during maximum filling	mm	9,3 - 15,1
Aortic diameter during maximum expansion in the systolic phase	mm	7,2 - 11,9
Contractility index	%	39,0 - 61,0
Ratio of left atrial size to aortic diameter		0,95 - 1,65

Some cats may show significant enlargement of the left atrium (often more than 22 mm) and localised damage to the left ventricular myocardium in the form of hyperechoic spots or bands. There may also be enlargement of the right heart and other structural and functional changes characteristic of endomyocardial fibrosis, which is an exceptional form of progressive restrictive cardiomyopathy and has an extremely poor prognosis [17, 74, 129, 147, 165].

According to echocardiography, the hypertrophic form of cardiomyopathy can be characterised by the following indicators:

— thickening of the interventricular septum and left ventricular wall greater than 6 mm at the end of diastole; thickening may be segmental;

— hypertrophy of the papillary muscles of the left ventricle;

— possible enlargement of the left atrium greater than 16 mm;

— systolic function is normal, unless secondary mitral valve insufficiency is severe;

— ventricular size is reduced due to hypertrophy of the ventricular walls and interventricular septum;

— massive left ventricular hypertrophy is sometimes observed: the thickness of the interventricular septum and left ventricular wall may exceed 7.5 mm at the end of diastole [17, 48, 74, 129, 147].

The main echocardiographic characteristics of dilated cardiomyopathy include:

— enlargement of the left atrium greater than 16 mm;

— enlargement of the left ventricular cavity (end-diastolic size greater than 21 mm);

— reduction in the thickness of the left ventricular wall and interventricular septum (or may reach the lower limits of normal);

— reduction in the contractility index to less than 25-30% [17, 48, 74, 129, 147].

Echocardiography is the best and most sensitive method for detecting myocardial hypertrophy by phenotype [187].

A schematic representation of the characteristics of different forms of cardiomyopathy is shown in Fig. 1.4 [30].

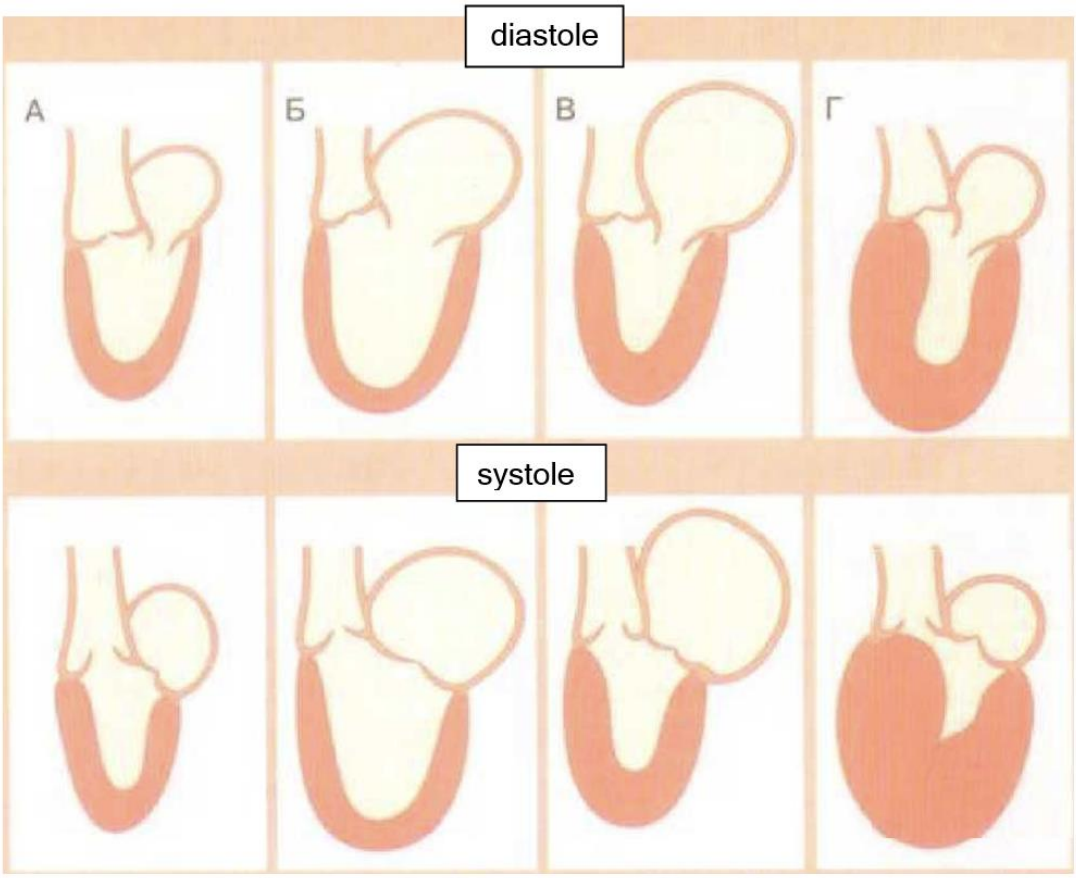

Fig. 1.4. Schematic characteristics of various forms of cardiomyopathy: A - normal, B - dilated cardiomyopathy, C - restrictive cardiomyopathy, D - hypertrophic cardiomyopathy [30]

1.6. Treatment of animals with cardiomyopathy

There is currently no effective treatment for cardiomyopathy in animals. Treatment is mainly symptomatic and pathogenetic in nature, namely: reduction and elimination of congestive phenomena (oedema and effusions) and manifestations of tachyarrhythmias. In the presence of systolic dysfunction, it is necessary to improve myocardial contractility. In the presence of diastolic dysfunction, it is necessary to improve myocardial relaxation and ventricular filling with blood. It is also necessary to prevent the development of arterial thromboembolism and to treat animals for the development of such pathology. Extending the life expectancy and quality of life of sick cats, along with eliminating existing aetiological factors and preventing concomitant pathologies, is the main goal of treating cats with cardiomyopathy [122, 165].

Treatment of cats with cardiomyopathy usually begins when clinical symptoms

appear. To date, there are no studies or evidence that would at least theoretically indicate the effectiveness of drugs in the treatment of cats with cardiomyopathy. For example, in the subclinical form of hypertrophic cardiomyopathy, there are very few drugs (Ca blockers, ACE inhibitors, b blockers) that are used empirically and show a positive effect on the course of the disease [177, 216].

When a patient with cardiomyopathy develops pulmonary oedema, arterial thromboembolism, pericardial or pleural effusion, i.e. conditions that are progressive and may be life-threatening, diuretics can be used to quickly reduce left ventricular filling pressure, such as loop diuretics (furosemide or torasemide, which cause rapid and strong diuresis) or competitive aldosterone antagonists (spironolactone) [20, 74, 128, 143]. The use of thiazide diuretics (e.g., hydrochlorothiazide) for the treatment of cats with HCM is ineffective, as evidenced by the lack of information on their use in cats [23]. Cats with asymptomatic cardiomyopathy without congestive heart failure should not be treated with diuretics, as they can activate the RAAS system, which has an adverse effect. Asymptomatic cardiomyopathy in cats can sometimes last for years, and cats can live a full life during this time [122].

In the case of accumulation of large amounts of effusion in the pleural or pericardial cavities, thoracocentesis with subsequent evacuation of fluid is used to eliminate the development and manifestation of respiratory distress syndrome [20, 74]. To reduce tissue hypoxia, oxygen therapy is used to improve gas exchange in the lungs [49, 128].

Beta-blockers for the treatment of cats with cardiomyopathy are effective only as antiarrhythmics. Their effectiveness in hypertrophic cardiomyopathy has also not been noted [24, 165, 179].

Given that neurohumoral activation plays a major role in the processes of cardiac decompensation, the leading drugs for the treatment of sick animals with cardiomyopathy should be considered angiotensin-converting enzyme (ACE) inhibitors, which suppress the RAAS (renin-angiotensin-aldosterone) system. Among these drugs, the most common are enalapril, lisinopril, captopril or their analogues [122, 126, 137, 143, 165, 209]. Experimental and clinical studies show the high

efficacy of RAAS blockers in reducing myocardial fibrosis [60, 148, 215].

In human medicine, there is experience with the use of angiotensin II receptor antagonists (e.g., valsartan), which affect the RAAS system, as do ACE inhibitors, and have high organ-protective potential. However, despite significant experience in the use of these drugs, there are no clear recommendations for their use [126, 196].

Aspirin, which inhibits platelet aggregation, can be used to prevent thromboembolism and treat cats with a severely enlarged left atrium [49, 74, 122]. The use of thrombolytic drugs (e.g., streptase or urokinase) for the lysis of already formed thrombi is inappropriate due to the occurrence of side effects, and the use of anticoagulants for this purpose is ineffective, as they do not act on already formed thrombi [122].

Diet therapy makes a significant contribution to the prevention and treatment of heart disease. The use of nutrients such as the essential amino acids arginine, carnitine (a compound of lysine and methionine), taurine; vitamin E, omega-3 polyunsaturated fatty acids, etc. provides the animal's body with the necessary amount of energy, minimises oxidative stress, reduces inflammatory processes, ensures electrolyte balance and, thus, improves heart function. Currently, leading global companies and the Centre for Animal Nutrition WALTHAM have developed specialised diets that meet the nutritional needs of small pets with cardiac disorders [8, 34, 143].

The prognosis for cats with any type of cardiomyopathy is difficult to determine. Some cats may respond poorly to treatment and live only a few days, while others may respond positively to treatment and live for many years. The prognosis for cardiomyopathy in cats depends on the response to treatment and the financial and moral capacity of the owners to treat the animal [20, 49].

Summarising the literature review, it should be noted that cardiomyopathies in domestic cats are very common diseases that are predominantly asymptomatic and often lead to the death of the animal. The most common forms of cardiomyopathy in domestic cats are hypertrophic, restrictive and dilated.

In most cases, the aetiological factors of various forms of cardiomyopathy have remained and remain unclear and continue to be considered primary diseases. The

situation has changed and will continue to change during the study of the structure and function of the myocardium in various systemic diseases.

An important issue in the research and study of cardiomyopathies in domestic cats is the diagnostic approach to these diseases. Although laboratory diagnostic methods, such as biochemical blood analysis, are an integral part of the diagnosis of many pathologies, they are not sufficient for the final diagnosis of any form of cardiomyopathy. Instrumental examination methods, namely ultrasound examination of the heart, allow cardiomyopathy to be confirmed.

Despite significant progress in the study of cardiomyopathies in domestic cats, there are still relevant and insufficiently studied aspects of aetiology, improvement of diagnostic methods, and the development of prevention and treatment regimens for cardiomyopathies in domestic cats.

In-depth research and study of cardiomyopathies in domestic cats will allow for the early detection of animals suspected of having these diseases, timely diagnosis and classification of the type of cardiomyopathy, and individualised preventive measures and treatment of sick animals.

UNIT 2
RESEARCH AREAS, MATERIALS AND METHODS OF WORK

2.1. Selection of research areas

After analysing information on heart pathology, namely cardiomyopathy, in mammals in general and in domestic cats in particular, as reported in domestic and foreign literature, we focused our attention on the study of cardiomyopathies in domestic cats, which requires in-depth clinical, laboratory and instrumental studies of sick animals.

Clinical studies of domestic cats with cardiomyopathy continue to be of great importance, as the disease in outwardly healthy cats can occur in a subclinical form [20, 67, 74, 128].

Laboratory studies of domestic cats with cardiomyopathy have recently been receiving increasing attention.

These studies allow the identification of the aetiological factors of the disease and the establishment of the pathogenetic factors of myocardial damage in sick animals [115, 158, 175, 203]. Instrumental research methods allow the visualisation of pathological changes in the heart in various diseases in general and in cardiomyopathies in particular.

Thus, with the help of ultrasound examination of the heart, it is possible to obtain data characterising the structural and anatomical changes in the heart and to establish haemodynamic disturbances in sick animals with cardiomyopathy [20, 47, 73, 76, 77, 116, 128].

The results of our studies can serve as a basis for the effective use of specific echocardiographic indicators of the heart and laboratory indicators of blood serum in the diagnosis and classification of cardiomyopathy in domestic cats.

2.2. Subject and location of research

The research presented in the monograph was conducted at the Department of Internal Diseases of Animals of the National University of Life and Environmental

Sciences of Ukraine. Experimental studies were conducted in a network of clinics of the Alden Vet veterinary centre in Kyiv.

The subjects of the study were healthy and sick domestic cats (Ge1I8 dotesiiz) with cardiomyopathy. To establish the diagnosis, clinical, laboratory and instrumental methods of animal examination were used according to the scheme (Fig. 2.1). During the study, 16 clinically healthy and 58 domestic cats with cardiomyopathy were examined. The study was conducted in the following order:

— establishing clinical manifestations of cardiomyopathy in domestic cats;

— performing echocardiography and determining the echocardiographic characteristics of various types of cardiomyopathy in domestic cats;

— conducting general and biochemical blood tests for cardiomyopathy in domestic cats;

— conducting ELISA assay of blood serum for cardiomyopathy in domestic cats to study the concentration of cardiac troponin I;

developing a scheme for diagnosing cardiomyopathy in domestic cats.

2.3. Research methods

2.3.1. Clinical research methods

After the animal was admitted to the veterinary clinic, it was registered and its medical history was collected. The clinical examination of the animals was carried out according to a general scheme with an examination of the functional state of organs and systems [47]. Blood samples were taken from the animals for laboratory tests, and echocardiography and, if necessary, X-ray and electrocardiography examinations were performed.

In some cases, when cats were admitted to the veterinary clinic in urgent conditions (pulmonary oedema, arterial thromboembolism), emergency medical care was first provided to stabilise the clinical condition, and then the general procedure was followed.

We have identified and described the most informative indicators of echocardiographic examination of the heart, which most reliably characterise the anatomical and functional state of this organ.

2.3.2. Blood testing methods

Blood for research in domestic cats was collected on an empty stomach from the superficial vein of the forearm (V. cephalica) or from the superficial vein of the pelvic limb (V. saphena).

During the analysis of the animals' blood, the concentration of haemoglobin, the number of erythrocytes, leukocytes and leukogram were examined. A complete blood count was performed using a BC-2800Vet haematology analyser from Mindray (China).

Biochemical blood tests were performed using a Stata Fax 1904+ biochemical analyser manufactured by Awareness Technology (USA) with reagents from BioSystems. The following indicators were studied in the blood serum of cats: AST and ALT activity, creatinine, urea and glucose concentrations, as well as total protein content.

An immunoenzymatic analysis of domestic cat blood serum was used to study the concentration of troponin I. For this purpose, a Rayto-3100 immunoenzymatic analyser from Rayto (China) was used.

Among the results of general and biochemical blood tests, there are no indicators that clearly indicate the presence of cardiomyopathy in domestic cats. However, these tests are necessary to rule out concomitant pathologies or pathologies that may serve as aetiological factors in the development of cardiomyopathy in domestic cats.

2.3.3. Instrumental research methods

Echocardiography was performed using the MyLab Class C ultrasound systems from Esaote and the Imagic Agile from Kontron Medical, using sectoral (phased) multi-frequency sensors. To perform echocardiography, the animal was placed in a right lateral position on a special table with U-shaped cutouts so that the area of the chest with the most palpable heartbeat was located above the cutout of the table, and the lower chest limb was maximally abducted cranially. The hair from the examined area of the animal's body was shaved as much as possible, and a special acoustic gel was applied to the skin. Standard echocardiographic accesses were used to visualise

the heart structures, namely: right parasternal access along the long axis of the left ventricle; right parasternal access along the short axis of the left ventricle, at different levels of visualisation of the anatomical heart structures. An important aspect of conducting research is not the position of the sensor relative to the chest of the animal being studied, but obtaining a clear visualisation of the anatomical structures of the heart in the necessary sections [47].

Using echocardiography, we also assessed the systolic function of the left ventricle, which is determined by the contractility index. To calculate the contractility index, or contractility fraction, we used the following formula:

$$EF = (LVd - LVs) : LVd \times 100\%,$$

where:

EF is the ejection fraction,

LVd is the end-diastolic dimension of the left ventricle,

LVs is the end-systolic dimension of the left ventricle.

A decrease or increase in the systolic function of the left ventricle is called hypokinesis and hyperkinesis, respectively.

X-ray imaging was performed using the Vatel-1 digital X-ray complex with Digipax software. Images of the animal's heart were obtained in standard direct (dorsoventral or ventrodorsal) and right lateral projections using the generally accepted method.

Electrocardiography was performed using the Heart Screen 60G (VET) veterinary electrocardiograph. The animal was examined in the right lateral position or in another position convenient for cats, according to the generally accepted method.

All instrumental examinations were performed without the use of sedatives or anaesthetics in order to exclude their influence on heart rate, heart chamber size and cardiac contractility parameters.

2.3.4. Pathohistological research methods

Pathohistological studies of the myocardium of domestic cats were conducted

in the laboratory of the Kyiv Regional Oncology Dispensary using a standard method with haematoxylin-eosin staining of tissues.

During the study, five domestic cats that had died from cardiomyopathy underwent pathological autopsy. The above-mentioned animals were admitted to the veterinary clinic in urgent condition (pulmonary oedema due to acute cardiovascular failure) and died within the first three days. The age of the deceased animals ranged from 1 to 8 years.

The statistical processing of the research results was carried out using the Microsoft Excel 2010 and Statistica 10 programmes, obtaining an assessment of the reliability of the indicators ($p<0.001$; $p<0.01$; $p<0.05$) according to Student's criterion.

UNIT 3

DIAGNOSIS OF CARDIOMYOPATHY IN A DOMESTIC CAT

3.1. Symptoms of cardiomyopathy in domestic cats

During the study, we examined 16 clinically healthy cats (control group) and 58 sick cats (experimental group) who were diagnosed with cardiomyopathy during a comprehensive examination. Given that cardiomyopathy in domestic cats can be asymptomatic, this pathology was ruled out in the control group animals by performing echocardiography.

As a result of the analysis of the breed predisposition of cats to cardiomyopathy, we found that this disease is most often registered in domestic cats of the Scottish Fold (36.2%) and British Shorthair (29.3%) breeds (Table 3.1).

Table 3.1.

Analysis of cardiomyopathy in cats of different breeds, n=58

Breed	Number of animals	%
Scottish Fold	21	36,2
British Shorthair	17	29,3
European Shorthair	9	15,5
Maine Coon	3	5,2
Spynx	3	5,2
Persian	3	5,2
Siamese	1	1,7
Ocicat	1	1,7
Total	**58**	**100**

The age of the study cats ranged from 6 months to 16 years. During the analysis of data on age dynamics of animal morbidity, we found that domestic cats are susceptible to cardiomyopathy at any age. At the same time, the incidence of cardiomyopathy in cats aged 10 years and older was 2.5-3 times lower compared to

animals under 10 years of age (fig. 3.1). This may be due to the high mortality rate of animals with cardiomyopathy before they reach the age of 10. During our research, 22 animals died, which is 37.9% of the total number of cats in the study group. The average life expectancy of cats in the study group was 5.8 years.

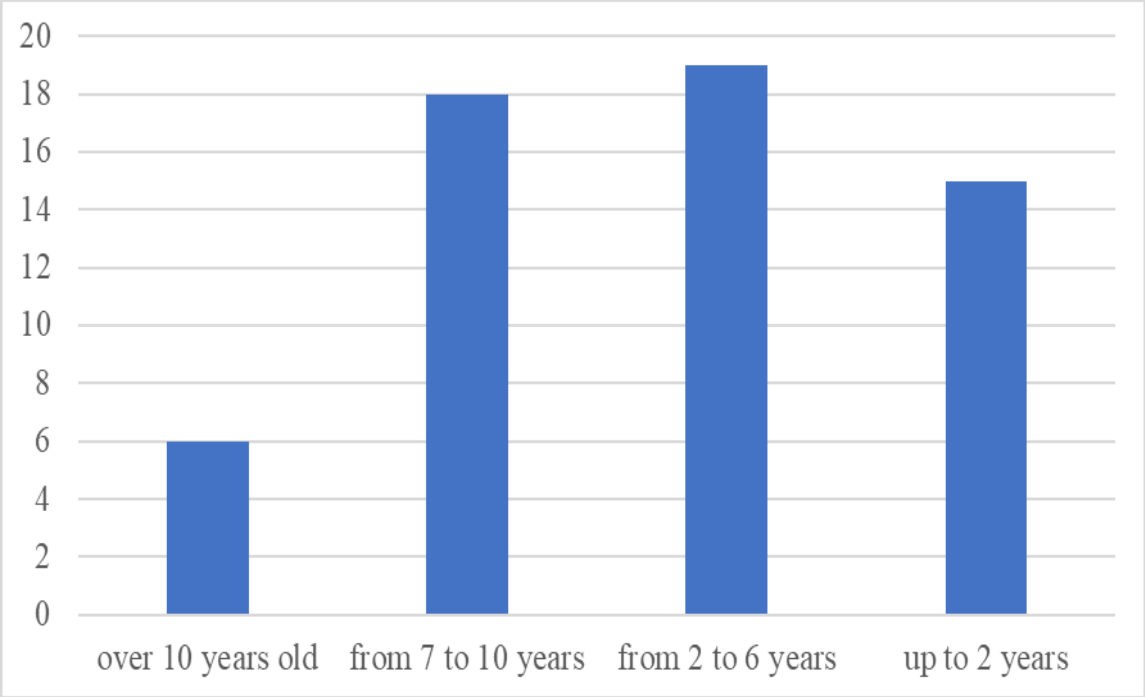

Fig. 3.1. Age-related dynamics of the incidence of cardiomyopathy in domestic cats, n=58

We also analysed the gender dependence of domestic cats with cardiomyopathy. As a result, it was found that males with cardiomyopathy were almost twice as numerous (65.5%) as females (34.5%).

During the collection of medical history, it was established that the vast majority of pet owners fed their pets with industrial concentrated dry and wet food, trying to adhere to the daily doses and terms recommended by the manufacturer. This type of feeding is acceptable, since the above-mentioned foods are balanced in all respects.

In two cases, cats were fed from the table, i.e., a natural diet. This type of feeding can lead to a lack of taurine in the diet, which is the cause of the development of dilated cardiomyopathy [185, 205]. Based on the results of echocardiography, these cats were diagnosed with dilated cardiomyopathy.

A large number of cases of cardiomyopathy in domestic cats remain undiagnosed due to the absence of pronounced clinical signs of this pathology [20].

All animals in the experimental group were examined by us according to the generally accepted scheme [47]. The physical examination of the animals included: auscultation of the chest, with determination of the heart rate per minute (HR, beats/min); counting the respiratory rate per minute (RR) and rectal thermometry (T, °C). The results of the medical history and clinical examination of the animals in the experimental group are presented in Tables 3.2 and 3.3.

Table 3.2

Clinical manifestations of cardiomyopathy in domestic cats

Clinical manifistation	Sick cats, n=58	
	Number of animals	%
Asymptomatic	30	51,7
Shortness of breath	19	32,8
Pulmonary oedema	15	25,9
Arterial tromboembolism	3	5,2

In most cases, shortness of breath remained an unnoticed symptom for pet owners. Owners explained their pets' shortness of breath as excitement or high ambient temperature. We also found that animals that were admitted to the veterinary clinic in urgent conditions (pulmonary oedema and arterial thromboembolism) had no history of cardiomyopathy symptoms.

Based on the results of our research, we found that the difference in the average heart rate (HR) and rectal temperature in cats with cardiomyopathy and clinically healthy cats is insignificant, as it did not reach the threshold of reliability. However, it should be noted that the HR in sick and clinically healthy cats was significantly higher than the reference values in the literature, by 51.7% and 69.2%, respectively. This is explained by the fact that even clinically healthy cats visiting the doctor show physiological tachycardia, which is associated with increased sympathetic tone in animals experiencing anxiety and stress during a visit to the clinic.

We also found a significant increase in respiratory rate by 16.2% per minute

in sick animals ($p<0.05$) compared to clinically healthy animals. This may indicate the onset of dyspnoea in asymptomatic cats with cardiomyopathy.

Table 3.3

Results of clinical examination of clinically healthy cats and cats with cardiomyopathy, M±m, n=58

Indexes	Reference values for normal [47]	Clinically healthy cats		Cats with cardiomyopathy	
		Cat	M±m	Cat	M±m
Heart rate, bpm	110 -130	176 - 224	203,6 ± 16,5	156 - 234	182,4 ± 20,6
Respiration rate per minute	18 - 20	18 - 24	21,0 ± 1,20	22 - 26	24,4 ± 1,3*
Body temperature, °C	38 -39,5	38 -38,7	38,5 ± 0,2	37,9 - 38,9	38,4 ± 0,3

Note: * $p<0,05$ compared to clinically healthy cats

Thus, based on the results of the studies, it was established that the vast majority of domestic cats with cardiomyopathy have an asymptomatic course of the disease.

3.2. Blood tests for domestic cats with cardiomyopathy

The results of common blood tests (red blood cell and white blood cell counts, haemoglobin levels, leukogram) in clinically healthy cats and cats with cardiomyopathy, mainly hypertrophic cardiomyopathy, are presented in Table 3.4.

It has been established that in the blood of cats with cardiomyopathy, the number of erythrocytes and leukocytes, haemoglobin content and leukogram indicators do not differ statistically from clinically healthy animals.

Therefore, the absence of differences in the results of complete blood counts in clinically healthy cats and cats with cardiomyopathy indicates that such tests are not effective for confirming the diagnosis of cardiomyopathy in domestic cats. However, conducting a general blood test of a domestic cat with cardiomyopathy is necessary, as its results allow to exclude the inflammatory process and the presence of anaemia in animals, which may be an aetiological factor of the secondary form of cardiomyopathy [46, 114, 128].

