Anaesthesia in patients with cardiac disease often poses a challenge for the veterinarian. Due to cardiovascular dysfunction, these patients have an increased anaesthetic risk. This review article summarizes the most important pathological alterations with cardiac disorders in dogs and cats and their relevance for the anaesthetist. Pre-anaesthetic evaluation, premedication, induction and maintenance of anaesthesia as well as monitoring of anaesthetised patients and possible complications are also discussed.

Keywords: anaesthesia, cardiac disease, dog, cat, monitoring, blood pressure

This paper originally appeared in:
*Wiener Tierärztliche Monatsschrift – Veterinary Medicine Austria* 99 (2012)

**Abbreviations used:**
ACE = angiotensin converting enzyme;  
AVA = Association of Veterinary Anaesthetists;  
DCM = Dilated Cardiomyopathy;  
CRI = constant rate infusion;  
ETCO2 = end-tidal carbon dioxide;  
HCM = Hypertrophic Cardiomyopathy;  
HDO = High-Definition Oscillometry;  
SACHC = International Small Animal Cardiac Health Council;  
LiDCO = Lithium Dilution Cardiac Output

**Introduction**

For many veterinary practitioners, anaesthesia in cardiac patients represents both a challenge and a psychological barrier. This is why the majority of such patients are referred to specialist clinics. However, a sound knowledge of the pathophysiology of heart disease, good perioperative monitoring and management as well as appropriate medication will enable every small animal practitioner to carry out anaesthesia in patients with cardiac disease.

Technically speaking, the left ventricle of the heart can be considered as a “pressure pump”, as it pumps the blood into a high pressure system, while the right ventricle works as a “volume pump” pumping the blood into a low pressure system. This explains why the left ventricle tolerates high pressures without major problems (e.g. in subaortic stenosis or systemic hypertension), while the right ventricle is well able to compensate for volume increases (e.g. in tricuspid regurgitation).
There are some basic terms, which are important in order to understand the pathophysiology of heart disease, e.g. preload and afterload. Preload is the end diastolic volume of the heart and is basically determined by the venous return to which the hearts’ pumping capacity automatically adapts, although within physiological limits. In the healthy heart, increased venous return also increases the ejection volume of the heart. In the pathologically altered heart, an increased preload may initially contribute to maintenance of cardiac output, but in the long run, eccentric hypertrophy, myocardial remodelling, apoptosis of the myocardial cells and general worsening of the cardiac disease occur [BORGARELLI, 2005]. Cardiac afterload is the impedance to ventricular emptying presented by aortic pressure, against which the cardiac muscle has to pump blood into the arteries (aorta and pulmonary artery). Elevated systemic vascular resistance leads to increased afterload and, as a consequence, to an increase in myocardial strain and oxygen consumption. Chronically increased afterload (e.g. due to systemic hypertension or stenosis of arteries near to the heart) causes concentric hypertrophy [BORGARELLI, 2005]. Contractility of the heart is defined as the intrinsic ability of the myocardium to contract. It can be increased and decreased, respectively, by adapting to the actual preload and afterload as well as by positive or negative inotropic drugs (see Table 1).

Maintenance of blood pressure is necessary to ensure peripheral perfusion. The arterial blood pressure is closely related to the stroke volume, the heart rate and the vascular resistance.

**Perianaesthetic considerations for animals with cardiac disease**

To perform anaesthesia in cardiac patients, some basic preconditions are required, i.e. establishing an intravenous access, use of an oxygen supply, equipment for intubation and ventilation, appropriate drugs for emergencies and devices for monitoring cardiovascular function (electrocardiography [ECG] machine, blood pressure unit, pulse oximeter, capnograph) [SKARDA et al., 1995a; HARVEY and ETTINGER, 2007]. Any excitement of the patient must be avoided. In some cases, the intramuscular administration of a sedative drug before establishing the venous access may reduce the stress for the patient (PASCOE, 2005).

Preoxygenation before inducing anaesthesia reduces myocardial hypoxia and avoids apnoea during induction. During maintenance of anaesthesia, oxygen supply (via oxygen tubing, oxygen mask or laryngeal mask) prevents hypoxia due to hypoventilation (ERHARDT, 2004). In order to keep the duration of anaesthesia as short as possible, the animal must be dosed with a drug that is able to reduce both the heart rate and the contractility, which is achieved with the use of non-depolarising neuromuscular blocking drugs (see Table 1).