Table 3.4

Complete blood count results for healthy cats and cats with hypertrophic cardiomyopathy, M±m; n=5

Indexes	Reference values for normal [17]	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
RBC, T/l	5,5-10,7	7,32 10,7	8,82±0,90	7,89 11,10	10,07±0,90
Haemoglobin, g/l	100,0-150,0	132,0 142,0	138,20 ±2,20	122,0 154,0	137,2 ±10,1
WBC, G/lл	6,40-13,50	5,6-10,0	8,32±1,49	6,20 13,10	8,48±2,01
Leucograma:					
Eosinophils, %	0-6,0	4,0-18,0	8,20±4,24	4,0-20,0	6,80±5,28
Band Neutrophils, %	1,0-6,0	3-12	5,60±2,72	1,0-8,0	3,60±2,32
Segmented Neutrophils, %	43,0-68,0	20-65	43,60 ±14,48	32,0-79,0	53,60 ±14,08
Lymphocytes, %	25,0-50,0	30-61	39,0±9,2	11,0-60,0	33,60 ±12,32
Monocytes, %	0-6,0	2-9	4,40±2,08	1,0-4,0	2,40±0,88
Basophils, %	0-1,0	0	0	0	0

At the same time, the 14.2% increase in the number of erythrocytes in the blood of cats with cardiomyopathy compared to clinically healthy cats is a sign of hypoxia, which can be observed during heart disease [17].

The results of studies of biochemical indicators of blood serum in clinically healthy cats and cats with cardiomyopathy who had no symptoms of the disease are presented in Table 3.5.

The values we obtained for AST and ALT activity, creatinine, urea and glucose concentrations, as well as total protein content in the blood serum of clinically healthy cats are within the acceptable range, which is consistent with reference values and literature data.

Table 3.5

Biochemical indicators of domestic cat blood serum in hypertrophic cardiomyopathy, M±m, n=5

Indexes	Reference values for normal [47]	Clinically healthy cats		Cats with cardiomyopathy	
		Queen	M±m	Tom	M±m
AST, U/l	10-48	15,6-69,5	33,08±12,14	9,2-40,6	27,6±8,67
ALT, U/l	10-49	38-76	58,16±11,47	36,2-64	54,24±8,91
Cretinine, μmol/l	25-140	70,6-103	84,8±12,16	70-122	104±17,6
Urea, mmol/l	2,5-10	5,7-9,9	7,5±1,33	4,8-9	6,76±1,4
Common protein, g/l	56-80	60-84	69,26±8,59	60-87	71,24±7,41
Glucose, mmol/lл	3,5-6,2	3,8-5,07	4,71±0,37	5,1-8,43	6,43±1,15*

Note: * $p<0,05$ compared to clinically healthy cats

At the same time, in the blood serum of domestic cats with cardiomyopathy, we found a decrease in AST activity by 16.6%, urea concentration by 9.9%, an increase in creatinine concentration by 22.2% with a significant increase in glucose concentration by 36.5% ($p<0.05$) compared to clinically healthy animals.

An increase in serum glucose concentration in domestic cats with cardiomyopathy may indicate the presence of transient hyperglycaemia. This phenomenon can also be observed in irritated and frightened cats, or in cats experiencing stress during clinical examination. In such cases, the concentration of glucose in the cat's blood serum can reach 6.25-9.37 mmol/l [120].

The data obtained allow us to conclude that the biochemical indicators of domestic cat blood serum in cardiomyopathy, as well as the morphological indicators of their blood and haemoglobin content described earlier, are not indicative for confirming the diagnosis of cardiomyopathy. That is, their determination in domestic cats for this purpose is not effective.

In mammals with uraemia (hyperuremia), which can be observed in chronic kidney disease during acute or chronic renal failure, concentric myocardial hypertrophy may develop, which can lead to the development of a secondary hypertrophic form of cardiomyopathy [2, 4, 79, 114, 128].

We determined the content of creatinine and urea in the blood serum of cats aged 4 to 16 years with cardiomyopathy, most of which had a symptom complex of pulmonary oedema, indicating a more severe course of heart disease. During ultrasound examination of the kidneys in the study animals, moderate and severe nephrotic-nephritic changes were also noted.

The results of the study show that the concentration of creatinine and urea in the blood serum of domestic cats with cardiomyopathy is 2.2 and 2.5 times higher, respectively, compared to clinically healthy animals (Table 3.6). Our data are reliable and confirm the presence of hypertrophic cardiomyopathy in domestic cats with hyperuremia in 8 animals (13.8%) out of 58 studied.

Table 3.6

Concentration of creatinine and urea in the blood serum of domestic cats with hypertrophic cardiomyopathy, M±m, n=5

Indexes	Reference values for normal [47]	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
Creatinine, µmol/l	25-140	70,6 103,0	84,8±12,16	146-229	188,20±27,4*
Urea, mmol/l	2,5-10,0	5,7-9,9	7,50±1,33	11,6-26,8	18,80±2,91**

Note: * p<0,001, ** p<0,01 compared to clinically healthy cats

It should be noted that hyperazotemia, as a factor of autointoxication in chronic kidney disease, is an important aetiological factor in hypertrophic changes in the myocardium in domestic cats, i.e. a factor that causes the development of secondary hypertrophic cardiomyopathy in animals of this species.

3.3. Study of cardiac troponin I concentration in the blood of domestic cats with cardiomyopathy

Currently, the most effective biomarker for myocardial damage is troponin, which consists of three components: troponin T, troponin I, and troponin C. Troponin I specifically reflects damage to the heart muscle. In the past, biomarkers such as creatine phosphokinase, lactate dehydrogenase, and aspartate aminotransferase were widely used for diagnostic purposes. However, due to the presence of a large number

of isoenzymes of these enzymes in skeletal muscles, these biomarkers no longer have diagnostic value [84, 100, 115, 175].

During our work, we determined the concentration of cardiac troponin I in the blood serum of clinically healthy cats and cats with cardiomyopathy, mainly hypertrophic cardiomyopathy. The results of the studies are presented in Table 3.7.

Our research showed a 12.5-fold increase in cardiac troponin I concentration in the blood serum of cats with cardiomyopathy compared to clinically healthy cats. These data indicate significant damage to cardiomyocytes in animals with cardiomyopathy, which is characteristic of both hypertrophic and other forms of cardiomyopathy.

Table 3.7

Concentration of cardiac troponin I in the blood serum of domestic cats with hypertrophic cardiomyopathy, M±m, n=5

Indexes	Reference values for normal [172]	Clinically healthy cats		Cats with cardiomyopathy	
		Queen	M±m	Tom	M±m
Troponin I, ng/ml	0,00-0,05	0,00-0,04	0,012±0,01	0,051- 0,28	0,15±0,06*

Note: * p<0,001 compared to clinically healthy cats

It should be noted that during the course of our research, we identified individual patients (n=3) who had a cardiac troponin I concentration of 0.022±0.012 ng/ml in their blood serum due to cardiomyopathy. This is only 1.83 times higher than in clinically healthy animals and is within the normal reference range. Taking this into account, we concluded that the degree of cardiomyocyte damage is insignificant, but in cats with cardiomyopathy, echocardiography diagnosed hypertrophic cardiomyopathy. This form of cardiomyopathy in these animals should be considered secondary, since in primary hypertrophic cardiomyopathy, which is currently considered genetically determined, myocardial damage is permanent [175].

Also, in the course of our research, we found that the serum of one British Shorthair cat had an extremely high (up to 1.64 ng/ml) concentration of cardiac troponin I, which is almost 137 times higher than in clinically healthy animals. Today, it has been proven that the concentration of cardiac troponin increases in

relation to the degree of cardiomyocyte damage. The higher this indicator, the more extensive and severe the damage to cardiomyocytes [88, 100]. High concentrations of troponin I in the blood serum of animals suggest myocarditis as one of the causes of cardiomyocyte damage [40, 84, 107, 211]. This gives us reason to suspect myocarditis in cats with extremely high levels of cardiac troponin in their blood serum.

Thus, the concentration of cardiac troponin I in the blood serum of a domestic cat can be used to assess the degree of myocardial damage in an animal with cardiomyopathy.

3.4. X-ray examination of a domestic cat for cardiomyopathy

In the course of our work, we conducted X-ray examinations of the chest of clinically healthy cats and cats with cardiomyopathy.

During the analysis of the X-rays, we determined the topographical features of the heart and large vessels, assessed the condition of the respiratory tract and lungs of the animals using indicators such as the Buchanan coefficient (Viskapap) and cardiotoracic index, assessed the silhouette of the heart and determined the presence of its enlargement in cardiomyopathy in sick cats (Table 3.8).

The data we obtained indicate that the average Buchanan coefficient in cats with cardiomyopathy is 20% higher, and the cardiothoracic index is 38% higher compared to clinically healthy animals, but these data are not reliable.

Table 3.8

Buchanan's coefficient and cardiothoracal index values in healthy cats and cats with cardiomyopathy, M±m, n=5

Indexes	Reference values for normal	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
Buchanan's coefficient	9,0-10,0	8,09-10,11	8,87±0,53	8,60-14,0	10,64±2,29
Cardiothoracal index	0,55	0,50-0,62	0,55±0,04	0,56-1,0	0,76±0,19

Thus, the assessment of the heart silhouette size based on data obtained by chest X-ray examination to confirm the diagnosis of cardiomyopathy in domestic cats

is not very informative in these animals.

We assessed the condition of the lungs and respiratory tract based on the presence of shadows in the lungs, signs of hydrothorax, and the position of the trachea in the animals. In cats with cardiomyopathy, we detected the following signs using X-ray examination: tracheal elevation, congestive phenomena in the lungs, pulmonary oedema, and atrial enlargement (Fig. 3.2).

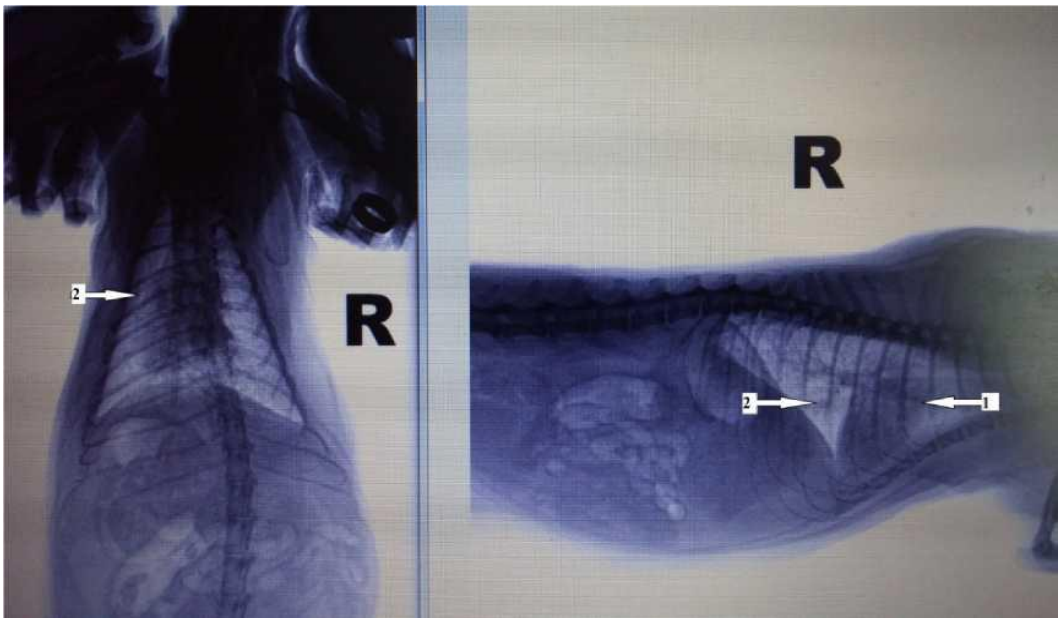

Fig. 3.2. X-ray of Newton, an 8-year-old British Shorthair cat. Diagnosis: hypertrophic cardiomyopathy. Signs of atrial enlargement (1), pulmonary oedema and venous congestion in the lung tissue (2) are visible.

Thus, X-ray examination data allow assessing the severity of cardiomyopathy in domestic cats. For example, tracheal elevation in cats with cardiomyopathy provokes coughing in these animals. Congestion in the lungs and enlargement of the atria lead to shortness of breath, which is a manifestation of respiratory failure in these animals. This, in turn, provokes the development of pulmonary oedema in sick cats, which is a life-threatening condition and can lead to the death of the animal.

3.5. Electrocardiographic studies of domestic cats with cardiomyopathy

Electrocardiographic studies in cats have certain peculiarities compared to dogs and other mammals. Normally, electrocardiographic complexes in cats are low-voltage, which in some cases becomes a serious problem. Often, electrocardiogram data can be distorted by various noises caused by electrical interference, the animal's

purring, trembling, anxiety, and stress experienced by cats during the study. Abnormal data during electrocardiography, caused by the stressful state of the animals, can also be obtained in clinically healthy cats [40, 75].

Nevertheless, in the course of our work, we conducted an electrocardiographic study of clinically healthy cats and cats with cardiomyopathy, during which we obtained the results presented in Table 3.9.

The results of the studies show an increase in the average heart rate by 67.7% in clinically healthy cats and by 70% in cats with cardiomyopathy compared to the reference values in animals of this species. That is, in cats of both groups, we observed the manifestation of sinus tachycardia, which can be explained by the anxiety of the animals and their feeling of stress during electrocardiographic studies. In the vast majority of cats in both groups, a regular sinus rhythm was recorded during the studies, and only one cat with cardiomyopathy had an irregular sinus rhythm. Two cats with cardiomyopathy had signs of myocardial ischaemia, and two others had signs of delayed myocardial depolarisation.

Based on electrocardiography data, we determined that the duration of electrocardiographic complexes in healthy cats and cats with cardiomyopathy did not differ significantly and did not reach the significance level of $p<0.05$. The duration of electrocardiographic complexes in cats of both groups is within the acceptable range, which is consistent with the reference normative values for animals of this species [17, 47].

Table 3.9

Electrocardiogram readings for a domestic cat with cardiomyopathy, M±m, n=5

Indexes	**Reference values for normal [17]**	**Clinically healthy cats**		**Cats with cardiomyopathy**	
		Lim	**M±m**	**Lim**	**M±m**
Heart rate, bpm	120±10	176-224	200±17	140-280	204±45
P wave duration, sec	0,04	0,04-0,05	0,042±0,003	0,03-0,04	0,038±0,003

Continuation of Table 3.9

P wave voltage, mV	0,20	0,01-0,15	0,11±0,02	0,10-0,15	0,13±0,02
PQ interval duration, sec	0,07±0,02	0,05-0,08	0,066±0,009	0,06-0,09	0,070±0,007
QRS interval duration, sec	0,04	0,04-0,05	0,040±0,003	0,02-0,05	0,04±0,007
R wave voltage, mV	0,90	0,30-0,60	0,38±0,10	0,30-1,10	0,62±0,30

During the analysis of electrocardiograms, we observed a decrease in the average voltage of P waves by 45% in clinically healthy cats and by 35% in cats with cardiomyopathy compared to the reference standards. We also noted a decrease in the average voltage of the K wave in clinically healthy cats and a decrease in this indicator in cats with cardiomyopathy by 31.1% compared to the reference values for cats within the normal range (Fig. 3.3).

It should be noted that in one cat with cardiomyopathy, an increase in the voltage of the K wave to 1.1 mV was observed, which is 22.2% higher than the reference standard. The increase in the voltage of the K wave on the electrocardiogram of this cat indicates increased myocardial excitability, which can be observed in sinus tachycardia and is a physiological response to stress in an excited animal. This animal had the highest heart rate, namely 280 beats per minute.

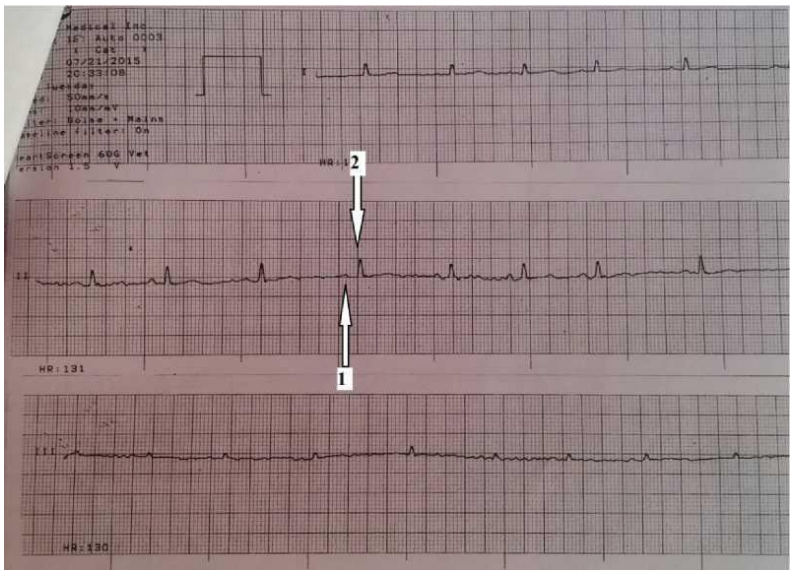

Fig. 3.3. Electrocardiogram of Newton, a British Shorthair cat, 8 years old: sinus arrhythmia, decreased voltage of P (1) and K (2) waves.

Thus, the electrocardiography method does not provide reliable data for the diagnostic confirmation of cardiomyopathy in domestic cats.

3.6. Echocardiographic examination of a domestic cat for cardiomyopathy

Ultrasound examination of the heart (echocardiography) allows visualisation of the anatomical structures of this organ, identification of a number of specific sonographic signs of cardiomyopathy, detection of haemodynamic disturbances, and determination of the functional state of the heart in domestic cats.

Ultrasound examination is based on the ability to absorb or reflect ultrasound waves at the boundary between tissues with different acoustic densities (echogenicity). Accordingly, changes in the echogenicity of the examined tissue can be used to judge the presence of fibrotic changes in the myocardium of a domestic cat. For example, increased echogenicity (hyperéchogenicity) of the myocardium in animals is a sign of fibrotic changes in the myocardium. The characterisation of tissue by echogenicity is a subjective assessment of the condition of the examined structure, as it has no quantitative expression. However, the doctor's understanding of the essence and principle of sonography allows them to identify and assess the state of the pathological process, i.e. the development of fibrotic changes, in the examined tissue [37, 47, 73, 129].

We have established that in clinically healthy cats, during an ultrasound examination of the heart, the chambers of the heart filled with blood appear as anechoic structures. The myocardium of the interventricular septum and heart walls also appears as distinctly hypoechoic, almost anechoic, formations.

The border between the heart chambers and the endocardium, epicardium and pericardium, valves, appears as thin hyperechoic lines, which makes it possible to visualise the anatomical shape and structures of the heart (Fig. 3.4 and Fig. 3.5).

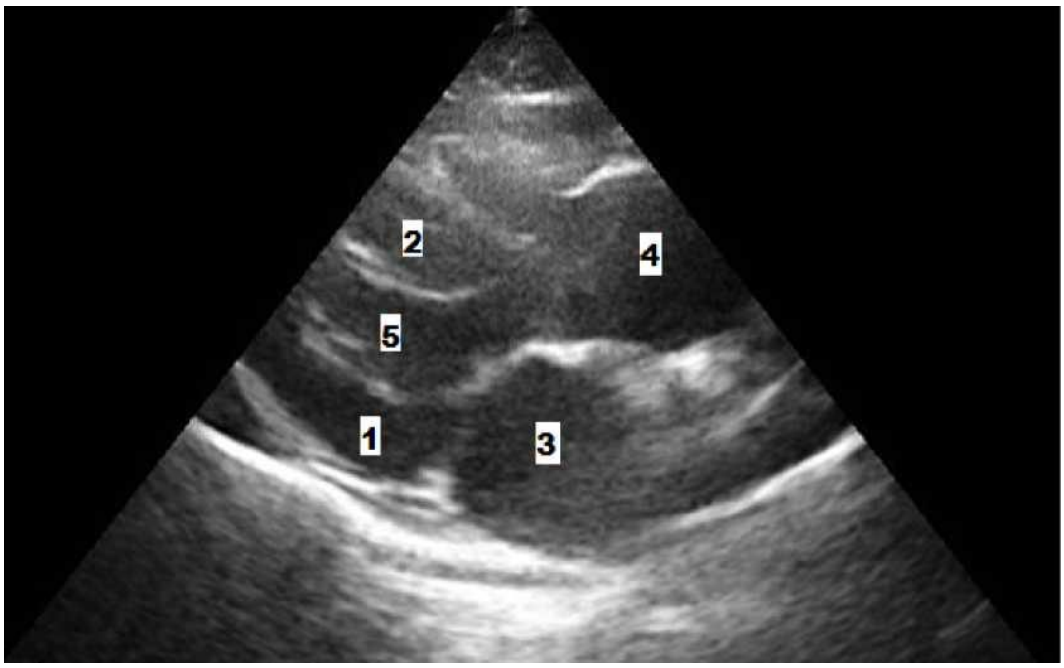

Fig. 3.4. Ultrasonogram of the heart of a British Shorthair cat named Rocky, aged 4 years. B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: anechoic wall of the left ventricle myocardium (1) and interventricular septum (2), left (3) and right (4) atrial cavities, left ventricular cavity (5) within normal limits.

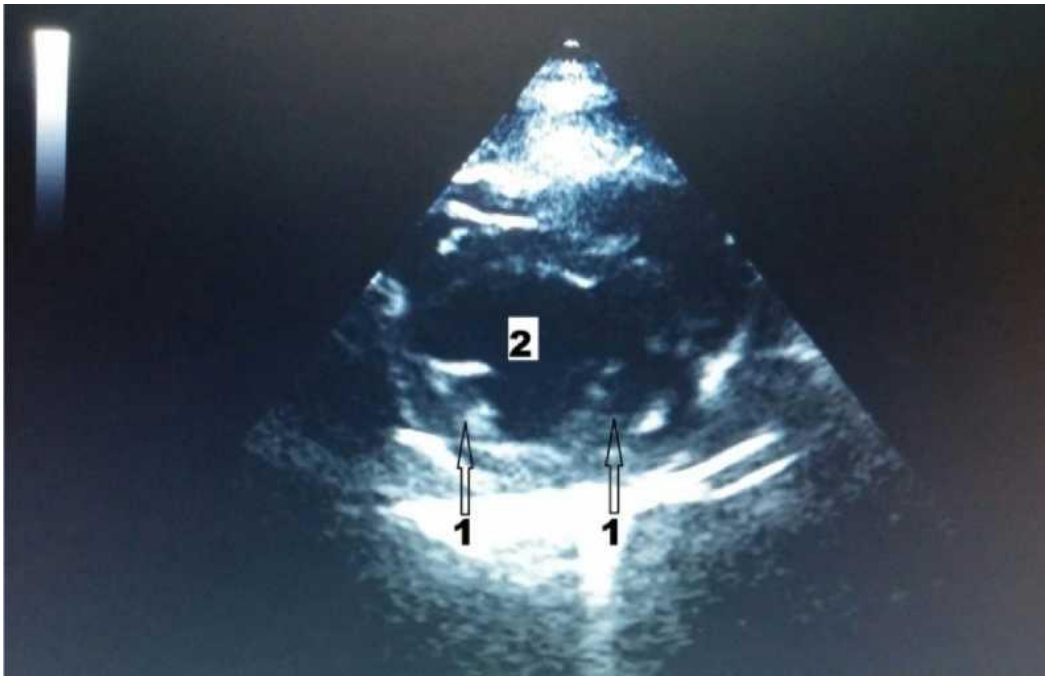

Fig. 3.5. Ultrasonogram of the heart of a Scottish Fold cat named Oliva, aged 1 year. B-mode from the right parasternal access along the short axis of the left ventricle at the level of the papillary muscles. Visualised: papillary muscles of the left ventricle (1) and left ventricular cavity (2) within normal limits.

A particular form of cardiomyopathy in a domestic cat can be diagnosed based on a number of specific sonographic signs and changes in the size of the anatomical structures of the heart, which we took into account when performing echocardiography of cats with the specified pathology (Fig. 3.6).

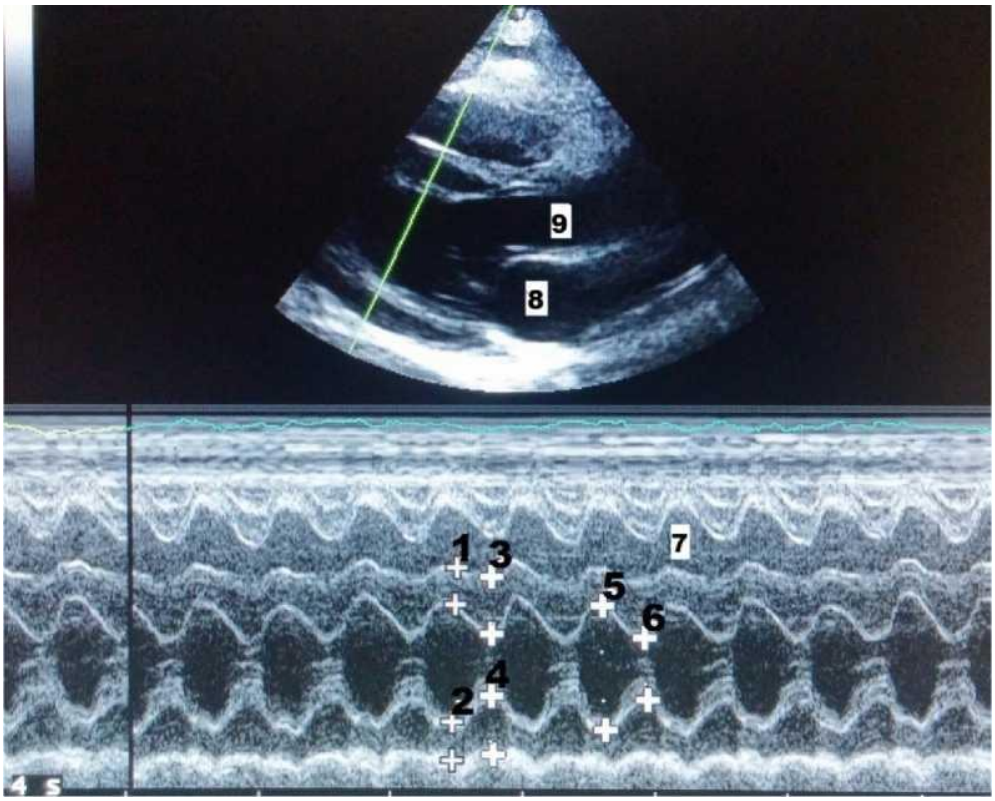

Fig. 3.6. Ultrasonogram of the heart of a Scottish Fold cat named Oliva, aged 1 year. M-mode from the right parasternal access along the long axis of the left ventricle at the level of the papillary muscles. Visualised: interventricular septal thickness in diastole (1), left ventricular wall thickness in diastole (2), interventricular septal thickness in systole(3), left ventricular wall thickness in systole (4), the end-diastolic size of the left ventricle (5), the end-systolic size of the left ventricle (6), the right ventricular cavity (7), the left atrial cavity (8), the aortic orifice - the outflow tract of the left ventricle (9).

We determined haemodynamic disturbances in the heart using Doppler ultrasound (Doppler echocardiography). Doppler echocardiography allows visualisation of blood flow disturbances in the heart chambers and large vessels, which can confirm the presence and degree of heart valve insufficiency.

For example, the application of this methodological approach using Doppler echocardiography in the form of colour Doppler mapping in B-mode from the right parasternal access along the long axis of the left ventricle during ultrasonography of a

cat's heart allowed us to establish severe mitral valve insufficiency (Fig. 3.7).

Thus, during an ultrasound examination of a cat's heart using colour Doppler mapping, atrioventricular valve insufficiency will appear as a colour (orange-yellowish) flow directed from the ventricle cavity to the atrium cavity during systole. The degree of atrioventricular valve regurgitation can be assessed by the size and intensity of the coloured flow. Therefore, the more severe the bicuspid valve insufficiency, the greater the enlargement of the left atrium and the more likely the development of congestion in the pulmonary veins, which is a risk factor for the development of pulmonary oedema in cats with cardiomyopathy.

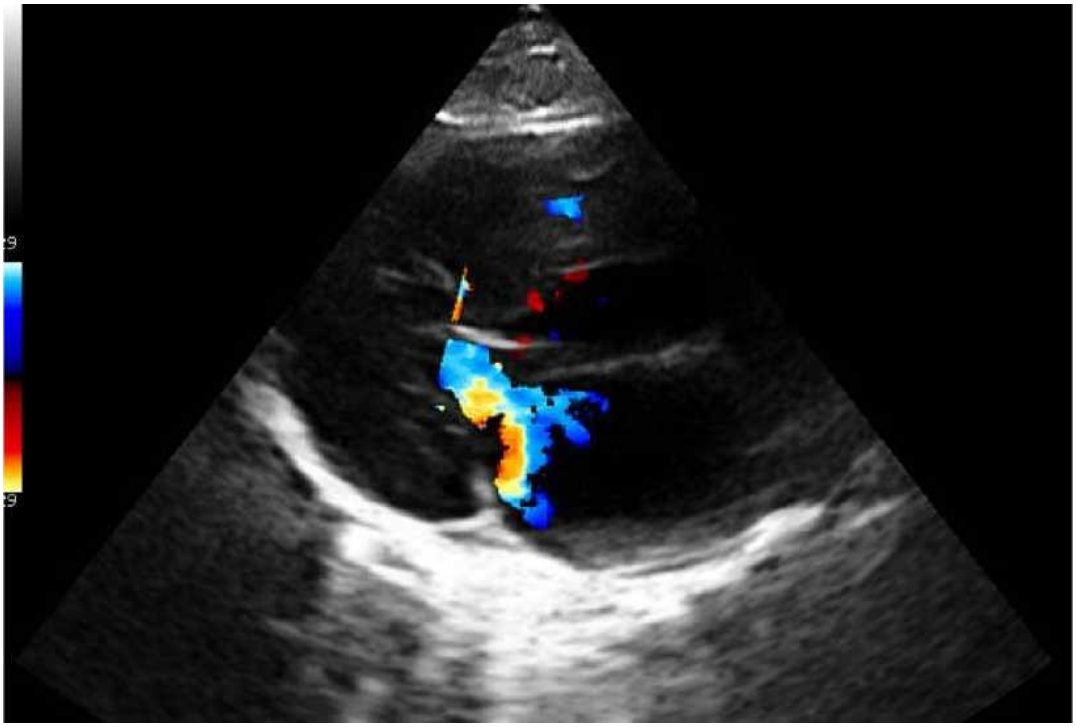

Fig. 3.7. Ultrasonogram of the heart of a Persian cat named Bronya, aged 10 years. B-mode from the right parasternal access along the long axis of the left ventricle using Doppler echocardiography in the colour Doppler mapping modification: severe mitral valve insufficiency is visualised.

3.6.1. Echocardiographic changes in domestic cats with hypertrophic cardiomyopathy

In the course of our work, we conducted ultrasound examinations of the hearts of clinically healthy cats and cats with hypertrophic cardiomyopathy (n = 5), aged 1 to 14 years.

The results of our research are presented in Table 3.10.

Based on the results of ultrasound examinations of the heart of domestic cats, we determined that the main parameters of the anatomical structures of the heart in clinically healthy animals are within the acceptable range, which is consistent with the reference values in the literature [17, 47, 129, 165].

Table. 3.10

Echocardiography results for a domestic cat with hypertrophic cardiomyopathy, M±m, n=5

Indexes	**Reference values for normal [17]**	**Clinically healthy cats**		**Cats with cardiomyopathy**	
		Lim	**M±m**	**Lim**	**M±m**
Left ventricular wall thickness in diastole, mm	2,2-4,4	3,8-3,9	3,88±0,03	6,2-6,8	6,46±0,23*
Interventricular septum thickness in diastole, mm	2,2-4,0	3,3-3,9	3,60±0,17	5,5-6,7	6,28±0,38*
Left ventricular end-systolic dimension, mm	5,2-10,8	6,0-8,0	6,7±0,7	3,7-6,7	5,6±1,1
Left ventricular end-diastolic dimension, mm	12,0-19,8	13,0-16,6	14,2±1,1	10,7-16,4	13,5±2,1
Left atrial cavity size, mm	9,3-15,1	13,6-14,8	14,3±0,4	13,9-19,1	16,74±1,85**
Contractility index, %	39,0-61,0%	50,3-55,1	53,0±1,3	48,9-65,4	58,7±4,1%

Note: * p<0,001; **p<0,05 compared to clinically healthy cats

The results of our studies show that in cats with hypertrophic cardiomyopathy, there is a significant (p<0.001) thickening of the left ventricular wall in diastole by 65.6% and the interventricular septum in diastole by 72.5%, compared to clinically healthy animals.

Therefore, thickening of the left ventricular wall and interventricular septum in diastole can be considered with high confidence as a sign of hypertrophic

cardiomyopathy in domestic cats.

At the same time, the size of the left atrial cavity in cats with hypertrophic cardiomyopathy was 17.1% significantly larger ($p<0.05$) compared to clinically healthy animals.

However, not all cats with hypertrophic cardiomyopathy have enlargement of the left atrium. Thus, in the group of cats with hypertensive cardiomyopathy, one animal did not show enlargement of the left atrium, and in two cats it was mild (10.85%) compared to clinically healthy animals. In contrast, two cats had a fairly pronounced enlargement of the left atrium (by 33.2%) compared to clinically healthy animals. All sick cats had varying degrees of bicuspid valve insufficiency (regurgitation). Based on our results, the possible factors that determine the enlargement of the left atrial chamber in domestic cats, in our opinion, are the presence and degree of mitral valve regurgitation, as well as the manifestation of compensatory mechanisms of disturbed haemodynamics in the body of cats with hypertrophic cardiomyopathy.

In addition, echocardiography results show that domestic cats with hypertrophic cardiomyopathy have a 16.42% reduction in left ventricular cavity size during systole and a 5.2% reduction during diastole compared to clinically healthy animals. A reduction in the left ventricular cavity leads to a decrease in stroke and minute volume of the heart and, as a compensatory mechanism, is accompanied by an increase in cardiac contractility. We have established that the cardiac contractility index in cats with hypertrophic cardiomyopathy is increased by 10.1% compared to clinically healthy animals.

In one animal with hypertrophic cardiomyopathy, hypertrophic changes in the papillary muscles of the left ventricle and the presence of free fluid in the pericardial cavity with a volume of 15 ml were found. The amount of fluid in the pericardial cavity is directly proportional to the volume of the distance from the epicardium to the pericardium. In this case, the distance from the epicardium to the pericardium, which is 1 mm, is approximately equal to a volume of 1 ml of fluid [47].

Myocardial thickening in hypertrophic cardiomyopathy in domestic cats may

be asymmetrical, i.e., there may be thickening of individual parts of the interventricular septum, left ventricular wall, or left ventricular papillary muscles (Table 3.11).

Table 3.11

Echocardiography results for a domestic cat with asymmetric hypertrophic cardiomyopathy, M±m, n=5

Indexes	Reference values for normal [17]	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
Left ventricular wall thickness in diastole, mm	2,2-4,4	3,8-3,9	3,88±0,03	4,7-6,5	5,52±0,39*
Interventricular septum thickness in diastole, mm	2,2-4,0	3,3-3,9	3,64±0,20	4,9-6,1	5,52±0,38*
Basal interventricular septum thickness in diastole, mm	2,2-4,0	3,3-3,9	3,64±0,20	6,4-7,4	6,80±0,27*
Contractility index, %	39,0-61,0	50,3-55,1	53,02±1,3	53,7-60,1	57,28±1,82**

Note: * p<0,001, ** p<0,05 compared to clinically healthy cats

Based on the results of our research, we have established that cats with asymmetric hypertrophic cardiomyopathy show a significant (p<0.001) thickening of the left ventricular wall in diastole by 41.54% and of the interventricular septum in diastole by 51.65% compared to clinically healthy cats. It should be noted that such myocardial thickening in cats with asymmetric hypertrophic cardiomyopathy is less pronounced compared to animals with symmetric hypertrophic cardiomyopathy.

At the same time, cats with asymmetric hypertrophic cardiomyopathy also showed a significant thickening of the basal part of the interventricular septum in diastole by 86.8% (p<0.001) compared to clinically healthy animals.

It should be noted that thickening of the interventricular septum in domestic cats leads to obstruction of the left ventricular outflow tract, which is called obstructive hypertrophic cardiomyopathy. The contractility index we established in

cats with obstructive hypertrophic cardiomyopathy is 8.0% significantly higher ($p<0.05$) compared to clinically healthy cats.

In one cat with obstructive hypertrophic cardiomyopathy, we also found hypertrophic changes in the papillary muscles of the left ventricle, and in three cats, we found mitral valve insufficiency. Another cat with obstructive hypertrophic cardiomyopathy was found to have a small amount of pericardial effusion. The volume of pericardial effusion was 14 ml.

In cats with hypertrophic cardiomyopathy, we also noted an increase in myocardial echogenicity, indicating the development of fibrotic changes in the heart muscle [92].

Thus, our results allow us to conclude that hypertrophic cardiomyopathy in domestic cats can be reliably diagnosed based on a number of specific signs observed during ultrasound examination of the heart. These signs include: significant thickening ($p<0.001$), i.e. hypertrophic changes, of the left ventricular wall and interventricular septum in diastole (Fig. 3.8.) and/or asymmetric thickening ($p<0.001$) of the basal part of the interventricular septum (Fig. 3.9.); significant enlargement ($p<0.05$), i.e. dilation changes, of the left atrium (Fig. 3.9.), which is observed in most cases; the dimensions of the left ventricle cavity remain normal or may be reduced (Fig. 3.8.); hypertrophic changes in the papillary muscles of the left ventricle (Fig. 3.10.), which may not be observed in all cases; the presence of fluid in the pericardial cavity, i.e. pericardial effusion (Fig. 3.9 and Fig. 3.11), which is visualised in some cases; mitral valve insufficiency (Fig. 3.7), which is visualised in most cases; increased contractility index, i.e. myocardial hyperkinesis; increased echogenicity of the left ventricular wall and interventricular septum, which is a sign of fibrotic changes (Fig. 3.12).

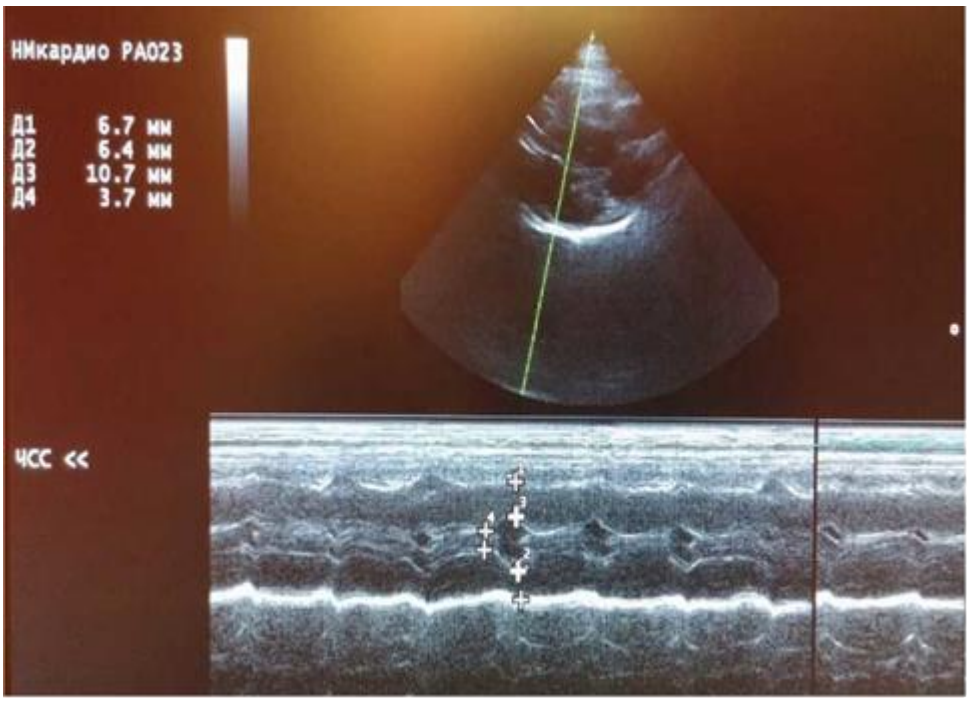

Fig. 3.8. Ultrasonogram of the heart of a British Shorthair cat named Umka, aged 8 years. M-mode from the right parasternal access along the long axis of the left ventricle at the level of the papillary muscles. Visualised: thickening of the left ventricular wall in diastole to 6.4 mm and the interventricular septum in diastole to 6.7 mm, reduction to 10.7 mm of the end-diastolic and to 3.7 mm of the end-systolic dimensions of the left ventricle.

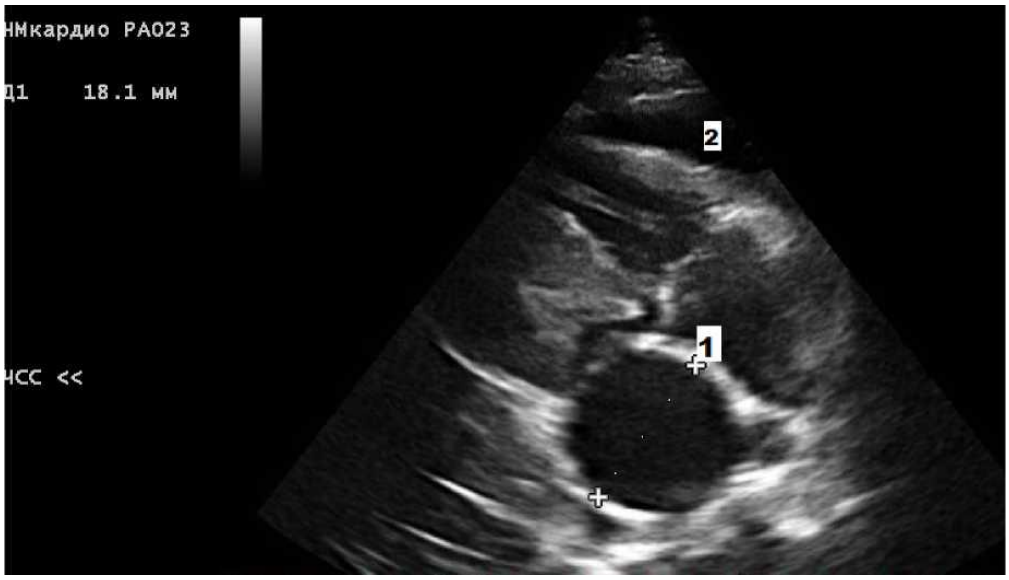

Fig. 3.9. Ultrasonogram of the heart of a Scottish Fold cat named Alice, aged 7 years. B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: enlargement of the left atrium chamber to 18.1 mm (1), presence of pericardial effusion(2).

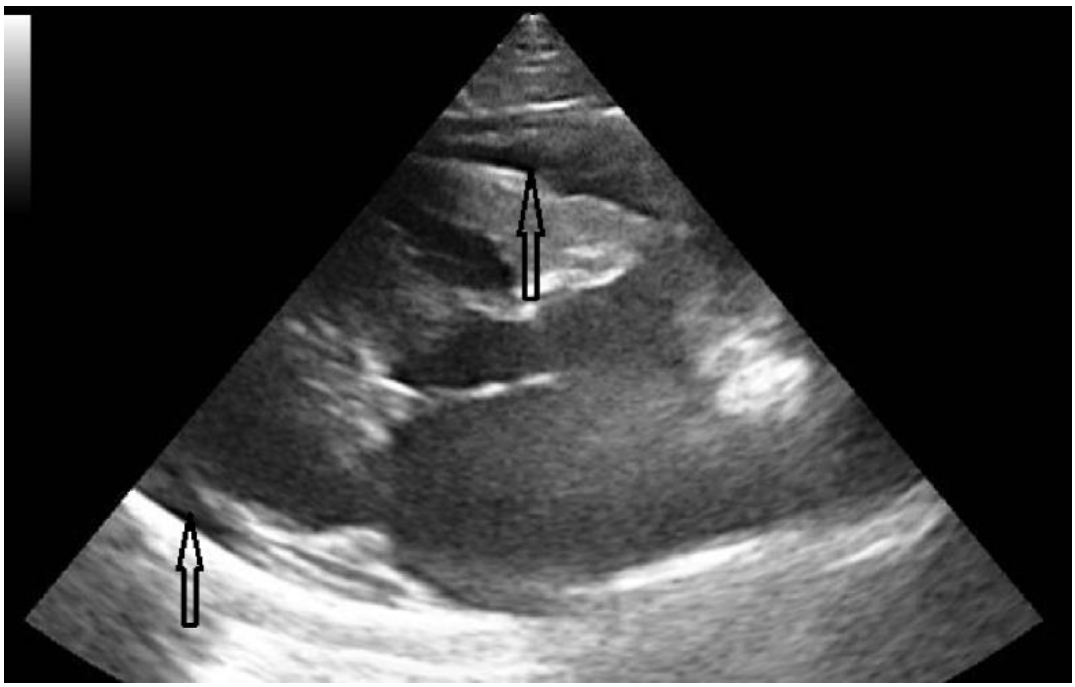

Fig. 3.10. Ultrasonogram of the heart of a British Shorthair cat named Bambaleo, aged 2 years. B-mode from the right parasternal access along the short axis of the left ventricle at the level of the papillary muscles. Visualised: hypertrophic changes and signs of fibrosis of the papillary muscles of the left ventricle (1) and a reduction in the left ventricular cavity (2).

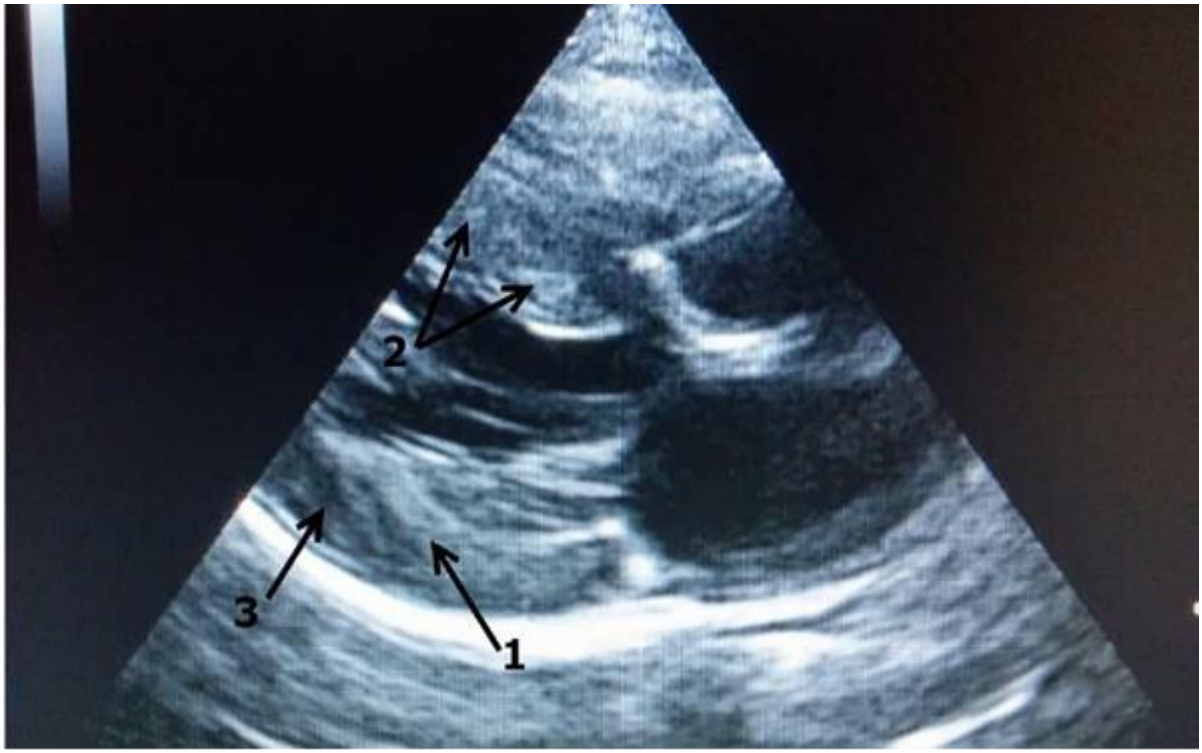

Fig. 3.11. Echocardiogram of a short-haired cat named “Pupsik”; parasternally accessed via the long axis; pericardial effusion (indicated by arrows)

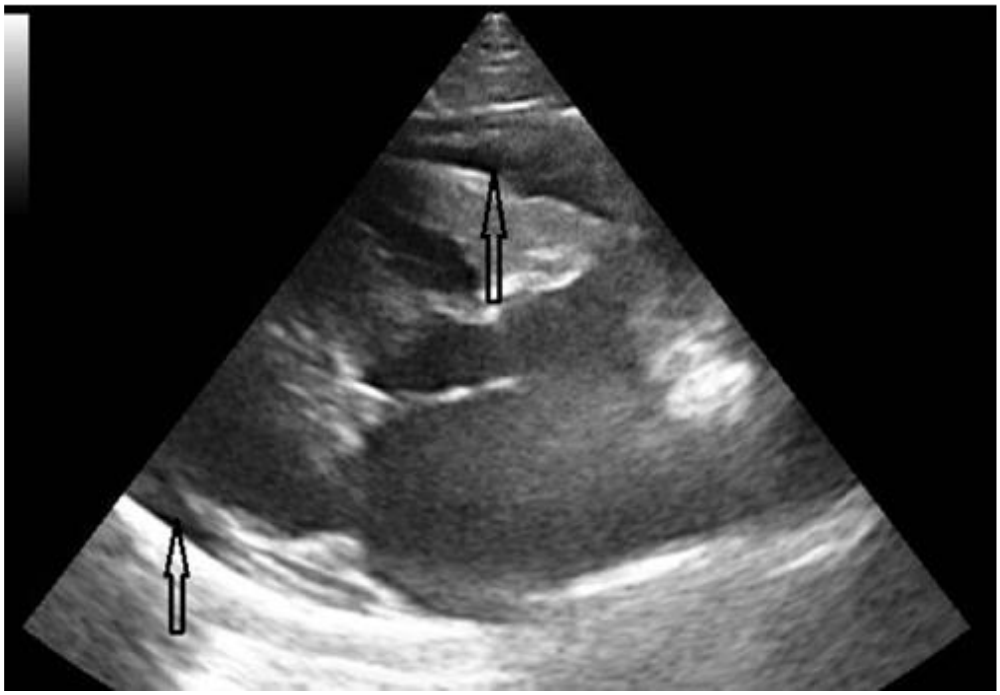

Fig. 3.12. Ultrasonogram of the heart of a British Shorthair cat named Margo, aged 11 years. B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: hyperechoic endomyocardial patch in the form of a strip of left ventricular wall myocardium (1) and focal hyperechoic patches in the interventricular septum myocardium (2), which is a sign of fibrotic changes, and an area of the myocardium with low echogenicity, without fibrotic changes (3).

3.6.2. Echocardiographic changes in domestic cats with restrictive cardiomyopathy

The results of the studies show that patients with restrictive cardiomyopathy in cats have a significant ($p<0.001$) thickening of the left ventricular wall in diastole by 60.5%, and the interventricular septum in diastole by 47.8% ($p<0.01$), compared to clinically healthy animals (Table 3.12).

Table 3.12

Echocardiography results in a domestic cat with restrictive cardiomyopathy, M±m, n=5

Indexes	Reference values for normal [17]	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
Left ventricular wall thickness in diastole, mm	2,2-4,4	3,8-3,9	3,88±0,03	5,7-7,3	6,26±0,40*
Interventricular septum thickness in diastole, mm	2,2-4,0	3,3-3,9	3,64±0,20	4,7-6,6	5,38±0,50**

Continuation of Table 3.12

End-systolic left ventricular size, mm	5,2-10,8	6,0-8,0	6,70±0,70	8,2-16,4	11,70±2,20**
End-diastolic left ventricular size, mm	12,0-19,8	13,0-16,6	14,24±1,10	13,6-20,4	17,38±1,60***
Left atrial cavity size, mm	9,3-15,1	13,6-14,8	14,30±0,40	21,0-36,5	28,26±4,00*
Contractility index, %	39,0-61,0	50,3-55,1	53,02±1,30	19,6-43,9	33,46±5,60**

Note: * p<0,001, ** p<0,01, *** p<0,05 compared to clinically healthy cats

In cats with restrictive cardiomyopathy, the left atrial cavity undergoes significant enlargement, namely by 97.6% (p<0.001) compared to clinically healthy animals. This phenomenon is explained by more pronounced mitral valve insufficiency, manifested in the regurgitation of a larger volume of blood back to the left atrium, compared to cats with hypertrophic cardiomyopathy. It has also been established that cats with restrictive cardiomyopathy have an increased size of the left ventricle of the heart. Thus, the end-diastolic size of the left ventricle in these animals is increased by 22.1% (p<0.05) in diastole and by 74.6% (p<0.01) in systole compared to clinically healthy cats. In cats with restrictive cardiomyopathy, the contractility index is significantly lower (36.9%, p<0.01) compared to clinically healthy animals. This phenomenon can be explained by fibrotic changes in the myocardium, which are more pronounced in cats with restrictive cardiomyopathy compared to cats with hypertrophic cardiomyopathy. Fibrous changes in the myocardium of cats with restrictive cardiomyopathy impair and limit the ability of the heart muscle to contract, i.e., reduce its contractile function.