### Table 1: Cardiovascular effects of some important drugs used for anaesthesia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Heart rate</th>
<th>Inotropy</th>
<th>Cardiac output</th>
<th>Vascular resistance</th>
<th>Arterial blood pressure</th>
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</thead>
<tbody>
<tr>
<td>Acepromazine</td>
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<td>Midazolam</td>
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<td>Butorphanol</td>
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<td>Buprenorphine</td>
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<td>Methadone</td>
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<td>Fentanyl</td>
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<td>Xylazine</td>
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<td>Medetomidine</td>
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<td>Ketamine</td>
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<td>Propofol</td>
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<td>Thiopental</td>
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<td>Alphaxalone</td>
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<td>Etomidate</td>
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<td>Isoflurane</td>
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<td>Sevoflurane</td>
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</tr>
</tbody>
</table>

↓: Decrease  ↑: Increase  ↓↓: pronounced decrease  ↔: no influence  ↑↓: initially increase, then decrease
short as possible, all preparations for surgery should be concluded by the time of induction of anaesthesia. In the cardiac patient, anaesthesia must aim at maintaining a stable cardiovascular system. Both heart rate and blood pressure should show only minimum variations. Due to cardiovascular depression induced by many anaesthetic drugs, deviations of heart rate and blood pressure values from those of unanaesthetized animals are unavoidable, but should be as minimal as possible (HARVEY and ETTINGER, 2007). It is therefore important to assess the individual baseline values during preanaesthetic examination.

Preanaesthetic examination - Risk assessment

A thorough preanaesthetic examination is of utmost importance, when it comes to the safety of the patient. Special attention should be paid to the parameters of the cardiovascular system: pulse rate and quality, colour of mucous membranes and capillary refill time; in addition, auscultation of heart and lungs, control of absence of pulse deficits and blood pressure measurement have to be performed. Relevant predisposing diseases should always be kept in mind, particularly in older animals as well as in certain breeds (e.g. Great Dane, Boxer; Maine Coon). The absence of clinical signs is no guarantee for the absence of cardiovascular disease. In a study performed in cats with a cardiac murmur but without any clinical symptoms, ultrasound examination of the animals revealed that 53 % of these cats suffered from heart disease [NAKAMURA et al., 2011].

The anaesthetic risk is increased for patients with cardiac disease, even if they are able to compensate for the heart failure; the risk is increased even more in animals with decompensated heart failure [SKARDA et al., 1995b; POSNER, 2007]. In cases of suspected cardiac disease, diagnostic ultrasonography should be performed to identify the type of disease and the degree of compensation [CLUTTON, 2007; HARVEY and ETTINGER, 2007]. Particularly for elective surgery, patients should be duly stabilized before the anaesthesia by administering appropriate drugs. Although patients have to be fasted before all kinds of surgical interventions under general anaesthesia, it is necessary and important to continue administering the prescribed drugs at the usual times in order to maintain effective blood levels of the agent [PASCOE, 2005].

Drugs and their effects on the cardiovascular system

Nearly all drugs used for anaesthesia have an effect on the cardiovascular system. While the healthy heart is able to tolerate these effects, pre-existing cardiac conditions may lead to acute decompensation and heart failure in these patients. Depending on the disease, an adequate anaesthetic protocol has to be chosen in order to keep the stress for the cardiovascular system as low as possible and, ideally, to provide additional support to the heart [HARVEY and ETTINGER, 2007. The most important cardiovascular effects of currently used anaesthetic drugs are summarized in Table 1.

Premedication

Phenothiazines

Acepromazine, a phenothiazine derivative psychotropic drug, is a frequently used sedative drug. It causes a dose dependent reduction of the stroke volume and the cardiac output. Due to alpha1-adrenergic blocking effects on the vascular walls, vasodilation occurs and arterial blood pressure sinks [FARVER et al., 1986]. Possible effects on the heart rate are discussed controversially in literature. LEMKE and TRANQUILLI (1994) as well as EBERSPÄCHER et al. (2005) reported a more or less constant heart rate when acepromazine was administered at moderate doses. According to ERHARDT et al. (2004) and PADDLEFORD and ERHARDT (1992a), a reflex increase in the heart rate was also observed. At very high doses (1 mg/kg), bradycardia and sinoatrial blocks may occur [LEMKEs and TRANQUILLI, 1994]. Acepromazine desensitizes the myocardium to the potentially arrhythmogenic effect of catecholamines. Due to its effect on the myocardial alpha1-receptors, it prevents the development of ventricular arrhythmias [LEMKES. TRANQUILLI, 1994].