In all cats with restrictive cardiomyopathy, we observed a significant amount of pericardial effusion (90-120 ml).

Therefore, our results allow us to conclude that restrictive cardiomyopathy in domestic cats can be diagnosed by ultrasound examination based on the following specific signs: significant thickening of the left ventricular wall

myocardium in diastole ($p<0.001$) and interventricular septum during diastole ($p<0.01$) (Fig. 3.13); significant enlargement of the left ventricular cavity during systole ($p<0.01$) and diastole ($p<0.05$) (Fig. 3.13); significant ($p<0.001$) enlargement of the left atrium (Figs. 3.14 and 3.15); possible presence of thrombi in the left atrium (Fig. 3.16); decreased contractility index, i.e. myocardial hypokinesis; marked increase in echogenicity of the left ventricular wall and/or interventricular septum, which is a sign of fibrotic changes (Fig. 3.14); presence of fluid in the pericardial cavity, i.e. pericardial effusion (Fig. 3.15), which is not visualised in all cases.

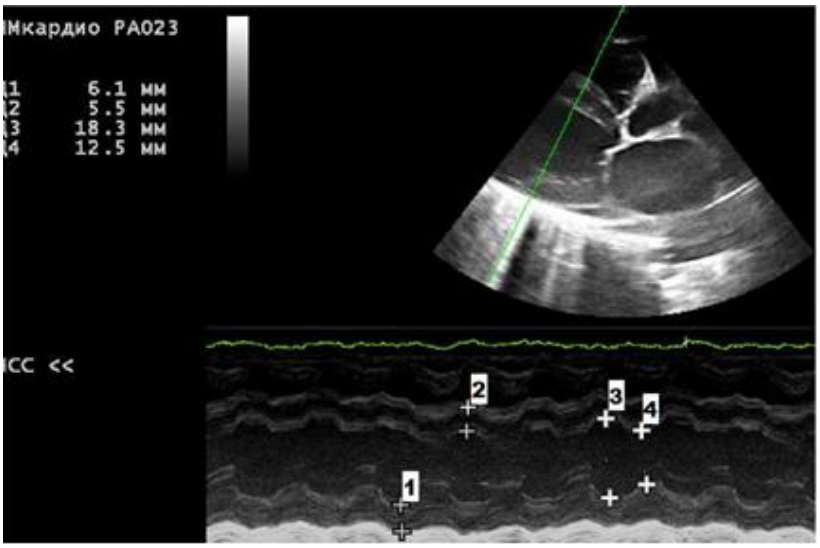

Fig. 3.13. Ultrasonogram of the heart of a cat, mixed breed, name ‘Murzik’, age 15 years. M-mode from the right parasternal access along the long axis of the left ventricle at the level of the papillary muscles. Visualised: thickening of the left ventricular wall to 6.1 mm in diastole and of the interventricular septum to 5.5 mm in diastole, increase in the end-diastolic size of the left ventricle to 18.3 mm and in the end-systolic size to 12.5 mm.

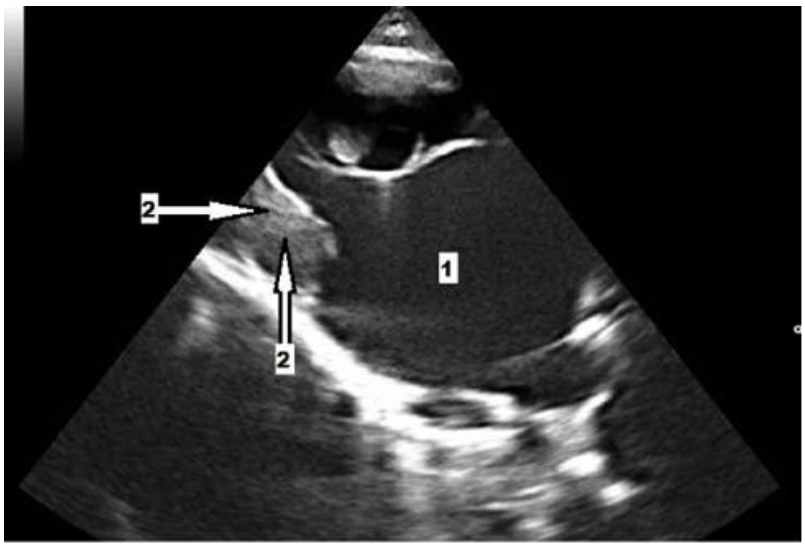

Fig. 3.14. Ultrasonogram of the heart of a British Shorthair cat named Amur, aged 3 years. B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: enlargement of the left atrium chamber to 36.5 mm (1), increased echogenicity of the left ventricular wall in the form of a hyperechoic endomyocardial spot (2), which is a sign of fibrotic changes.

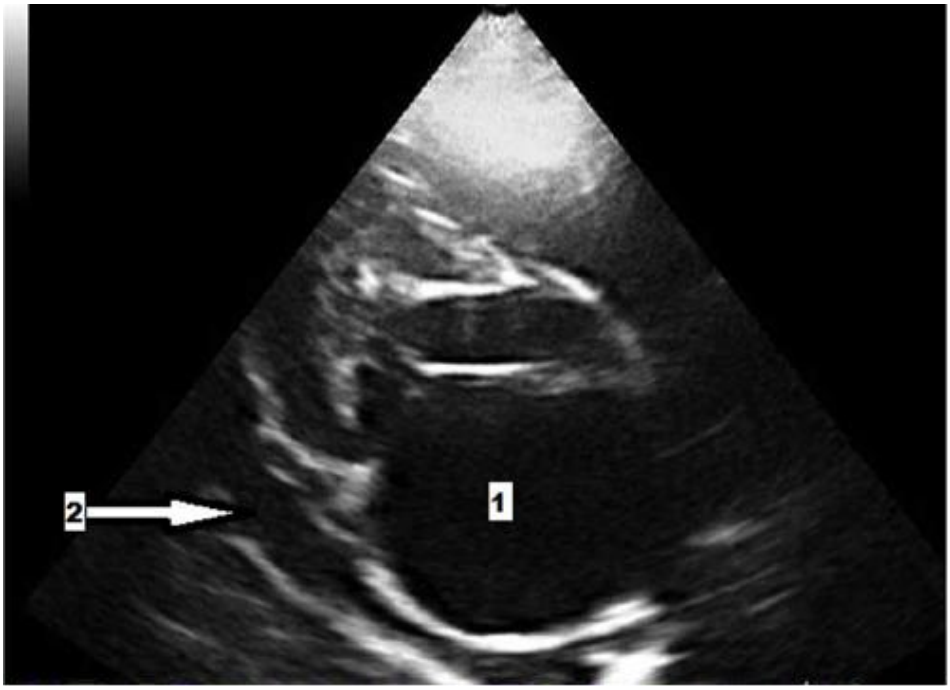

Fig. 3.15. Ultrasonogram of the heart of a British Shorthair cat named Amur, aged 3 years. B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: enlargement of the left atrium chamber to 36.5 mm (1), presence of pericardial effusion (2).

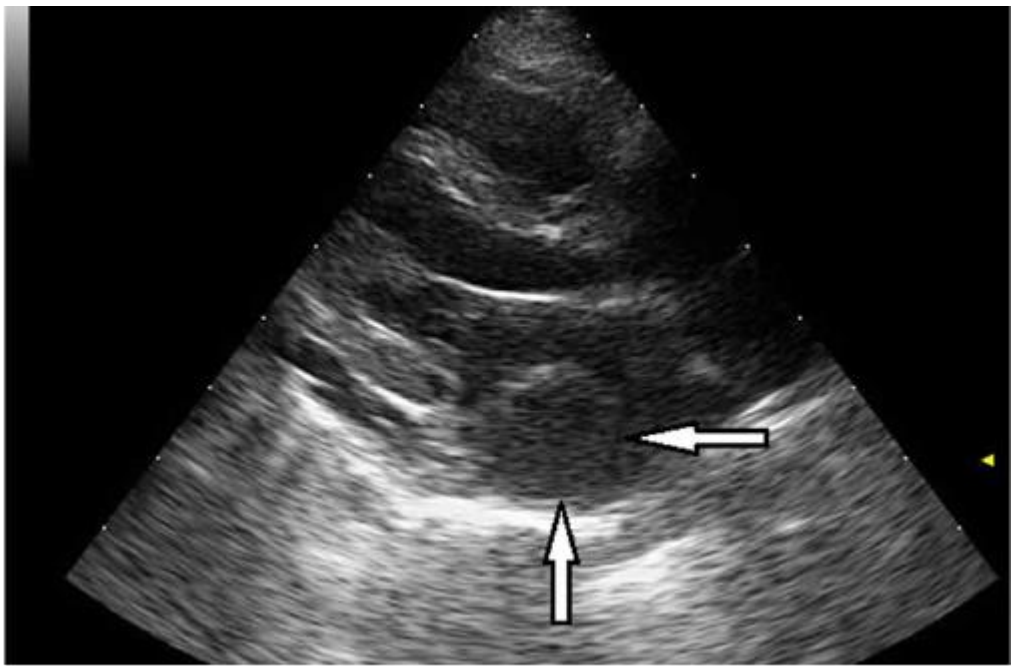

Fig. 3.16. Ultrasonogram of the heart of a British Shorthair cat named Masik, aged 10 years. B-mode from the right parasternal access along the long axis of the left ventricle. A thrombus is visualised in the left atrial cavity (indicated by arrows).

3.6.3. Echocardiographic changes in a domestic cat with dilated cardiomyopathy

Our research has shown that cats with dilated cardiomyopathy exhibit the following changes: the size of the left ventricular cavity in the systole phase is 11.38 mm, which is 69.9% significantly ($p<0.001$) larger than in clinically healthy animals; the size of the left ventricular cavity in the diastolic phase is 19.62 mm, which is 37.8% significantly ($p<0.001$) larger than in clinically healthy cats; the size of the left atrium cavity is 19.32 mm, which is 35.1% significantly ($p<0.01$) larger than in clinically healthy cats; the right ventricle cavity is 8.36 mm, which is 74.9% significantly ($p<0.05$) larger than in clinically healthy animals; the right atrium cavity is 17.62 mm, which is 46.8% significantly ($p<0.01$) larger than in clinically healthy cats (Table 3.13).

That is, cats with dilated cardiomyopathy have an increase in the size of all four chambers of the heart compared to clinically healthy animals. In all cats with dilated cardiomyopathy, we also found insufficiency of the atrioventricular valves, both bicuspid and tricuspid. Atrioventricular valve insufficiency was moderate to severe.

The mean values of left ventricular wall thickness and interventricular septal thickness in cats with dilated cardiomyopathy were almost identical to those in the control group.

However, during studies in three cats with dilated cardiomyopathy, we found that the thickness of the left ventricular wall and interventricular septum was less than that in clinically healthy cats. Given this, it can be assumed that thinning of the left ventricular wall and interventricular septum is a characteristic trend in some cases of dilated cardiomyopathy in cats.

We also found a significant decrease in myocardial contractility index in cats with dilated cardiomyopathy by 21.6% ($p<0.05$) compared to clinically healthy animals. In dilated cardiomyopathy, the systolic function of the left ventricle is impaired. Due to the enlargement of all chambers of the heart in general, and the left ventricle cavity in particular, they become overfilled with blood. The myocardium is unable to contract fully, which manifests itself as systolic insufficiency.

During the analysis of the anamnesis data, we found that the owners of two cats, of the Canadian and Don Sphynx breeds, fed them only natural food, i.e. 'from the table'. This type of feeding is the cause of taurine deficiency in the animals' diet, which in these cases can be considered an aetiological factor in the development of dilated cardiomyopathy [74, 205].

Table 3.13

Echocardiography results for a domestic cat with dilated cardiomyopathy, M±m, n=5

Indexes	Reference values for normal [17]	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
Left ventricular wall thickness in diastole, mm	2,2-4,4	3,8-3,9	3,88±0,03	3,3-4,1	3,76±0,20
Interventricular septum thickness in diastole, mm	2,2-4,0	3,3-3,9	3,64±0,20	3,2-4,7	3,68±0,50

Continuation of Table 1

Left ventricular end-systolic dimension, mm	5,2-10,8	6,0-8,0	6,70±0,70	9,5-13,3	11,38±1,50*
Left ventricular end-diastolic dimension, mm	12,0-19,8	13,0-16,6	14,24±1,10	18,8-20,7	19,62±0,50*
Left atrial cavity size, mm	9,3-15,1	13,6-14,8	14,30±0,40	17,0-24,5	19,32±2,10**
Right ventricular end-diastolic dimension, mm	2,7-9,4	3,9-6,4	4,78±0,80	5,8-11,3	8,36 ±1,80***
Right atrial cavity size, mm	_	11,6-13,2	12,00±0,50	15,5-21,5	17,62 ±1,80**
Contractility index, %	39,0-61,0	50,3-55,1	53,0±1,30	32,0-49,5	41,58±6,00***

Note: * p<0,001, ** p<0,01, *** p<0,05 compared to clinically healthy cats

In three other cats in the study group, dilated cardiomyopathy was the result of an intermediate form of cardiomyopathy.

The study also found that two cats with dilated cardiomyopathy had a moderate amount of free fluid in the pericardium, with a total volume of up to 45 ml.

Thus, dilated cardiomyopathy in domestic cats can be diagnosed by ultrasound examination based on the following reliable specific signs:

— significant enlargement of the left ventricle during systole and diastole ($p<0.001$);

— significant enlargement of the right ventricle ($p<0.05$);

— significant enlargement of the left and right atria ($p<0.01$) (Fig. 3.17);

— significant decrease in myocardial contractility index ($p<0.05$);

— presence of moderate and severe insufficiency of the bicuspid and tricuspid valves (Fig. 3.18).

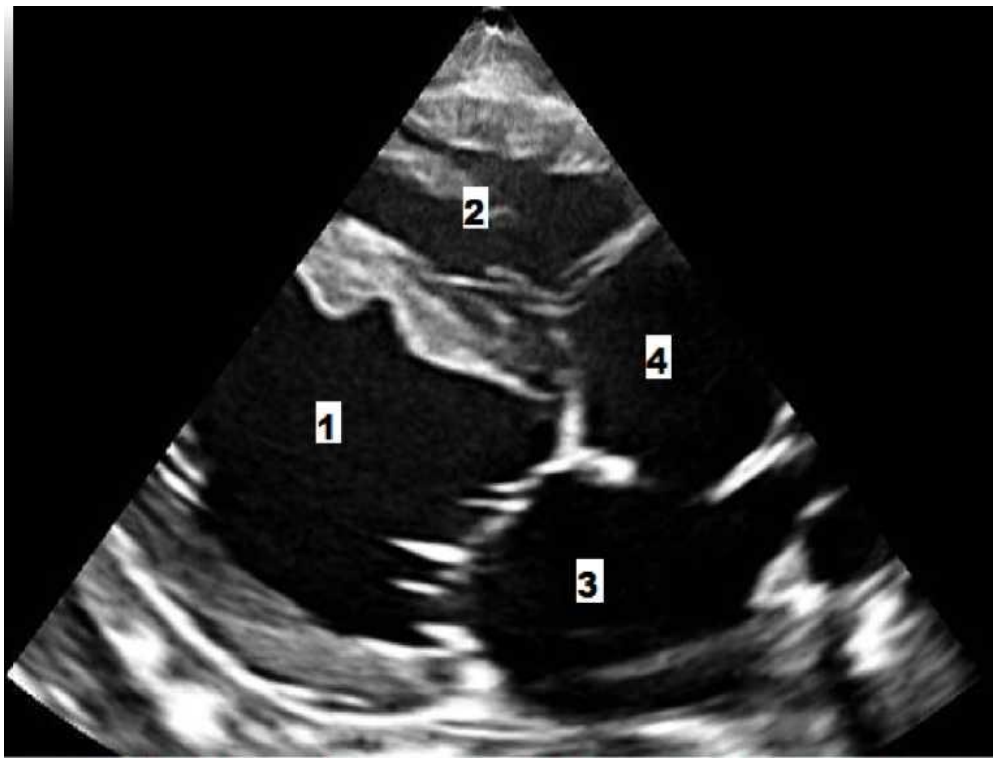

Fig. 3.17. Ultrasonogram of the heart of a cat, breed Canadian Sphynx, name 'Bagira', age 4 years. B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: enlargement of the left ventricle chamber to 19.6 mm in the diastolic phase (1), enlargement of the right ventricle chamber to 9.9 mm (2), enlargement of the left atrium chamber to 17.7 mm (3), enlargement of the right atrium chamber to 18.3 mm (4).

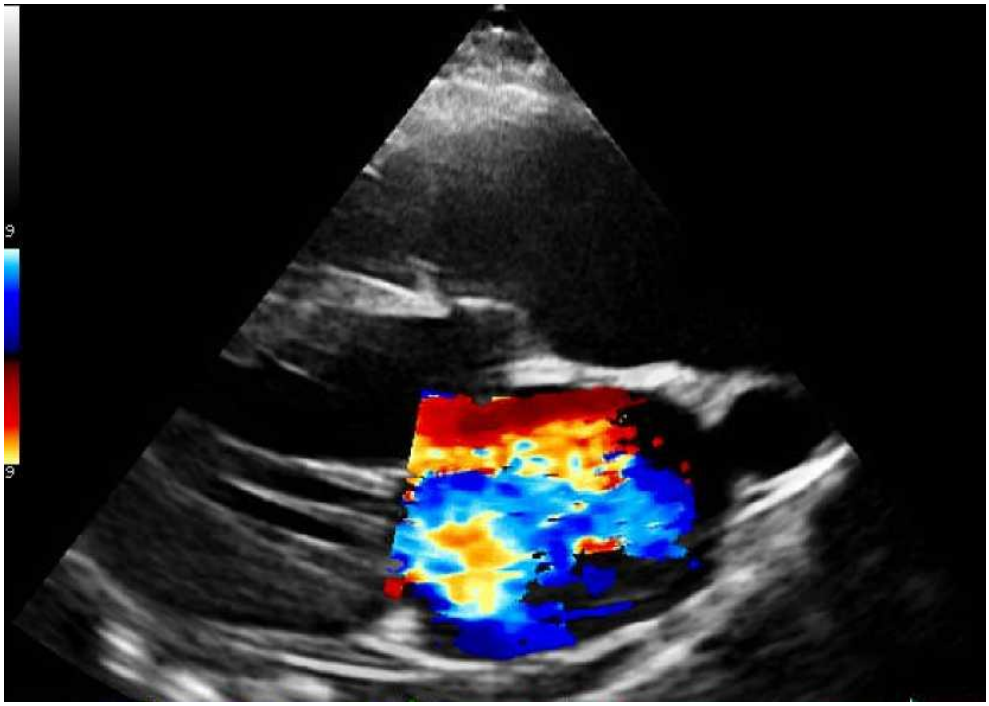

Fig. 3.18. Cardiac ultrasound scan of a 4-year-old Canadian Sphynx cat named 'Bagheera'. B-mode view from the right parasternal approach along the long axis of the left ventricle, using Doppler echocardiography in the form of colour Doppler mapping: severe mitral valve regurgitation is visualised

3.6.4. Echocardiographic changes in a domestic cat with intermediate cardiomyopathy

Based on the results of the first echocardiographic study, we found that cats with intermediate cardiomyopathy had a significant ($p<0.001$) thickening of the left ventricular wall in diastole by 61.5% and the interventricular septum in diastole by 72.5%, compared to clinically healthy cats. Also, during the first study, cats showed a significant enlargement of the left atrium by 31.5% ($p<0.05$) compared to clinically healthy animals. The left ventricular myocardial contraction index tended to increase by 13.2% compared to clinically healthy cats (Table 3.14).

The results of the first echocardiographic examination of the hearts of cats with intermediate cardiomyopathy allow us to diagnose hypertrophic cardiomyopathy in them.

During treatment and observation, we conducted repeated echocardiographic studies of the hearts of animals in this group, the results of which are presented in Table 3.15.

The results of the studies show that in cats with intermediate cardiomyopathy, the following changes were observed during repeated studies: a significant enlargement ($p<0.01$) of the left ventricular cavity during systole by 58.5%; significant enlargement ($p<0.001$) of the left ventricle cavity in diastole by 35.8%; significant enlargement ($p<0.01$) of the left atrium cavity by 36.4%; significant enlargement ($p<0.05$) of the right ventricle by 68.2% and significant enlargement ($p<0.01$) of the right atrium by 46.7% compared to clinically healthy animals. There was also a tendency towards a 15.4% decrease in the left ventricular myocardial contractility index.

All cats in the experimental group had varying degrees of atrioventricular valve insufficiency. Two animals had a small amount of free fluid in the pericardium (about 15-45 ml).

Thus, based on the results of our repeated echocardiographic studies of cats in the experimental group, dilated cardiomyopathy was diagnosed.

The time interval between the first and repeated echocardiographic studies ranged from 5 to 32 months. The average time interval between studies ranged from

12 to 20 months, and in the case of three sick animals, this period was 5-7 months. A comparison of the results of the first and repeated echocardiographic studies is presented in Table 3.16.

Table 3.14

Echocardiography results for a domestic cat with intermediate cardiomyopathy during the initial examination, M±m, n=5

Indexes	Reference values for normal [17]	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
Left ventricular wall thickness in diastole, mm	2,2-4,4	3,8-3,9	3,88±0,03	5,1-7,5	6,30±0,40*
Interventricular septum thickness in diastole, mm	2,2-4,0	3,3-3,9	3,64±0,20	5,1-7,6	6,28±0,70*
Left ventricular end-systolic dimension, mm	5,2-10,8	6,0-8,0	6,70±0,70	4,4-7,0	5,92±0,90
Left ventricular end-diastolic dimension, mm	12,0-19,8	13,0-16,6	14,24 ±1,10	12,7-18,2	14,40±1,60
Left atrial cavity size, mm	9,3-15,1	13,6-14,8	14,30 ±0,40	13,5-22,1	17,76 ±2,40***
Right ventricular end-diastolic dimension,	2,7-9,4	3,9-6,4	4,78±0,80	4,4-8,1	6,24±1,40
Right atrial cavity size, mm	–	11,6-13,2	12,00 ±0,50	12,7-15,3	14,2±0,7**
Contractility index, %	39,0-61,0	50,3-55,1	53,02 ±1,30	51,8-66,0	58,64±4,50

Note: * p<0,001, ** p<0,01, *** p<0,05 compared to clinically healthy cats

Thus, the results of the studies show that during repeated echocardiographic studies in sick cats with intermediate cardiomyopathy, there is a significant (p<0.001) thinning of the left ventricular wall in diastole by 36.2% and the interventricular septum in diastole by 41.9%, compared with the data for the same animals in the first study. Also, in repeated echocardiographic studies, in sick cats, there was a significant enlargement of the left ventricle in systole by 79.4% (p<0.01) and in diastole by 34.3% (p<0.01) and of the right atrium by 20.1% (p<0.05).

Table 3.15

Echocardiography results of a domestic cat with intermediate cardiomyopathy during a repeat examination, M±m, n=5

Indexes	Reference values for normal [17]	Clinically healthy cats		Cats with cardiomyopathy	
		Lim	M±m	Lim	M±m
Left ventricular wall thickness in diastole, mm	2,2-4,4	3,8-3,9	3,88±0,03	3,7-4,4	4,02±0,20
Interventricular septum thickness in diastole, mm	2,2-4,0	3,3-3,9	3,64±0,20	3,2-4,4	3,65±0,40
Left ventricular end-systolic dimension, mm	5,2-10,8	6,0-8,0	6,70±0,70	8,4-13,3	10,62±1,30**
Left ventricular end-diastolic dimension, mm	12,0-19,8	13,0-16,6	14,24 ±1,10	18,5-20,7	19,34±0,70*
Left atrial cavity size, mm	9,3-15,1	13,6-14,8	14,30 ±0,40	17,0-24,5	19,50±2,20**
Right ventricular end-diastolic dimension, mm	2,7-9,4	3,9-6,4	4,78±0,80	6,2-11,3	8,04±1,30***
Right atrial cavity size, mm		11,6-13,2	12,00 ±0,50	15,4-21,5	17,06±1,80**
Contractility index, %	39,0-61,0	50,3-55,1	53,02 ±1,30	33,3-54,6	44,84±5,10

Note: * p<0,001, ** p<0,01, *** p<0,05 compared to clinically healthy cats

The results of the studies indicate that the left atrial cavity of sick cats with intermediate cardiomyopathy already shows significant enlargement ($p<0.05$) compared to clinically healthy cats during the first examination, and repeated examinations show an increase in the left atrial cavity by another 9.8%.

The data we obtained allow us to draw the following conclusion. The initial (first) stage of intermediate cardiomyopathy, in which a concentric type of myocardial hypertrophy resembling hypertrophic cardiomyopathy is formed, is compensatory. However, over time, the compensatory capabilities of the body are exhausted, and the transition to the stage of decompensation forms an eccentric type of cardiomyopathy, resembling dilated cardiomyopathy.

Table 3.16

Comparative assessment of echocardiographic findings in domestic cats with intermediate cardiomyopathy, M±m, n=5

Indexes	Initial examination of cats with intermediate		Repeat examination of cats with intermediate	
	Lim	**M±m**	**Lim**	**M±m**
Left ventricular wall thickness in diastole, mm	5,1-7,5	6,30±0,43	3,7-4,4	4,02±0,20*
Interventricular septum thickness in diastole, mm	5,1-7,6	6,28±0,70	3,2-4,4	3,65±0,40*
Left ventricular end-systolic dimension, mm	4,4-7,0	5,92±0,90	8,4-13,3	10,62±1,30**
Left ventricular end-diastolic dimension, mm	12,7-18,2	14,40±1,56	18,5-20,7	19,34±0,60**
Left atrial cavity size, mm	13,5-22,1	17,76±2,41	17,0-24,5	19,50±2,20
Right ventricular end-diastolic dimension, mm	4,4-8,1	6,24±1,37	6,2-11,3	8,04±1,30
Right atrial cavity size, mm	12,7-15,3	14,20±0,68	15,4-21,5	17,06 ±1,80***
Contractility index, %	51,8-66,0	58,64±4,49	33,3-54,6	44,84 ±5,10***

Note: * p<0,001, ** p<0,01, *** p<0,05 compared with the results obtained in the first echocardiographic examination

It is the final, dilated form of cardiomyopathy, which developed in a domestic cat at the end of the disease during the intermediate form of cardiomyopathy, that should be considered the final nosological unit. A period of 5-7 months is sufficient for the transition from the initial to the final stage of the disease.

Thus, the intermediate form of cardiomyopathy is a pathological process of myocardial remodelling from hypertrophic to dilated cardiomyopathy.

3.6.5. Echocardiographic changes in domestic cats with viral infections

During the study, we analysed the results of echocardiographic changes in domestic cats with viral infections, including two Scottish Fold cats, one Maine Coon cat, and four domestic shorthair cats (n=7). The age of the studied animals ranged from 8 months to 7 years.

Among the cats studied, five had panleukopenia and two had coronavirus infection. The diagnosis was confirmed by enzyme-linked immunosorbent assay (ELISA) of the blood serum of the study cats, which revealed a high titre of antibodies to the panleukopenia virus (IgG FPLV = 1:160–1:640) and a low titre to coronavirus infection (IGG FcoV = 1:10–1:20). Based on the medical history and ELISA, we confirmed the asymptomatic course of these viral infections in 12.1% of the experimental cats. One cat had an acute course of coronavirus infection [89], and the presence of the pathogen in the blood of this animal was confirmed by a positive polymerase chain reaction (PCR) result.

Panleukopenia and coronavirus infection in domestic cats often have a subclinical (asymptomatic) course. One of the serious negative consequences of their course is myocarditis [105].

In three cats from the experimental group, we diagnosed hypertrophic cardiomyopathy during the first ultrasound examination of the heart (Fig. 3.19). Repeated ultrasound examinations of the heart made it possible to diagnose dilated cardiomyopathy in these same animals (Fig. 3.20). Such remodelling of the heart over time is a manifestation of an intermediate form of cardiomyopathy [90].

In addition, four cats in the experimental group had obstruction (stenosis) of the left ventricular outflow tract. Two more cats were diagnosed with signs of hydropericardium, with visualised free fluid in the pericardial cavity in an amount of up to 45 ml. We believe that the presence of free fluid in the pericardium may be a consequence of viral pleuropericarditis in these animals.

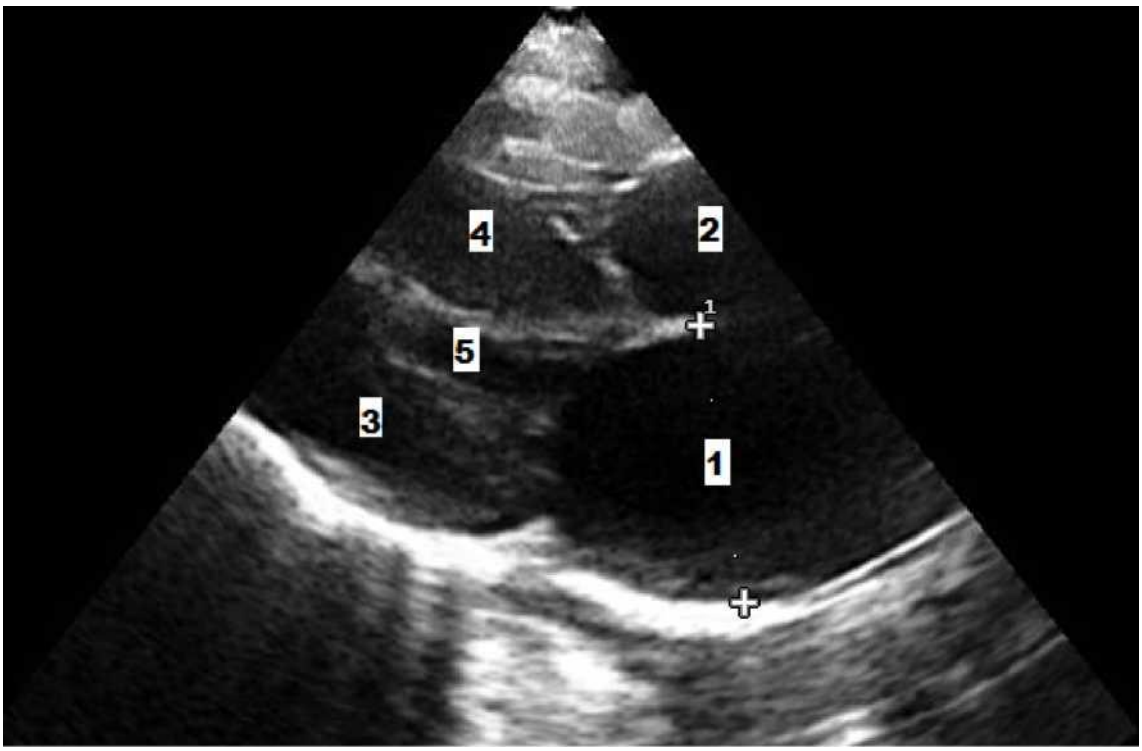

Fig. 3.19. Ultrasonogram of the heart of a cat named 'Tair' of the domestic shorthair breed, 1 year old (first examination). B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: enlarged left atrium chamber (1) to 17.9 mm; right atrium chamber within normal limits (2) at 12.7 mm; thickened free wall of the left ventricle (3) to 6.2 mm; interventricular septum thickened to 6.2 mm (4); pronounced anechogenicity of the left ventricle wall (3) and interventricular septum (4), left ventricle cavity (5).

It is very difficult (almost impossible) to diagnose the acute stage of myocarditis in a sick animal using echocardiography. Since the myocardium is a well-vascularised tissue, in clinically healthy animals and animals with acute myocarditis, the echogenicity of the heart muscle appears as a distinctly anechoic structure, which does not distinguish them from each other (Fig. 3.19).

With the development of fibrotic changes (proliferation of loose connective fibrous tissue), which are the result of incomplete completion of the inflammatory process in the tissues [1], echocardiography can be used to detect increased echogenicity of the left ventricular wall and/or interventricular septum. Signs of fibrotic changes in the myocardium can be visualised as hyperechoic spots or as a diffuse increase in the echogenicity of the myocardium of affected animals as a whole (Fig. 3.21) [92].

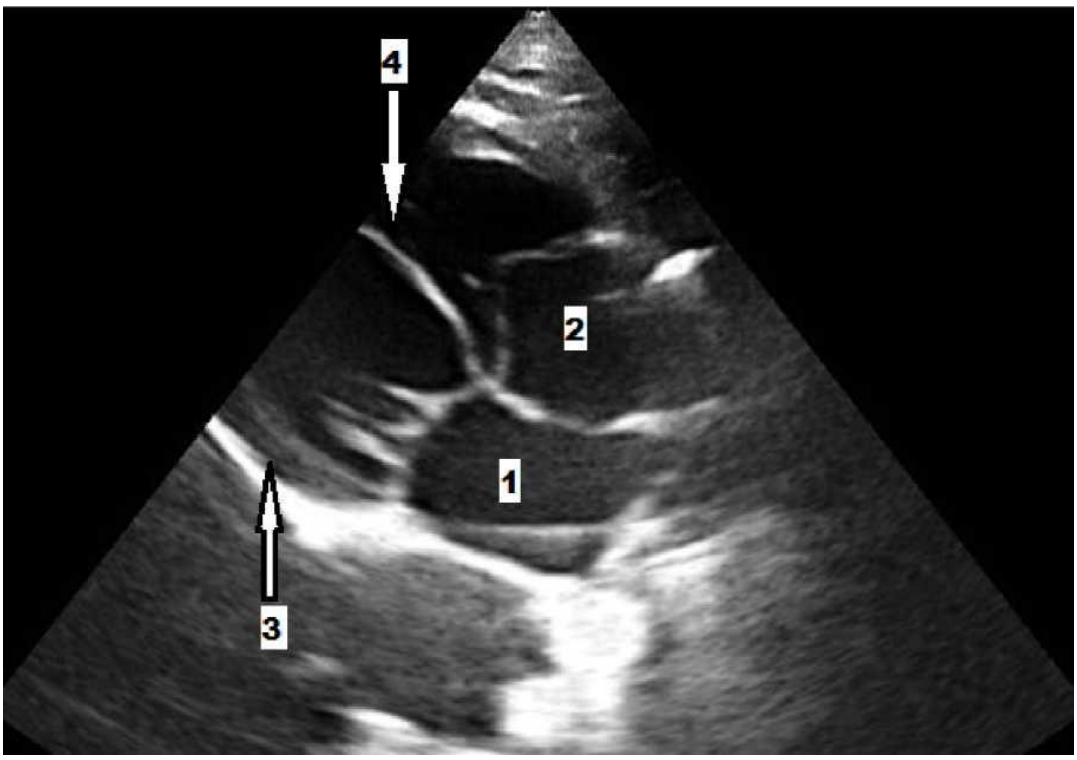

Fig. 3.20. Ultrasonogram of the heart of a cat named 'Tair' of the domestic shorthair breed, 1 year old (5 months after the first examination). B-mode from the right parasternal access along the long axis of the left ventricle. Visualised: enlarged left atrium (1) to 18.0 mm and right atrium (2) to 16.7 mm; Thinning of the free wall of the left ventricle to 4.1 mm (3) and the interventricular septum to 3.8 mm (4); Enlargement of the left ventricle cavity to 19.7 mm (5)

The role of infectious myocarditis can be confirmed by histological studies of the heart muscle of cats that died with a diagnosis of cardiomyopathy [92, 94].

At present, it is impossible to conduct histological studies of the myocardium of cats with cardiomyopathy by means of ante-mortem biopsy in small animal veterinary medicine.

According to recent studies, in 52% of cases, myocarditis is considered to be the cause of secondary cardiomyopathy. Viral infections are considered one of the main etiological factors of myocarditis, and the term inflammatory cardiomyopathy is used to describe heart diseases that develop in this case [2, 80, 114, 140].

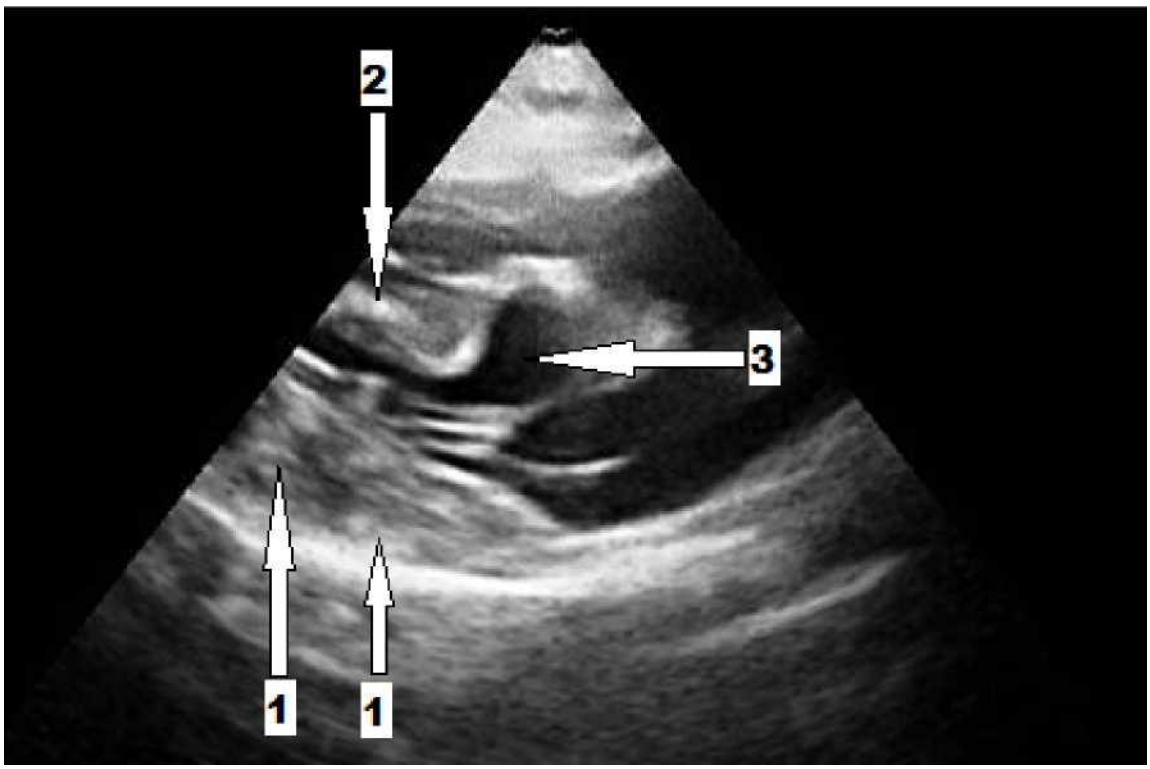

Fig. 3.21. Ultrasonogram of the heart of a Scottish Fold cat named Musya, aged 4 years (B-mode from the right parasternal access along the long axis of the left ventricle). Visualised: presence of hyperechoic spots in the myocardium of the free wall of the left ventricle (1) and the myocardium of the interventricular septum (2), stenosis (obstruction) of the left ventricular outflow tract (3).

The results of echocardiographic studies of domestic cats with viral infections allow us to draw the following conclusions:

— Myocarditis is one of the possible causes of cardiomyopathy in domestic cats.

— Coronavirus infection and viral panleukopenia are among the possible aetiological factors that can cause myocarditis (inflammatory cardiomyopathy) in domestic cats.

— Inflammatory cardiomyopathy that develops in domestic cats as a result of viral myocarditis can be considered secondary.

— Ultrasound examination of the heart two or more times at intervals of no more than 6 months allows for a more accurate classification of the form of cardiomyopathy in domestic cats.

3.7. Pathohistological studies of cardiomyopathies in domestic cats

We conducted pathohistological studies of the myocardium in five cases of death in cats diagnosed with hypertrophic cardiomyopathy, including three Scottish Fold cats, one British Shorthair cat, and one Maine Coon cat. The animals were aged between 1 and 8 years.

Based on the results of pathohistological studies of the cats' myocardium, we established the presence of hypertrophic changes in both individual cardiomyocytes (Fig. 3.22) and muscle fibre hypertrophy (Fig. 3.23) in all five animals. Such changes are most characteristic of the hypertrophic form of cardiomyopathy.

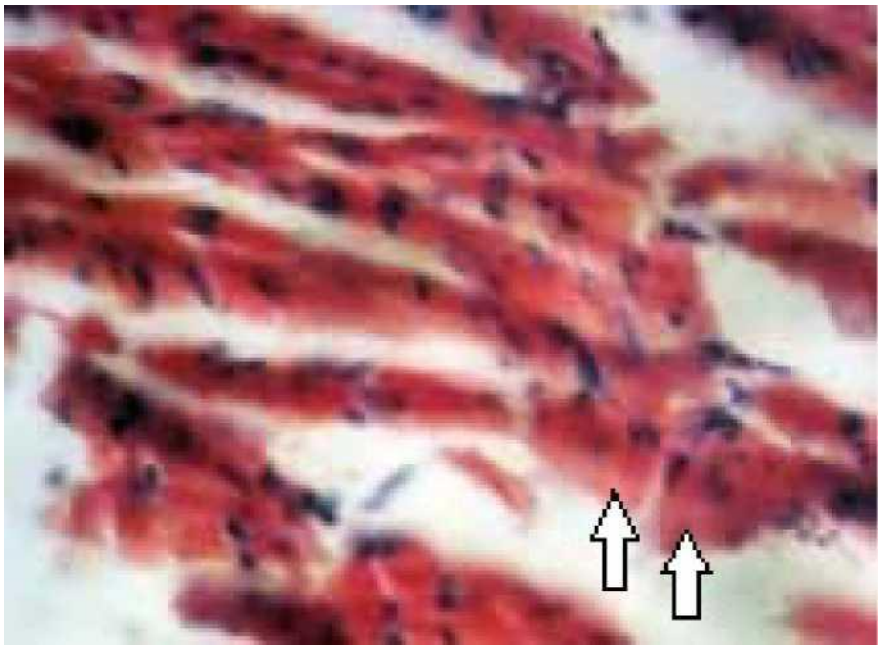

Fig. 3.22. Myocardium of cats with cardiomyopathy (magnification x1000, haematoxylin-eosin staining): hypertrophied individual cardiomyocytes are visualised.

In the myocardium of one cat, a combination of atrophy, dystrophy and hypertrophy of cardiomyocytes was found (Fig. 3.24). This phenomenon is the main characteristic of the dilated form of cardiomyopathy [136]. In our case, during echocardiographic examination, this cat was diagnosed with hypertrophic cardiomyopathy. However, it can be assumed that over time, this animal could have developed dilated cardiomyopathy as a result of the intermediate form of cardiomyopathy.

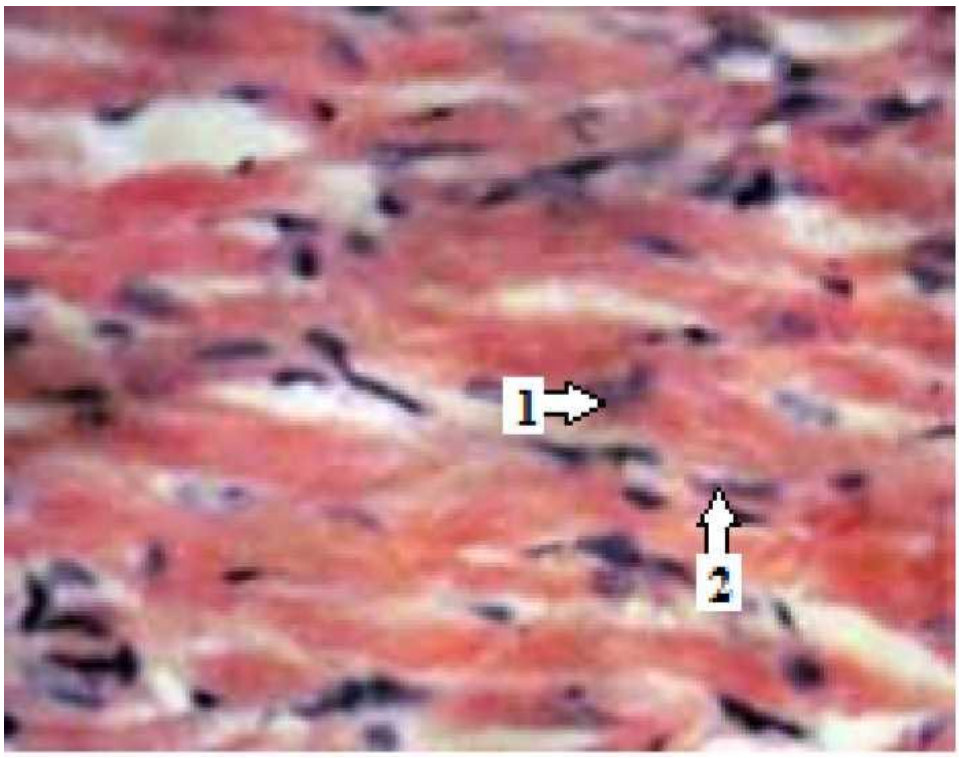

Fig. 3.23. Myocardium of cats with cardiomyopathy (magnification x400, haematoxylin-eosin staining): pronounced hypertrophy of muscle fibres (1) is visualised, interfibrous spaces are minimal in width, almost absent.

Fibrotic changes are an important pathognomonic indicator of cardiomyopathy. Fibrosis develops in areas of cardiomyocyte damage and may appear as focal sclerosis [55, 56].

We also found the presence of loose connective fibrous tissue (fibrotic changes) in the intercellular space of the myocardium of cats that died from cardiomyopathy (Fig. 3.25 and Fig. 3.26).

Fibrous changes in the myocardium of animals, on the one hand, may be the result of apoptosis — genetically programmed death of cardiomyocytes, although this issue remains open today [10, 127]. On the other hand, fibrotic changes in the myocardium develop as a result of chronic inflammation [1, 38, 55]. The development of fibrotic changes in the myocardium leads to a decrease in its elasticity, which, in turn, reduces contractile function and impairs diastolic function of the heart [38].

In the myocardium of two cats that died from hypertrophic cardiomyopathy, we found lymphohistiocytic infiltration in the intercellular space (Figs. 3.27 and 3.28).

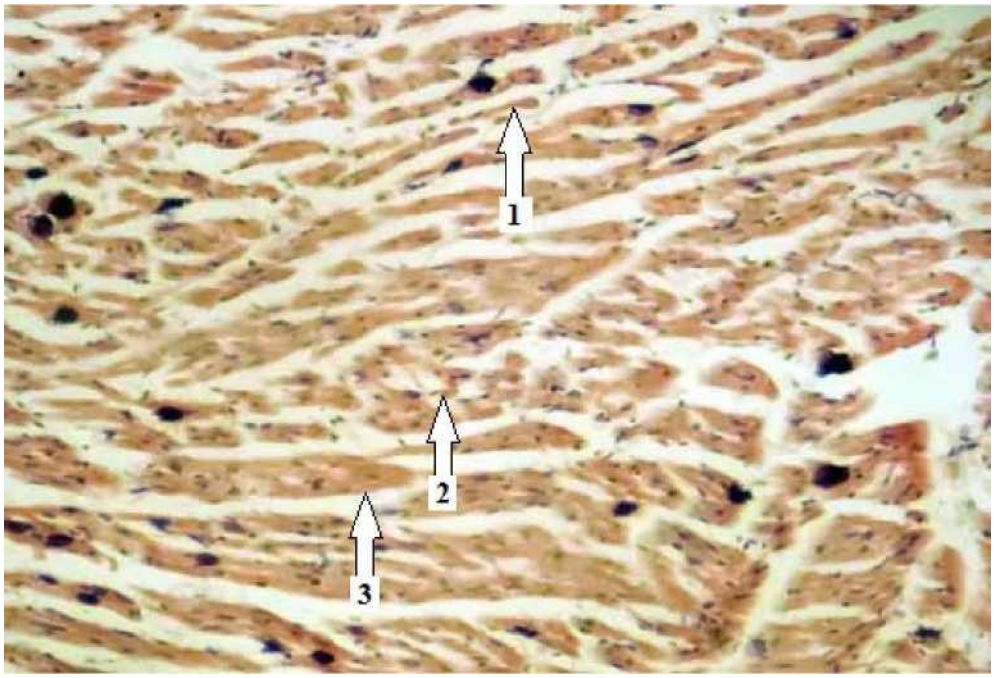

Fig. 3.24. Myocardium of cats with cardiomyopathy (magnification x100, haematoxylin-eosin staining): a combination of atrophy (1), dystrophy (2) and hypertrophy (3) of cardiomyocytes is visualised.

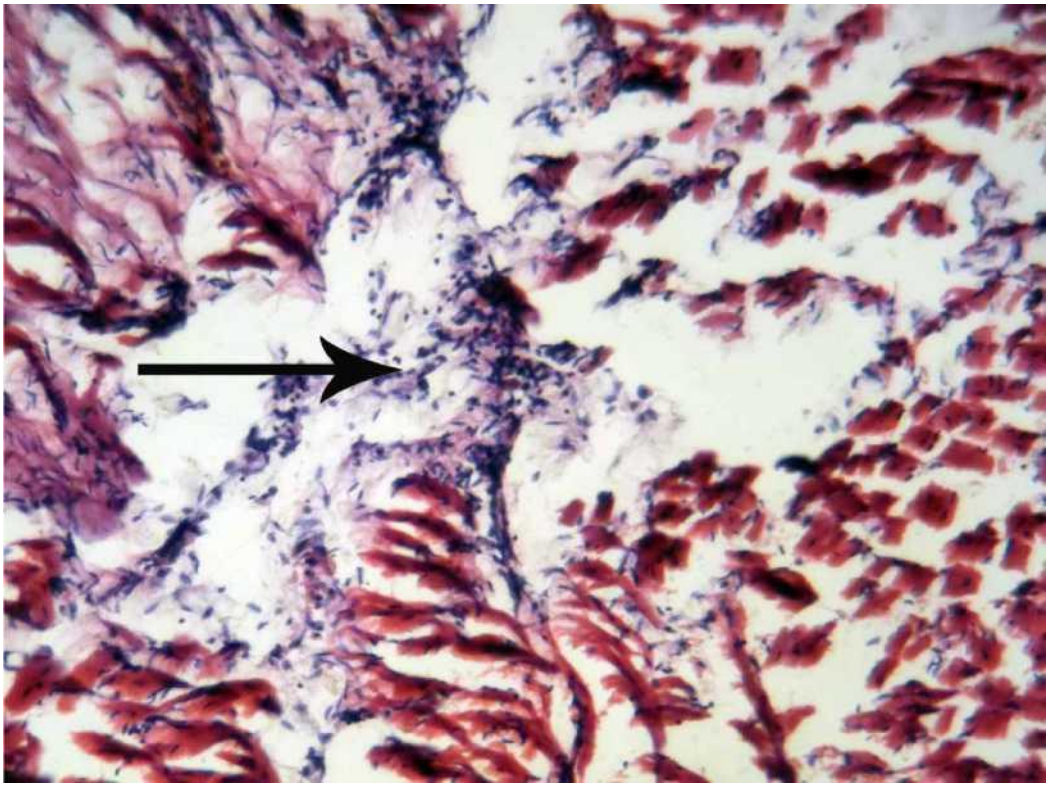

Fig. 3.25. Myocardium of cats with cardiomyopathy (magnification x100, haematoxylin-eosin staining): visualisation of a focus of loose connective fibrous tissue proliferation in the intercellular space (fibrotic changes)

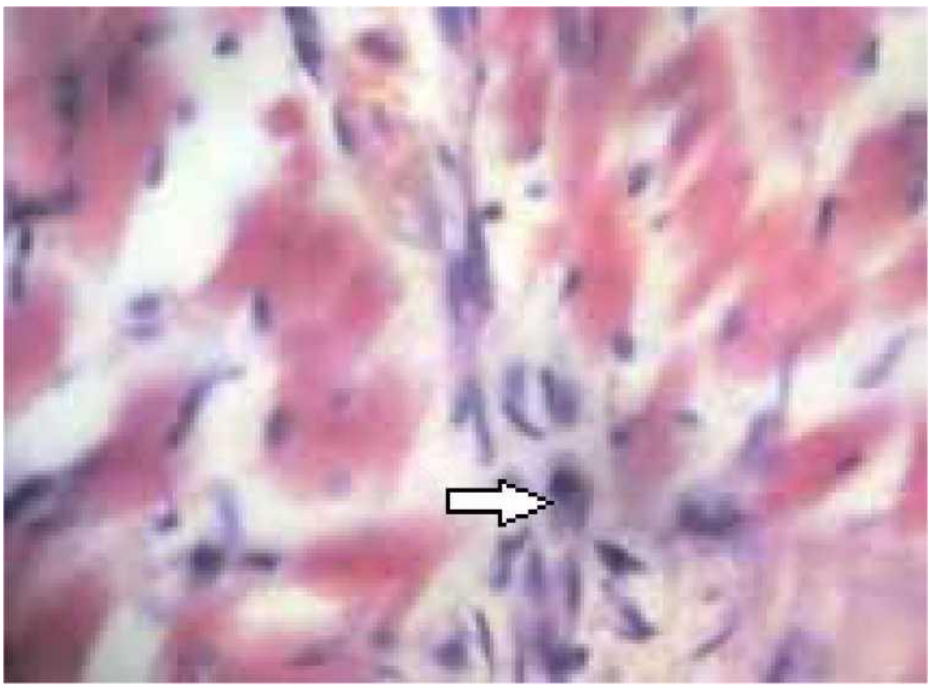

Fig. 3.26. Myocardium of cats with cardiomyopathy (magnification x400, haematoxylin-eosin staining): proliferation of fibroblasts in the connective tissue layer is visualised.

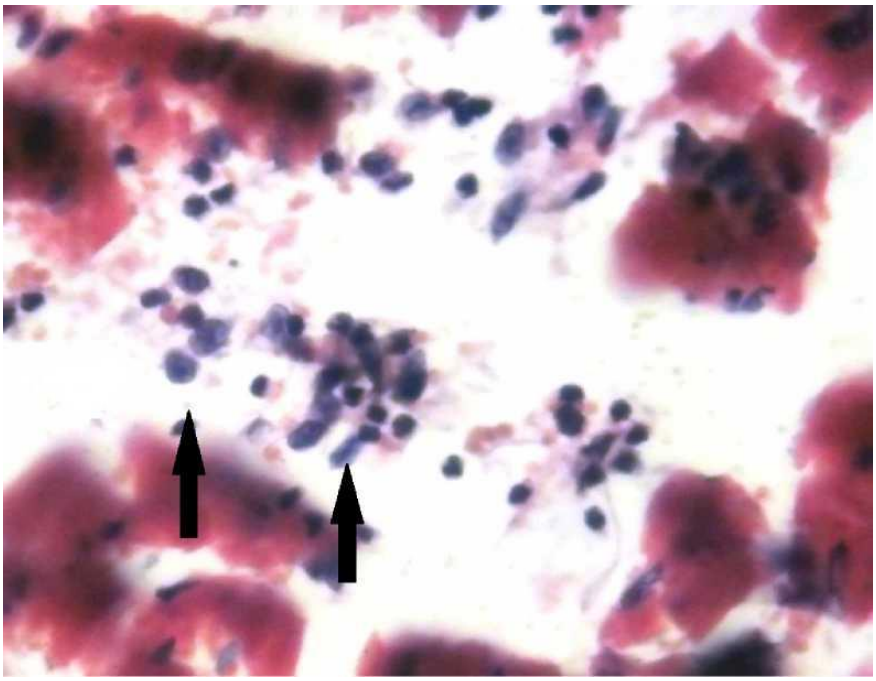

Fig. 3.27. Myocardium of cats with cardiomyopathy (magnification x400, haematoxylin-eosin staining): lymphohistiocytic infiltration in the intercellular space is visualised.

Lymphohistiocytic infiltration in the myocardium of a cat with cardiomyopathy is an important pathognomonic confirmation of infectious myocarditis. One of the aetiological factors that can cause such myocarditis in domestic cats is coronavirus infection and viral panleukopenia [93].

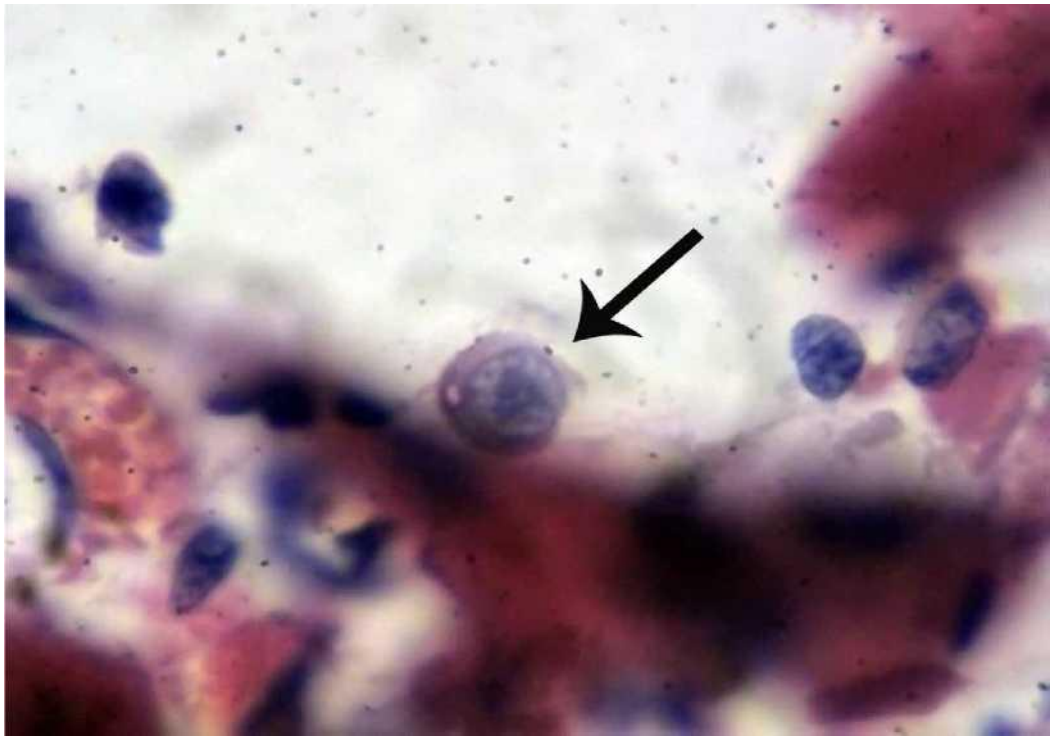

Fig. 3.28. Myocardium of cats with cardiomyopathy (magnification x1000, haematoxylin-eosin staining): lymphocytes are visualised in the intercellular space.

The results of many studies currently available indicate the role of a number of viral infections in the onset and development of myocarditis in animals. In turn, myocarditis leads to the development of cardiomyopathy, both the inflammatory form of cardiomyopathy in general and its dilated form in particular. [2, 114, 127, 140].

3.8. Treatment of domestic cats for cardiomyopathy and prevention of these pathologies

It should be noted that effective methods for treating animals with cardiomyopathy have not yet been developed [122].

The vast majority of cardiomyopathies in domestic cats are asymptomatic (subclinical). Based on this, there is no practically proven and scientifically substantiated data showing the positive effect of certain therapies on the course of asymptomatic cardiomyopathy and improving the prognosis in affected animals. In view of this, the treatment of animals for cardiomyopathy is carried out on an empirical basis [201, 216].

In the vast majority of cases, treatment of animals for cardiomyopathy begins

when the animal shows signs of the disease. The most common signs in domestic cats include shortness of breath with pronounced tachypnea, pulmonary oedema, and arterial thromboembolism.

The leading role in the pathogenetic mechanism of decompensated heart function in cardiomyopathy belongs to the neurohumoral activation of the renin-angiotensin-aldosterone system (RAAS) [49, 126, 137, 143, 166].

Experimental studies show that when drugs that can block the RAAS are used, there is a decrease in cardiomyocyte apoptosis and collagen synthesis by fibroblasts, and the growth of connective tissue (fibrosis) in the interstitial space of the myocardium slows down. As a result, left ventricular myocardial tension decreases and the process of its remodelling slows down [61, 215].

In the course of our work, we investigated the therapeutic effect of drugs that can block the RAAS system on the course of cardiomyopathy in sick cats. The treatment of animals was evaluated based on changes in echocardiographic parameters during repeated ultrasound examinations of the heart.

The experimental group included 9 cats aged 9 months to 10 years.

Two cats had an asymptomatic course of the disease. Six cats were admitted to the veterinary clinic with symptoms of severe shortness of breath and pulmonary oedema. All of these animals were diagnosed with hypertrophic cardiomyopathy during the first echocardiographic examination. The owners of one cat came to the veterinary clinic with symptoms of lameness, namely caused by the animal's inability to bear weight on its left hind limb.

In this animal, based on specific clinical signs and the results of X-ray and ultrasound examinations, we diagnosed thromboembolism of the left femoral artery and restrictive cardiomyopathy.

It should also be noted that prior to the onset of symptoms of pulmonary oedema, the animals lived without any signs of disease. In other words, the cats had an asymptomatic course of the disease for a long period of time.

Cats with symptoms of pulmonary oedema due to hypertrophic cardiomyopathy underwent urgent comprehensive treatment in the intensive care and

resuscitation unit of the veterinary clinic.

In the acute period, with a symptom complex of pulmonary oedema, the animals were treated with intravenous drip infusions using 0.9% sodium chloride physiological solution and diuretics (furosemide at a dose of 1-8 mg/kg). In cases of severe dyspnoea, oxygen therapy was administered to sick animals to improve gas exchange in the lungs.

Treatment of cats with cardiomyopathy was carried out using drugs that mainly affect the RAAS system: ACE inhibitors (enalapril at a dose of 0.125-0.25 mg/kg per day, continuously); calcium channel blockers (amlodipine at a dose of 0.5 mg/kg per day, continuously); diuretics (spironolactone 1-2 mg/kg per day, in courses of up to 1 month).

For severe symptoms of dyspnoea, loop diuretics were used (torasemide at a dose of 0.5-1.0 mg/kg per day, in courses of up to 7 days).

All drugs were administered orally to sick animals from the first day their owners brought them to the veterinary clinic.

A cat with femoral artery thromboembolism due to restrictive cardiomyopathy received oral acetylsalicylic acid (aspirin cardio k) at a dose of 10 mg/kg once every 72 hours, in a course of 10 times, to reduce platelet aggregation. It should be noted that this animal, apart from the inability to functionally use the left pelvic limb, had no clinical signs of cardiomyopathy.

Two animals diagnosed with hypertrophic cardiomyopathy died within the first 2 days after the onset of symptoms of pulmonary oedema. After the death of these animals, samples of pathological material were taken and histological studies of the myocardium were performed, the results of which are described in detail in section 3.7.

The other animals receiving treatment for cardiomyopathy underwent initial and repeat echocardiographic examinations.

During the first echocardiographic examination in cats, we diagnosed hypertrophic cardiomyopathy. The experimental animals were treated according to the above-described scheme. During repeated echocardiographic examinations in

these animals, we diagnosed dilated cardiomyopathy. The results of these studies, which we describe in detail in section 3.6.4, show the manifestation of an intermediate form of cardiomyopathy in sick animals.

The results of a comparative assessment of the first and repeat echocardiographic studies of cats with cardiomyopathy are presented in Table 3.16.

Thus, the results of our studies indicate that even when treating sick cats with drugs that have a blocking effect on the RAAS system, the process of myocardial remodelling from hypertrophic to dilated cardiomyopathy occurs.

After our treatment, the life expectancy of one cat was 10 months, and the other was 3 years. For the other three animals, the course of treatment continued at the time of completion of our work. The time required for the development of dilated cardiomyopathy with the manifestation of an intermediate form of the disease is 5-7 months.

Thus, cardiomyopathy in domestic cats is predominantly asymptomatic.

The main causes of secondary cardiomyopathy in domestic cats in metropolitan areas are hyperazotaemia, which develops in the course of chronic kidney disease, and myocarditis due to panleukopenia and coronavirus infections. This can be confirmed by laboratory tests of blood serum for creatinine and cardiac troponin I concentrations and immunoassays for viral infections.

The best non-invasive method for diagnostic confirmation and differential diagnosis of cardiomyopathy in domestic cats is echocardiography.

Treatment of cardiomyopathy in domestic cats is symptomatic and aimed at eliminating the consequences of heart failure.

Unit 4

ANALYSIS AND DISCUSSION OF RESEARCH RESULTS

Scientific advances in recent years have greatly influenced our understanding of heart pathologies in animals. Nevertheless, there are still a number of problems regarding the diagnosis of cardiomyopathies in domestic cats and the treatment of animals with this pathology.

In this paper, we describe the characteristics of cardiomyopathy in domestic cats, their clinical manifestations, morphofunctional disorders of the heart and metabolic processes, and the nature of echostructural changes in the heart in domestic cats with various forms of cardiomyopathy. The results of our research allow us to select informative diagnostic criteria that enable us to make a reliable diagnosis and apply the most effective therapeutic and preventive measures for animals with cardiomyopathies.

Over a period of four years, we conducted a clinical study of 58 domestic cats with cardiomyopathies. The indicators obtained from sick domestic cats were compared with those in clinically healthy animals (n = 16).

Cardiomyopathies in domestic cats are characterised by changes in the anatomical structures of the heart, leading to a disorder of its pumping function and severe haemodynamic disturbances. Despite the widespread prevalence of heart pathologies, the course of cardiomyopathy in domestic cats in Ukraine remains poorly studied.

Taking into account the above, we determined the diagnostic informativeness of physical examination data of animals with cardiomyopathy, laboratory blood parameters, and the results of instrumental research methods.

According to the anamnesis and clinical examination methods, the vast majority of cardiomyopathies in domestic cats are asymptomatic (Fig. 4.1).

Even in animals that were admitted to the veterinary clinic in an urgent condition (pulmonary oedema and arterial thromboembolism), according to the medical history, the disease was asymptomatic (Fig. 4.2).

One cat with pulmonary oedema was 13 years old, and another with arterial thromboembolism was 10 years old.

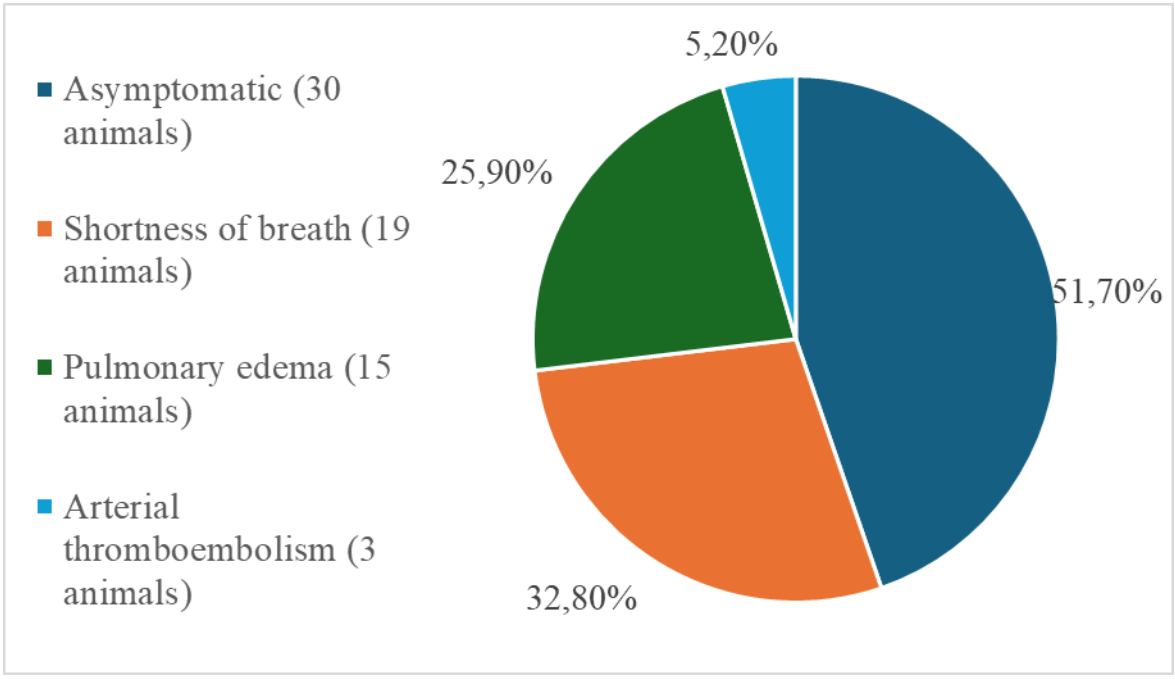

Fig. 4.1. Clinical manifestation of cardiomyopathy in a domestic cat, n=58

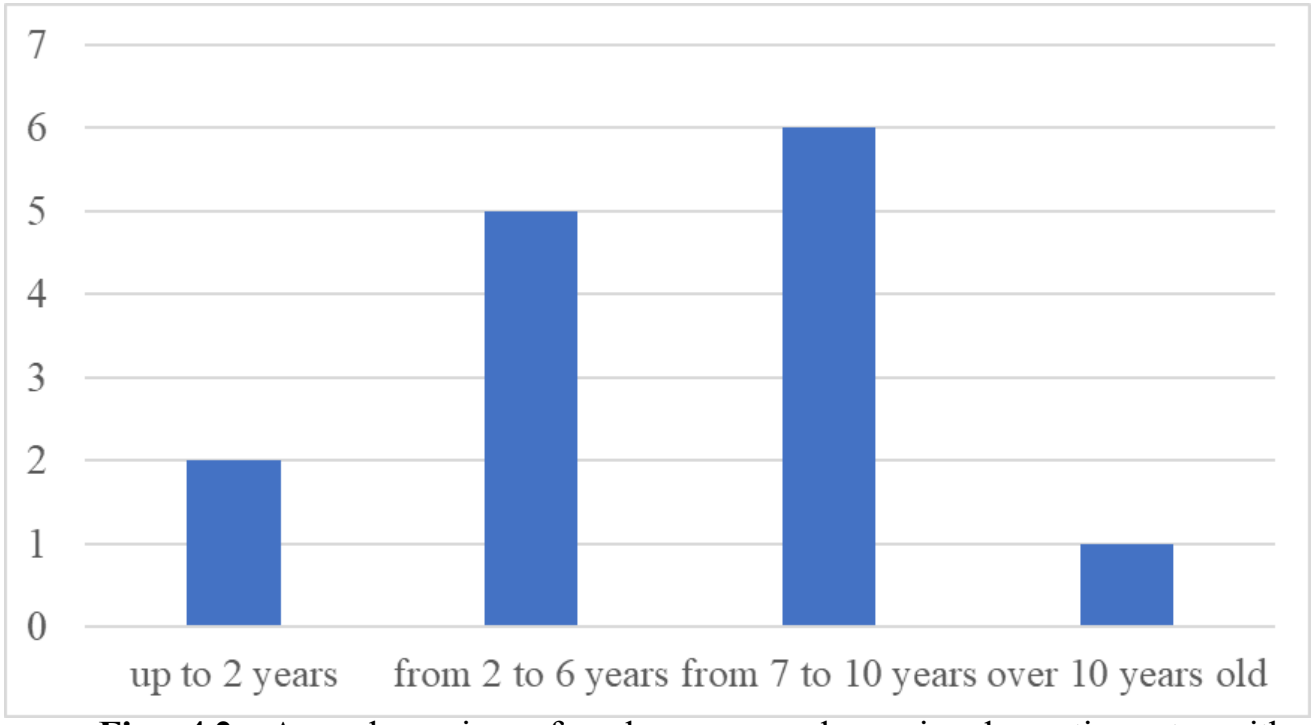

Fig. 4.2. Age dynamics of pulmonary oedema in domestic cats with cardiomyopathy, n=58

The results of studies on the breed predisposition of cats to cardiomyopathy indicate that a significant percentage of this pathology is registered in Scottish Fold

(36.2%) and British Shorthair (29.3%) breeds (Fig. 4.3).

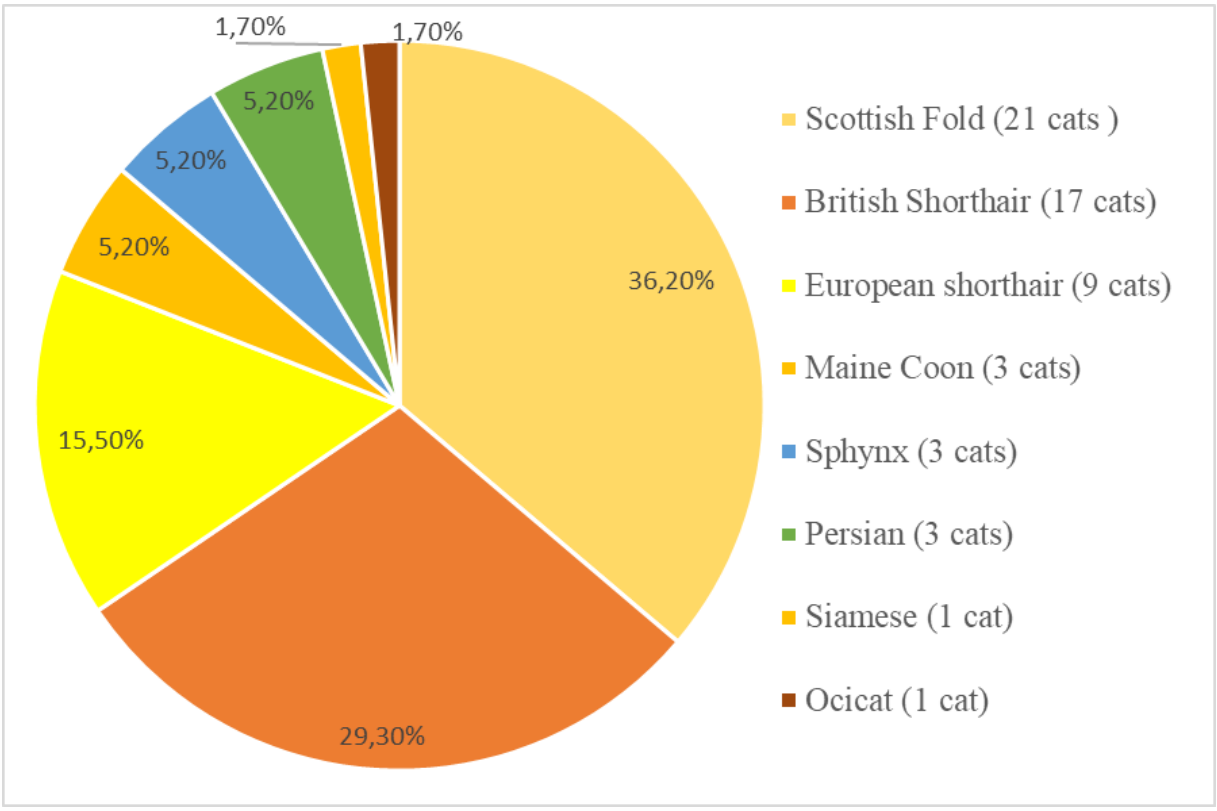

Fig. 4.3. Susceptibility of cats of certain breeds to cardiomyopathy, n=58

Therefore, if a domestic cat has shortness of breath and increased respiratory rate, cardiomyopathy may be suspected, but additional tests are required to confirm the diagnosis.

At the same time, blood test results show that the difference in indicators between cats with cardiomyopathy and clinically healthy cats is insignificant and not reliable. This indicates that blood tests are ineffective for diagnosing cardiomyopathy in domestic cats. However, cats with cardiomyopathy tend to have a 14.2% increase in red blood cell count compared to clinically healthy animals in the control group, which is a sign of hypoxia that can be observed in heart disease [17].

In addition, there is a 22.64% increase in serum creatinine concentration in cats with cardiomyopathy and a 1.22-fold increase in creatinine concentration and a 1.51-fold increase in urea concentration in cats with hypertrophic cardiomyopathy compared to clinically healthy animals in the control group (Fig. 4.4), which with high reliability confirms the presence of hyperazotemia and chronic kidney disease in sick animals, which can be considered an etiological factor of hypertrophic changes

in the myocardium in domestic cats, i.e., secondary hypertrophic cardiomyopathy in animals of this species (Fig. 4.4). The proportion of cats with hyperazotaemia in chronic kidney disease was 13.8% of the total number studied.

The study of troponin I concentration in blood serum allows determining the degree of cardiomyocyte damage in domestic cats with cardiomyopathy and differentiating the primary form of cardiomyopathy from the secondary one.

The results of our studies indicate that in the serum of domestic cats with cardiomyopathy, both elevated and normal concentrations of troponin I can be observed compared to clinically healthy animals (Fig. 4.5).

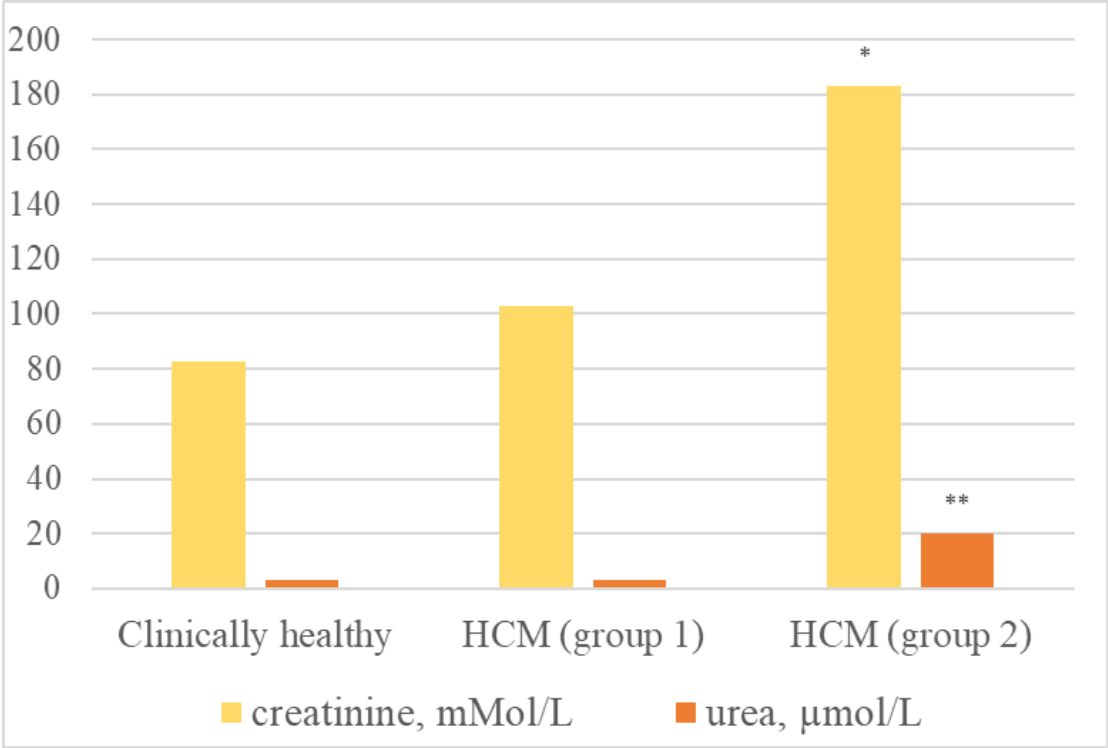

Fig. 4.4 Concentration of creatinine and urea in the blood serum of domestic cats with cardiomyopathy, M±t, n=5

Note: * $p<0.001$, ** $p<0.01$ compared to clinically healthy cats

The literature indicates that in primary, genetically determined HCM, the concentration of troponin I in the blood serum of animals should be constantly elevated over time, as the process of myocardial damage in this case is ongoing [175].

In our study, cats with cardiomyopathy in the second experimental group had normal

serum troponin I levels compared to the control group and reference data. This indicates that there is no cardiomyocyte damage in animals in this group, but the aetiological factor led to the development of a secondary form of hypertrophic cardiomyopathy.

A significant increase in the concentration of cardiac troponin I in the serum of domestic cats to 1.64 ng/ml (Fig. 4.5) was also found, indicating a very high degree of myocardial damage. The data obtained suggest the presence of myocarditis in cats with extremely high levels of cardiac troponin in the blood serum.

Thus, determining the concentration of cardiac troponin I in the serum of domestic cats with cardiomyopathy makes it possible to establish the severity and prognosis of the disease, as well as helps in the selection of therapeutic agents for the treatment of animals with this pathology.

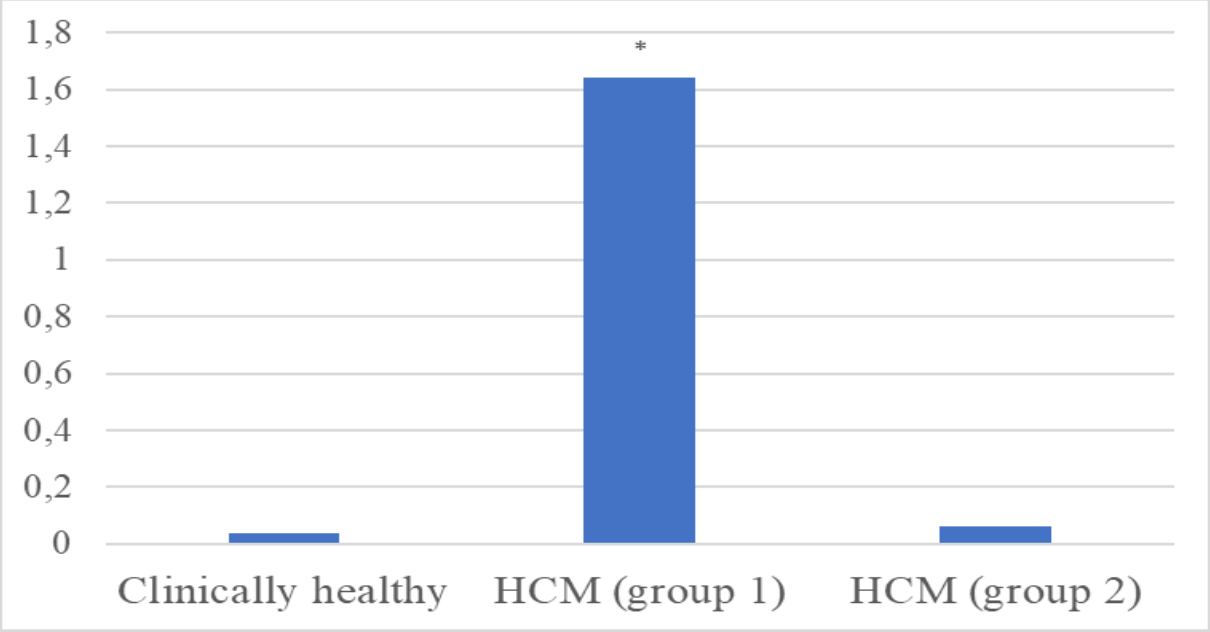

Fig. 4.5. Concentration of cardiac troponin I in the blood serum of domestic cats with cardiomyopathy, M±m, n=9

Note: * p<0,001 compared to clinically healthy cats

According to the results of X-ray studies of the chest cavity of experimental cats, no reliable differences were found that would confirm the diagnosis of cardiomyopathy in domestic cats (Fig. 4.6). At the same time, X-ray studies indicate a 20% increase in the Buchanan (Viskapap) coefficient and a 38% increase in the

cardiothoracic index. However, chest X-ray imaging allows visualisation of signs of congestion in the lungs and signs of pulmonary oedema in cats with cardiomyopathy, making it possible to assess the severity and predict the course of cardiomyopathy, as well as to provide the necessary treatment for animals with this pathology.

Conducting electrocardiographic studies on animals in general, and domestic cats in particular, has certain difficulties that may be caused by the restlessness, stress, and anxiety that animals experience during the studies. In some cases, this may make it impossible to use electrocardiography to study animals or, if used, may lead to distorted ECG data.

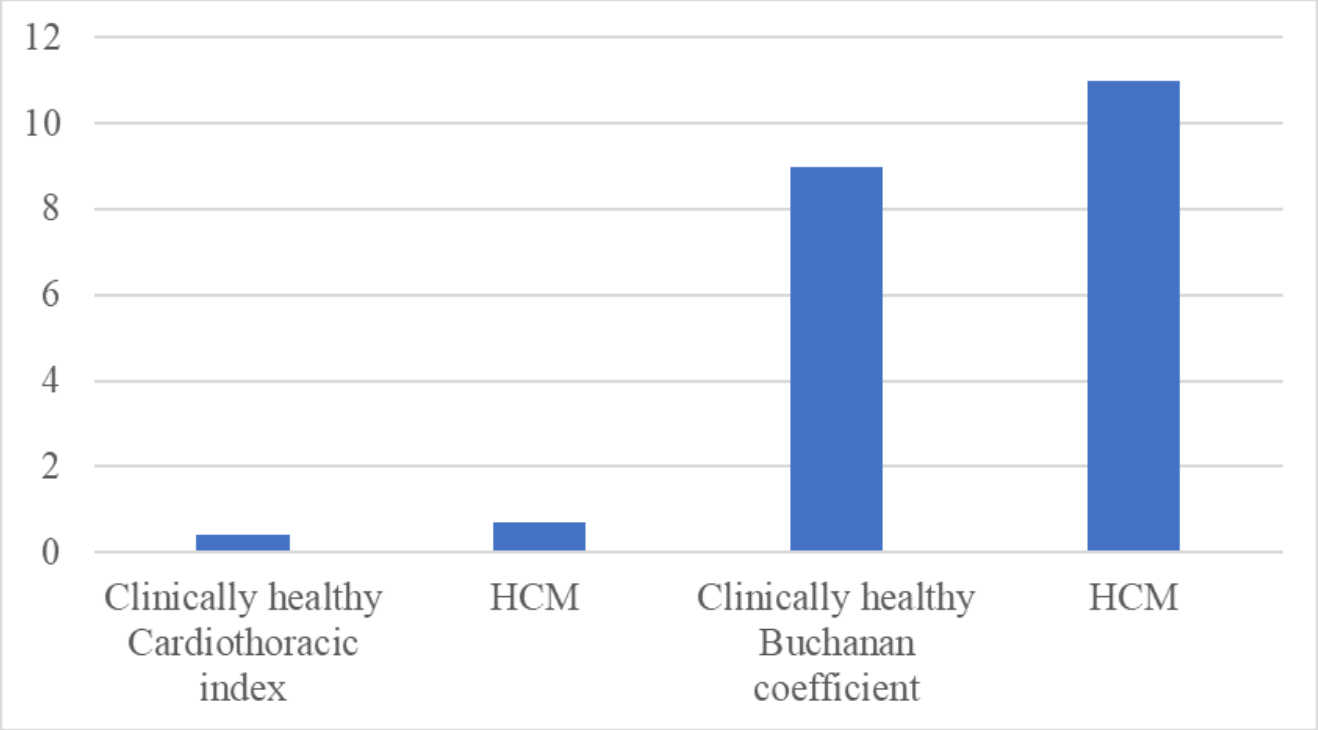

Fig. 4.6. X-ray examination data: comparative assessment of cardiotoracic index and Buchanan coefficient (Viskapap) in healthy cats and cats with cardiomyopathy, M±m, n=5

According to electrocardiography data, we did not find a significant difference between clinically healthy cats and cats with cardiomyopathy. At the same time, a decrease in the average voltage of the P and K waves was found in both clinically healthy cats and cats with cardiomyopathy compared to the reference data in the literature (Fig. 4.7).

In addition, sinus tachycardia was observed in clinically healthy cats and cats

with cardiomyopathy compared to reference data from the literature (Fig. 4.8). It should be noted that sinus tachycardia is a manifestation of increased myocardial excitability as a physiological response in animals in a state of anxiety and stress [40].

Thus, based on the results obtained, it can be concluded that the electrocardiography method does not provide reliable data for the diagnosis of cardiomyopathies in domestic cats, since it gives abnormal data, which, using this method, we also obtained in the case of studies of clinically healthy cats.

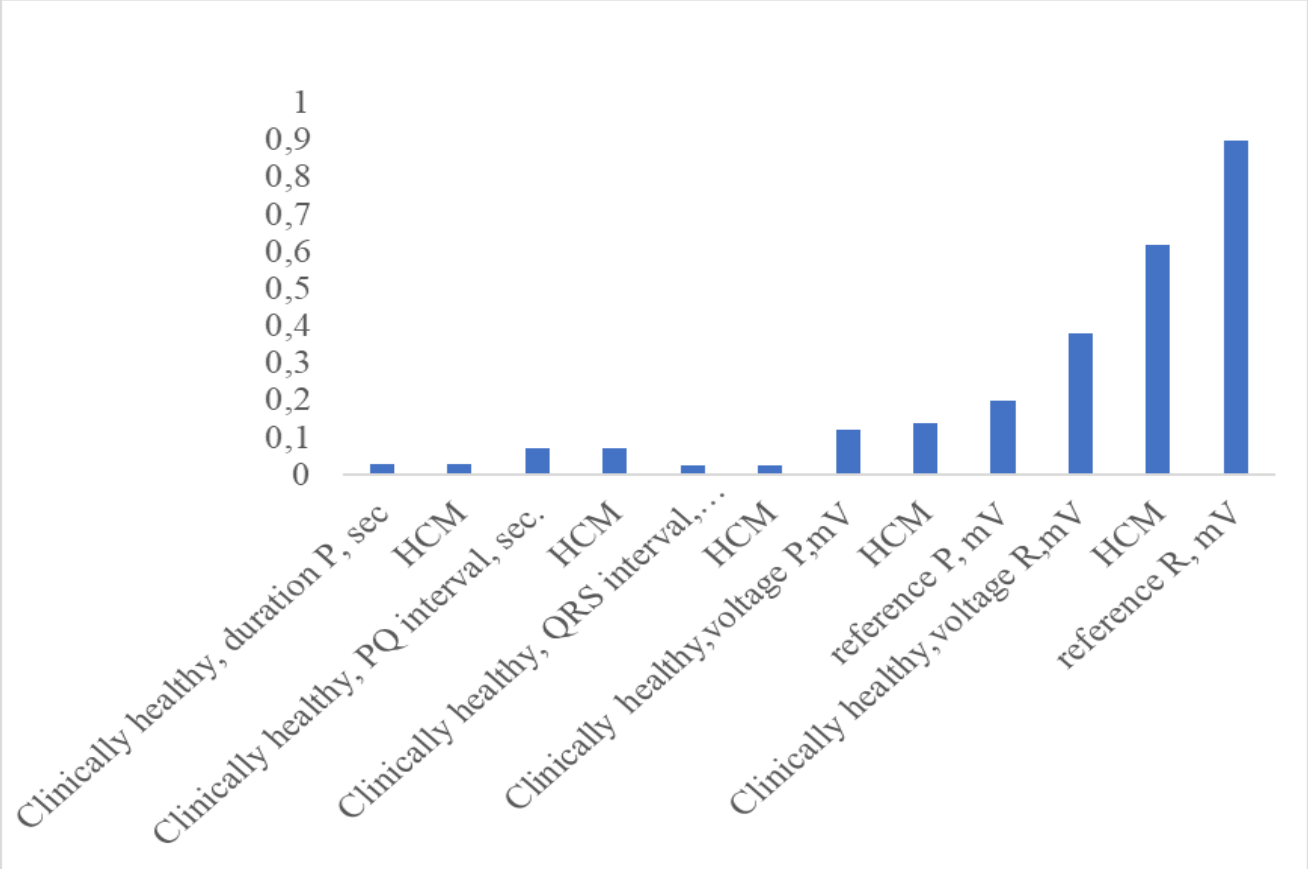

Fig. 4.7. Electrocardiogram parameters of a domestic cat with cardiomyopathy, M±m, n=5.

Studies have shown that it is possible to clearly visualise and record pathological abnormalities in the anatomical structure of the heart, reliably diagnose and classify existing cardiomyopathy in sick animals using the echocardiography method.

Thus, echocardiography has established that in cats with HCM and RCM, there is a significant thickening of the interventricular septum and left ventricular wall, while in DCM, this indicator remains within normal limits or does not show a reliable difference (Fig. 4.8). The sizes of the ventricles and atria also undergo significant changes in various forms of cardiomyopathy (Fig. 4.9).

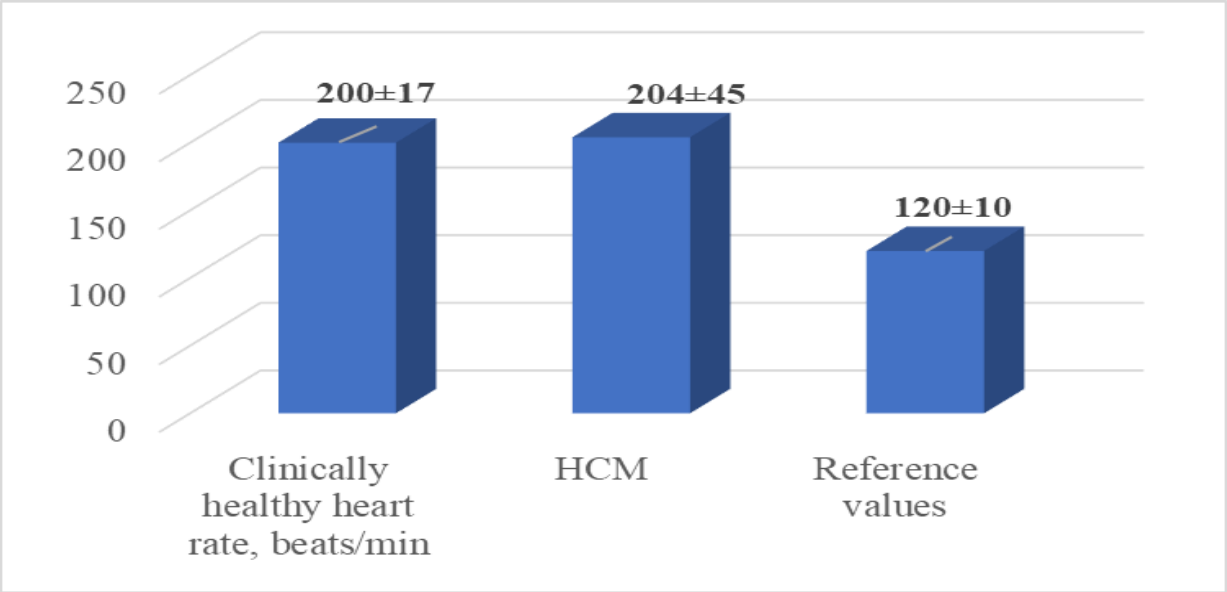

Fig. 4.8. Heart rate in a domestic cat during electrocardiographic examination, M±m, n=5

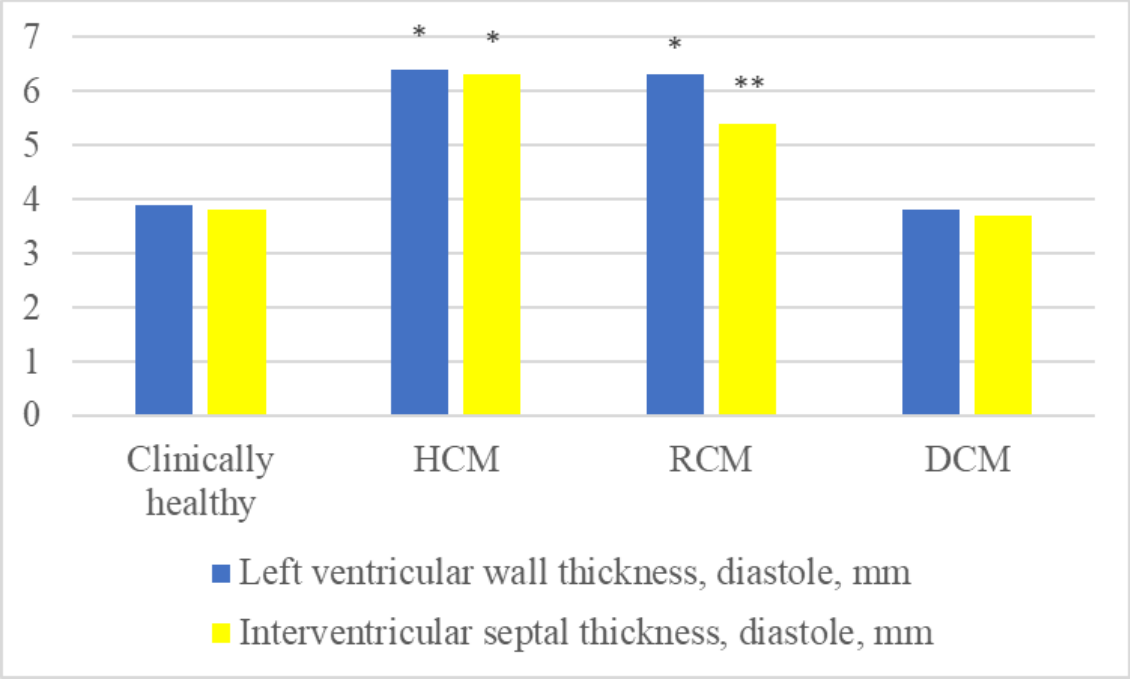

Fig. 4.9. The thickness of the interventricular septum and the wall of the left ventricle in diastole of the heart in different types of cardiomyopathies in a domestic cat, M±m, n=5

Notes: * p≤0.001, ** p≤0.01 compared with clinically healthy cats

Thus, in domestic cats with HCM, there is a decrease in the systolic and diastolic size of the left ventricle, which is associated with thickening of the interventricular septum and the wall of the left ventricle into the ventricle cavity itself. In RCM and DCM in domestic cats, the dimensions of the left ventricle are significantly increased.

Research results indicate that in all three forms of cardiomyopathy in domestic cats, there is a significant enlargement of the left atrium. In the case of RCM, it is most pronounced (Fig. 4.9). DCM in domestic cats is also characterised by a significant enlargement of the right ventricle ($p<0.05$) and right atrium ($p<0.01$) compared to clinically healthy animals.

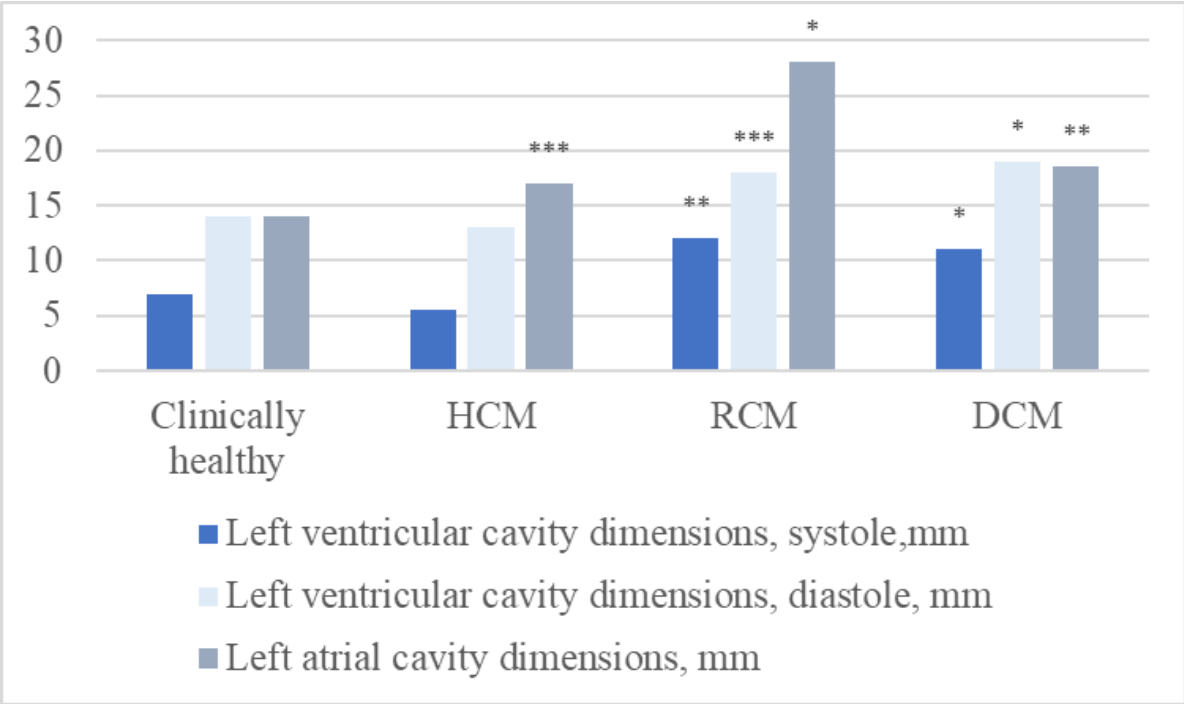

Fig. 4.10. Dimensions of the cavities of the left ventricle and left atrium of a domestic cat in different forms of cardiomyopathies, M±m, n=5

Notes: * $p\leq0.05$, ** $p\leq0.01$, *** $p\leq0.001$ compared with clinically healthy cats

In all cases of RCMD and DCMD in domestic cats, mitral valve insufficiency is recorded, which is explained by a significant enlargement of the left atrium. In DCM in domestic cats, tricuspid valve insufficiency is also noted. However, not all cats with cardiomyopathy according to HCM have mitral valve insufficiency and, accordingly, enlargement of the left atrium. The more the left atrium expands, the

more congestive phenomena develop in the pulmonary veins, and the greater the risk of pulmonary oedema and/or thrombus formation in the left atrial cavity.

The course of intermediate cardiomyopathy in domestic cats has its own characteristics. In this form of cardiomyopathy, HCM is diagnosed during the first echocardiographic examination of sick cats, and DCM is diagnosed during repeated echocardiographic examinations (Fig. 4.10 and Fig. 4.11).

A period of 5-7 months may be sufficient for the remodelling of the heart from HCM to DCM in the intermediate form of cardiomyopathy in domestic cats.

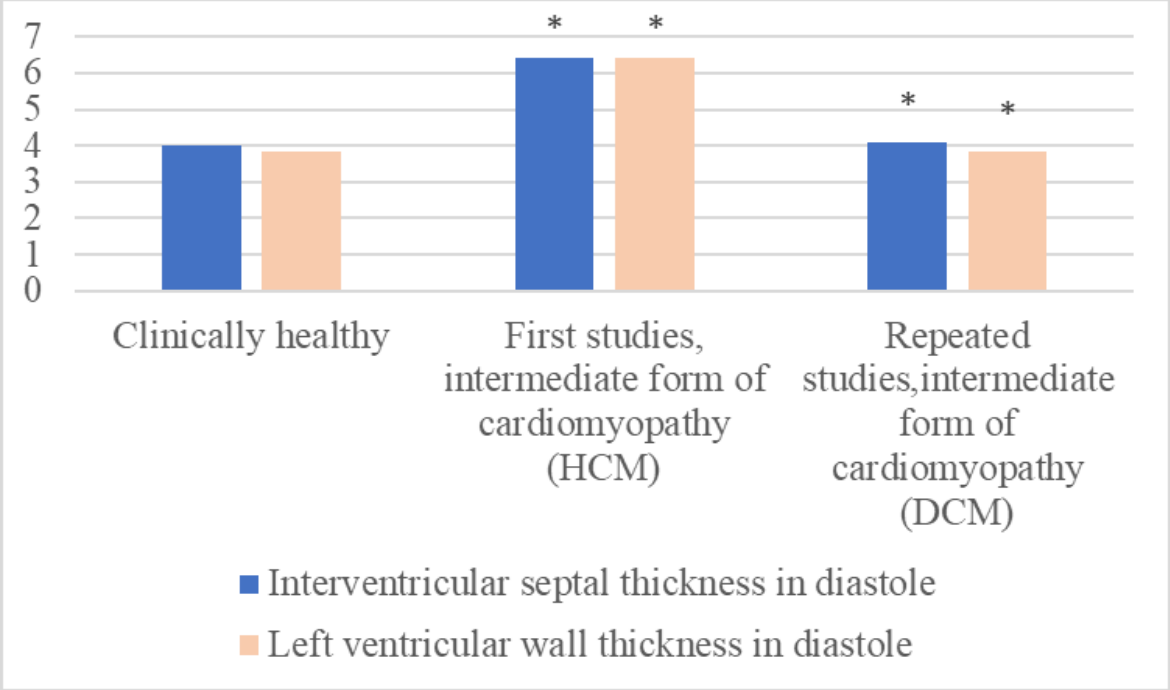

Fig. 4.11. Thickness of the interventricular septum and left ventricular wall during cardiac diastole in a domestic cat with intermediate cardiomyopathy, M±tm, n=5

Notes: *p<0.001 compared to clinically healthy cats

The formation of excessive fibrous tissue (proliferation of loose connective fibrous tissue) in the intercellular space of the myocardium of domestic cats with RCM leads to a decrease in the latter's compliance to contraction and relaxation, which explains the characteristic echocardiographic picture in this form of cardiomyopathy (Fig. 4.12).

During echocardiographic examination of cats using RCM, we found thickening of the interventricular septum and left ventricular wall, but to a lesser

extent than with HCM, and simultaneous enlargement of the left ventricular cavity and significant enlargement of the left atrial cavity [89].

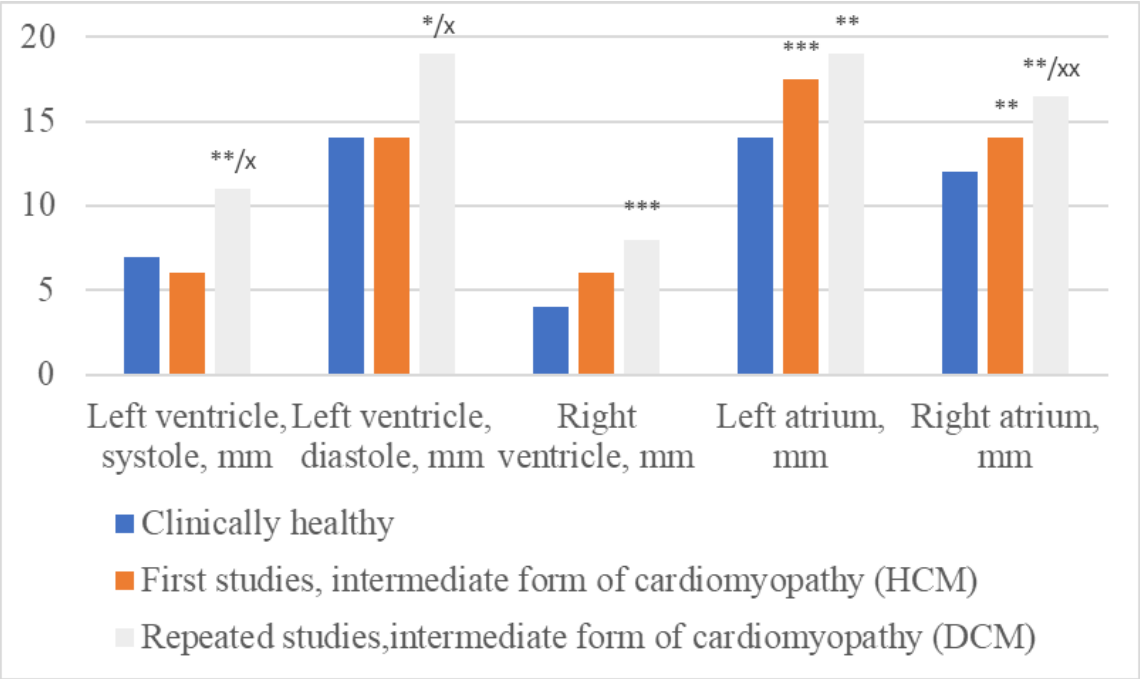

Fig. 4.12. Dimensions of the heart chambers of a domestic cat with intermediate cardiomyopathy, M±m, n=5

Notes: *p<0.001, **p<0.01, ***p<0.05 compared to clinically healthy cats; p<0.01, p<0.05 compared to the results of the first echocardiographic examination

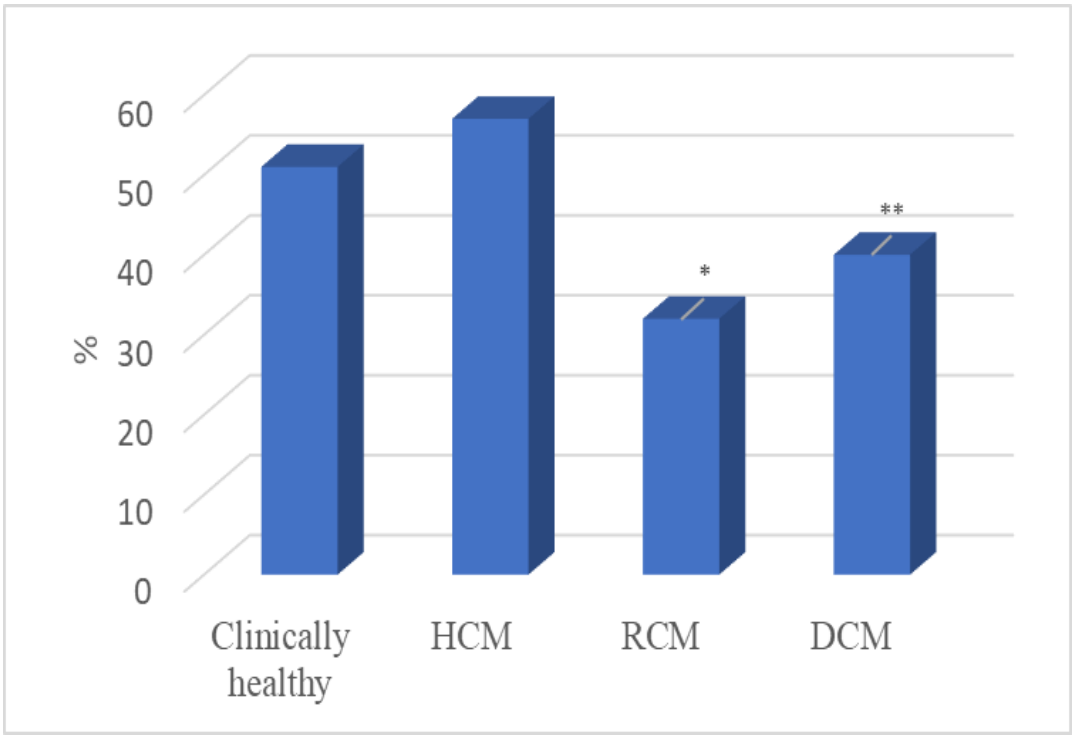

Fig. 4.13. Contractility index (CI) of the myocardium of domestic cats with different forms of cardiomyopathy, M±m, n=5

Notes: * p<0.01, **p<0.05 compared to clinically healthy cats

The decrease in myocardial compliance according to RCM is explained by a significant decrease in the contractility index ($p<0.01$). A significant decrease in SI is also observed in DCM ($p<0.05$), but to a lesser extent, which is explained by the overflow of the heart chambers in terms of volume in this form of cardiomyopathy. In HCM, on the contrary, the myocardial contractility index increases compared to clinically healthy animals, but does not reach the significance level of $p<0.05$.

The hypertrophic form of cardiomyopathy can also manifest itself in an asymmetrical form: thickening of individual parts of the interventricular septum (most often the basal part), the wall of the left ventricle or the papillary muscles of the left ventricle. The detection of such changes depends on the experience of the veterinarian and their understanding of the essence of the echocardiography method.

In our opinion, the development of restrictive cardiomyopathy in domestic cats can be considered a consequence of secondary hypertrophic cardiomyopathy, when the process of proliferation of loose fibrous connective tissue occurs more intensively in the intercellular space of the myocardium.

One of the causes of hypertrophic changes in the myocardium may be subclinical myocarditis, the aetiological factor of which in domestic cats is coronavirus infection and viral panleukopenia [94].

The data established during our dissertation research, which hypothetically confirm the presence and role of myocarditis in the development of secondary cardiomyopathies in domestic cats, especially DCM, are: a significant increase in the concentration of cardiac troponin I in the blood serum of sick animals; the presence of lymphohistiocytic infiltration in the intercellular space of the myocardium of cats that died from cardiomyopathy; confirmation of the aetiological role of viral infections in diagnosed cardiomyopathy in domestic cats; remodelling of the heart in cats with coronavirus infection or panleukopenia to DCM in the intermediate form of cardiomyopathy; proliferation of loose connective fibrous tissue (fibrotic changes) in the myocardium of cats with cardiomyopathy, as a possible consequence of chronic myocarditis.

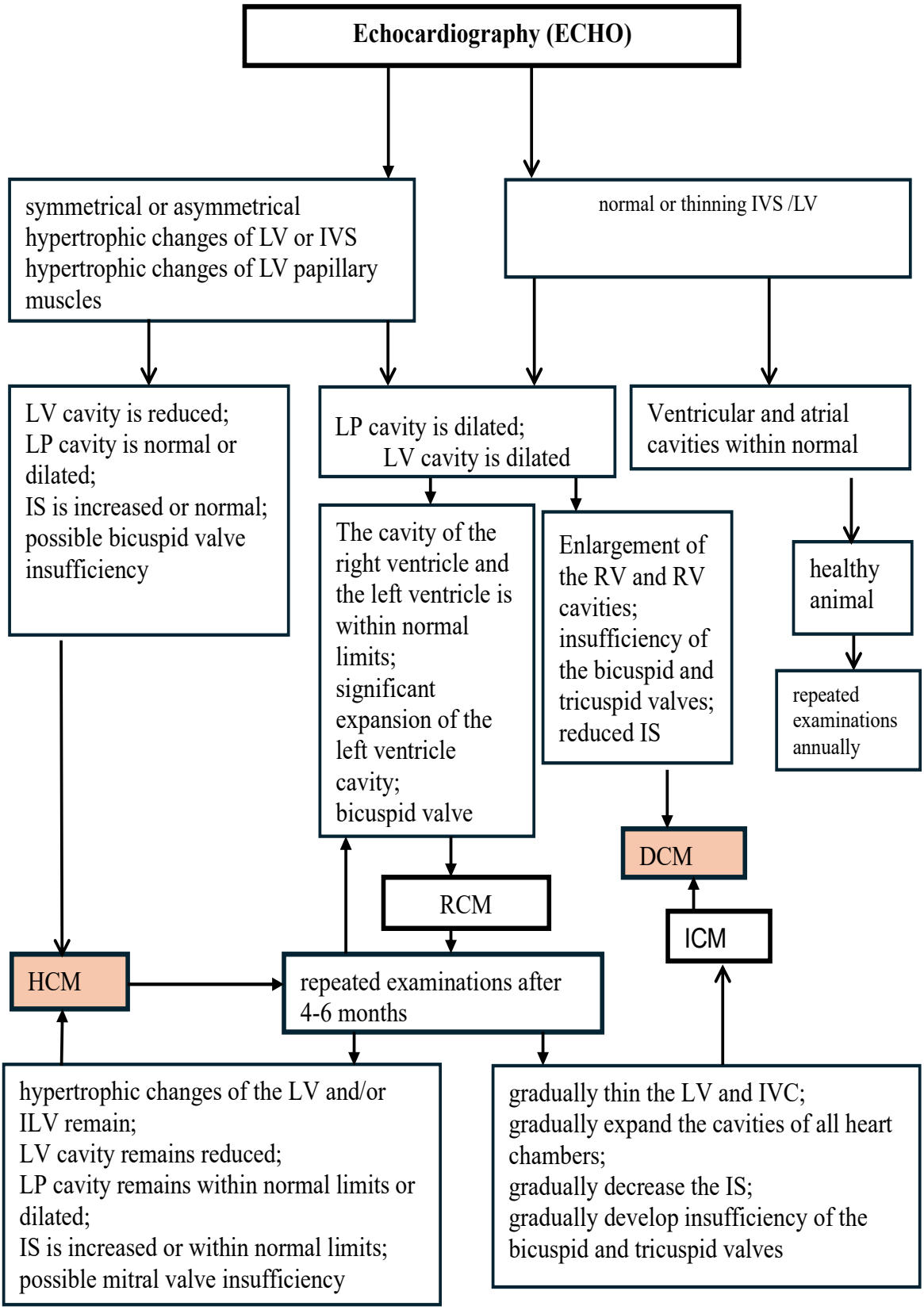

Fig. 4.14. Scheme of differential ultrasound diagnosis of different types of cardiomyopathies in a domestic cat.

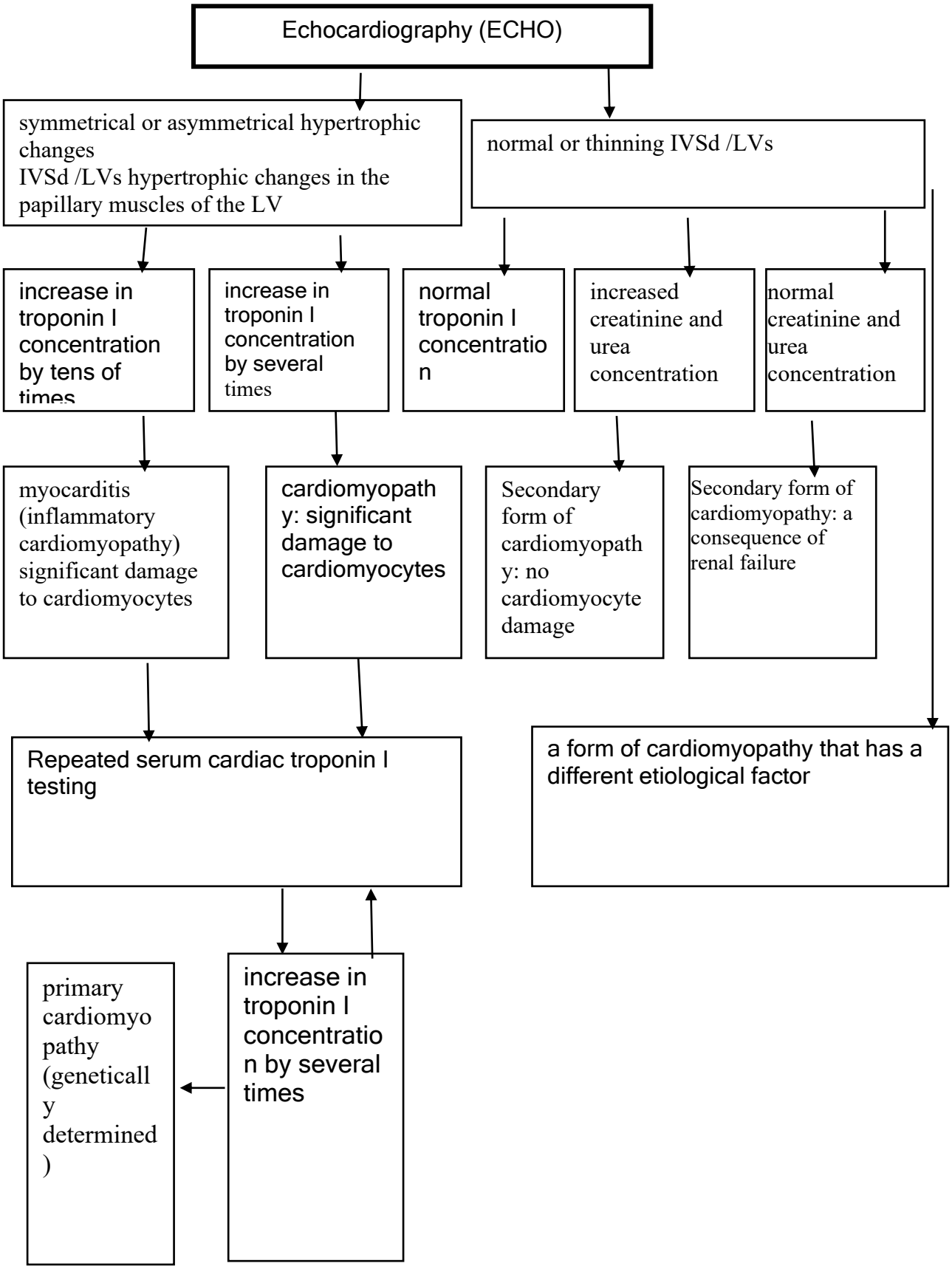

Fig. 4.15. Scheme of laboratory tests for cardiomyopathy in a domestic cat

Thus, according to the results of our studies, the main method of diagnosis and classification of forms of cardiomyopathy in domestic cats is ultrasound examination of the heart (echocardiography).

Laboratory diagnostic methods (blood tests) only in rare cases help to establish the aetiology of cardiomyopathy in domestic cats (fig. 4.16).

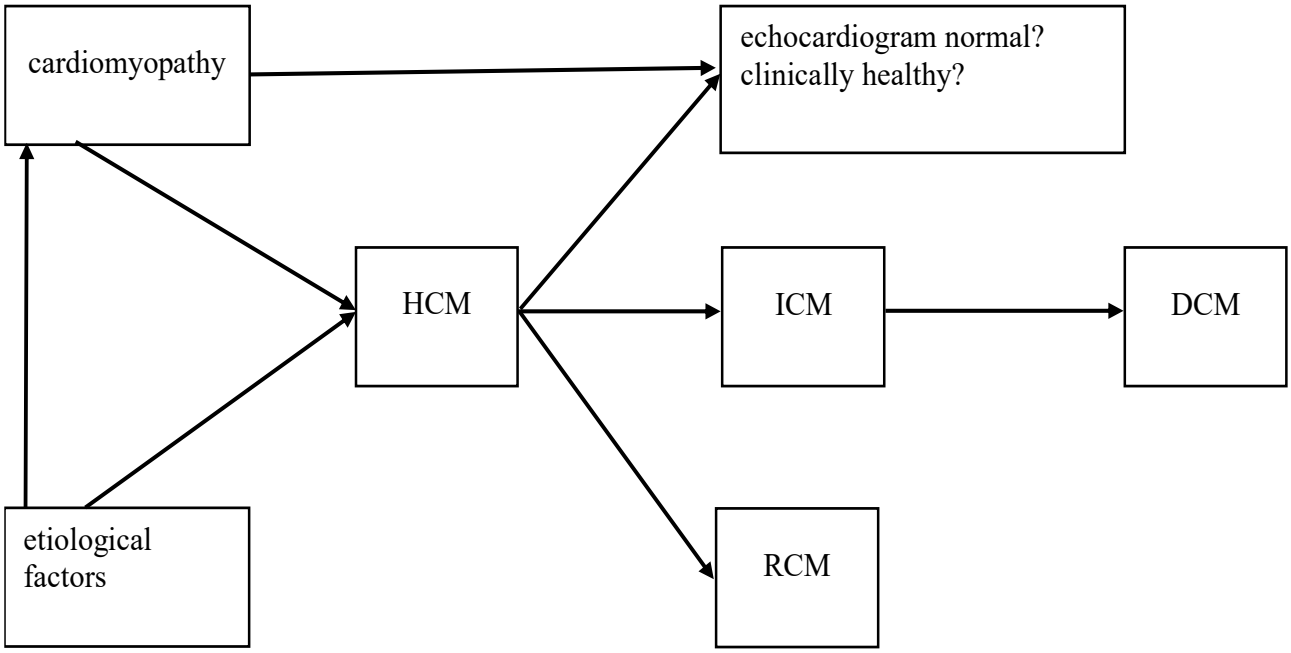

Fig. 4.16 Diagram of diagnostics cardiomyopathy for cats

If cardiomyopathy is confirmed, further echocardiography tests of the domestic cat's heart should be performed at intervals of 5-7 months. In the absence of echocardiographic signs of cardiomyopathy in domestic cats, subsequent routine ultrasound examinations of the heart can be performed every 1-2 years.

Special attention should be paid to laboratory determination of troponin I concentration in the blood serum of cats diagnosed with cardiomyopathy. In our opinion, persistently elevated troponin I levels in the blood serum of cats with HCM during repeated studies may indicate the genetic determinism of this disease. Cases where troponin I levels in the serum of sick domestic cats are within the normal range can be considered a manifestation of secondary cardiomyopathy in these animals. We suggest that a tenfold increase in troponin I concentration in the serum of domestic

cats should be considered a manifestation of inflammatory cardiomyopathy or myocarditis, depending on the echocardiographic picture of the animal's heart.

Instrumental diagnostic methods such as radiography and electrocardiography allow the severity of the disease to be assessed.

Finally, the data from the medical history and clinical examination of animals make it possible to suspect the presence of cardiomyopathy in domestic cats, especially given the predisposition of certain breeds to this pathology.

Thus, cardiomyopathy in domestic cats can have hypertrophic, restrictive, dilated, and intermediate forms of manifestation according to phenotype. The developed schemes allow diagnosing, differentiating, and classifying the form of cardiomyopathy in domestic cats.

Hypertrophic cardiomyopathy in domestic cats is diagnosed based on the following echocardiographic signs: left ventricular myocardial hypertrophy of 65.4% and interventricular septal hypertrophy of 72.5% in diastole ($p<0.001$), and/or asymmetric thickening of the basal part of the interventricular septum ($p<0.05$); dilatation of the left atrium by 17.1% ($p<0.05$); the dimensions of the left ventricle are normal or tend to decrease by 16.42% in systole and by 5.2% in diastole; in some cases, hypertrophic changes in the papillary muscles of the left ventricle, fluid in the pericardial cavity, mitral valve insufficiency, tendency to increase the contractility index (myocardial hyperkinesis); increased echogenicity of the left ventricular wall and interventricular septum, which is a sign of fibrotic changes.

Restrictive cardiomyopathy in domestic cats is diagnosed based on the following echocardiographic signs: left ventricular myocardial hypertrophy by 60.5% in diastole ($p<0.001$) and interventricular septum by 47.8% in diastole ($p<0.01$); dilatation of the left ventricle cavity by 74.6% in systole ($p<0.01$) and by 22.1% in diastole ($p<0.05$); marked enlargement of the left atrium by 97.6% ($p<0.001$); presence of thrombi in the left atrium, which may not be present in all cases; a 36.9% decrease in the contractility index (myocardial hypokinesis); a marked increase in the echogenicity of the left ventricular myocardium and/or interventricular septum, which is a sign of fibrotic changes; the presence of fluid in the pericardial cavity,

which is not visualised in all cases.

Dilated cardiomyopathy in domestic cats is diagnosed based on the following echocardiographic signs: left ventricular dilatation in systole by 69.9% and in diastole by 37.8% ($p<0.001$); right ventricular dilatation by 74.9% ($p<0.05$); dilatation of the left atrium by 35.1% and the right atrium by 46.8% ($p<0.01$); a 21.6% decrease in the myocardial contractility index ($p<0.05$); the presence of moderate to severe mitral and tricuspid valve insufficiency.

The intermediate form of cardiomyopathy in domestic cats is diagnosed by changes in echocardiography parameters during repeated examinations of the hearts of sick animals. It is characterised by a change in signs from hypertrophic cardiomyopathy, diagnosed during the first echocardiographic examination, to dilated cardiomyopathy, diagnosed during repeated echocardiographic examinations in domestic cats based on the following echocardiographic signs: a 36.2% thinning of the left ventricular wall in diastole and a 41.9% thinning of the interventricular septum in diastole, compared with the data for the same cats in the first examination ($p<0.001$); dilatation of the left ventricle in systole by 79.4% ($p<0.01$) and in diastole by 34.3% ($p<0.01$); dilatation of the right atrium by 20.1% ($p<0.05$). A period of 5-7 months is sufficient for the remodelling of the heart from a hypertrophic to a dilated form in the intermediate form of cardiomyopathy in domestic cats. The dilated form of cardiomyopathy, as manifested by the intermediate form of cardiomyopathy, is considered the final stage in the diagnostic classification.

The causes of secondary cardiomyopathy in domestic cats in the city of Kyiv are hyperazotaemia in chronic kidney disease (13.8% of cases) and animal diseases caused by parvoviruses and coronaviruses (12.1% of cases). In cases of hyperazotaemia in the blood serum of domestic cats with cardiomyopathy, the concentration of creatinine increases significantly by 2.2 times ($p<0.001$) and urea by 2.5 times ($p<0.01$).

The main pathogenetic factor in cardiomyopathy in domestic cats is structural and anatomical changes in the heart caused by an aetiological factor. An increased concentration of cardiac troponin I in the blood serum of a domestic cat with

cardiomyopathy by 12.5 times ($p<0.001$) allows the degree of myocardial damage to be assessed.

Domestic cats can develop cardiomyopathy at any age. Most often, cats of the Scottish Fold (36.2%) and British Shorthair (29.3%) breeds suffer from cardiomyopathy; males suffer from cardiomyopathy almost 2 times more often (65.5%) than females (34.5%). Asymptomatic cardiomyopathy is characteristic of 51.7% of cats, while the main clinical manifestations of cardiomyopathy in 48.3% of animals are shortness of breath (32.8%), pulmonary oedema (25.9%) and aortic thromboembolism (5.2%).

According to the results of pathohistological studies of the myocardium of cats that died with a diagnosis of hypertrophic cardiomyopathy, the following was established: a combination of atrophy, dystrophy and hypertrophy of cardiomyocytes; the presence of lymphohistiocytic infiltration and foci of loose connective fibrous tissue growth in the intercellular space. Lymphohistiocytic infiltration is an important pathognomonic confirmation of infectious myocarditis. One of the aetiological factors that can cause such myocarditis in domestic cats is coronavirus infection and viral panleukopenia.

A prerequisite for reliable diagnosis and determination of the form of cardiomyopathy in domestic cats is repeated ultrasound examinations of the heart at intervals of 4-6 months. The first echocardiographic examinations of domestic cats should begin at 6 months of age.

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