Benzodiazepines like midazolam and diazepam hardly have any effect on the cardiovascular system, if administered at usual doses. Diazepam does not produce any clinically relevant alterations of heart rate, myocardial contractility, cardiac output and arterial blood pressure [JONES et al., 1979]. In the dog, midazolam may increase heart rate and cardiac output by 10-20 %, if administered at higher doses (0.25 – 1 mg/kg) [JONES et al., 1979].

Ketamine is a dissociative anaesthetic drug and stimulates the cardiovascular system by activating the...
sympathetic nerve system [LIN, 2007]. This exerts a positive inotropic effect on the myocardium, causing an increase of heart rate, blood pressure and cardiac output [PADDLEFORD and ERHARDT, 1992b]. It also increases myocardial oxygen consumption and vascular tone [ZSIGMOND et al., 1974].

**Opioids**

μ-agonists (e.g. methadone, morphine, fentanyl) increase the vagal tone, thereby causing a dose dependent decrease in heart rate. However, myocardial contractility seems to remain unchanged under therapeutic doses of these drugs [PADDLEFORD and ERHARDT, 1992b]. At moderate doses, cardiac output and arterial blood pressure are only minimally influenced [LAMONT and MATHEWS, 2007]. Intravenous administration of morphine may cause vomiting and release of histamine, followed by vasodilation; for this reason, it is preferable to administer it via the intramuscular route. Butorphanol, the synthetic opioid with both agonist and antagonist activities, has only minimum influence on the cardiovascular system. It causes a clinically irrelevant decrease in heart rate and blood pressure, while stroke volume and peripheral vascular resistance remain unchanged [LAMONT and MATHEWS, 2007]. Partial agonist buprenorphine decreases both heart rate and blood pressure, but increases the peripheral resistance, although – like with butorphanol – the cardiovascular alterations are of no clinical relevance [MARTINEZ et al., 1997].

α2-agonists reduce cardiac output [VICKERY et al., 1988; FLACKE et al., 1993; PYPENDOP and VERSTEGEN, 1998]. Initially, a pronounced vasoconstriction with reflex bradycardia is observed [LEMKE, 2007]. In the further course, vasoconstriction gradually decreases, while bradycardia remains unchanged due to a direct effect on the central nervous system (by reducing the sympathetic tone) [LÖSCHER, 2003b]. When using xylazine, considerable cardiovascular alterations can be observed; these effects are more pronounced after intravenous administration than after intramuscular application. The drop in heart rate is comparable to that occurring after administration of medetomidine, while the increase in blood pressure is less with xylazine than with medetomidine [REDONDO et al., 1999; DIFILIPPO et al., 2004]. Xylazine reduces the cardiac output by 30–50 % and the blood pressure by 20–30 %, respectively [KERR et al., 1972; KLIDE et al., 1975; MUIR et al., 1979; HASKINS et al., 1986]. The cardiovascular alterations produced by dexmedetomidine and medetomidine are similar, although peripheral vasoconstriction lasts longer after the administration of dexmedetomidine [KUUSELA et al., 2003]. The administration of alpha2-agonists may produce first and second degree atrioventricular (AV) blocks even in healthy animals [VAINIO and PALMU, 1989; PADDLEFORD and ERHARDT, 1992a]. Most alpha2-agonists, particularly xylazine, sensitize the myocardium for adrenaline induced arrhythmias [MUIR et al., 1975; TRANQUILLI et al., 1988; LEMKE and TRANQUILLI, 1994]. In contrast, dexmedetomidine is even considered to have a certain cardioprotective effect, as it has been shown in a trial that even a threefold increase of arrhythmia producing doses of adrenaline did not induce any arrhythmias in animals treated with dexmedetomidine [SAVOLA, 1989; HAYASHI et al., 1991].

**Induction of anaesthesia**

Propofol induces a dose dependent decrease in both arterial blood pressure and cardiac contractility. At clinically relevant doses, the decrease in blood pressure is caused by arterial and venous vasodilation and only to a lesser extent by reduced myocardial contractility [ILKIW et al., 1992; GELISSEN et al., 1996]. Venous and arterial vasodilation also decreases both preload and afterload [MUZI et al., 1992; LOWE et al., 1996]. In addition, propofol inhibits the activity of the sympathetic nerve system and reduces the response to the baroreceptor reflex [EBERT et al., 1992; EBERT and MUZI, 1994; SELLGREN et al., 1994]. The barbiturate thiopental sensitizes the myocardium to catecholamines, which may cause arrhythmias, even in patients with no heart disease. Thiopental reduces both cardiac output and blood pressure. Pronounced vasodilation may occur, particularly if administered fast [PLUMB, 2005]. The decrease in blood pressure leads to a reflex increase in heart rate, which in turn increases the oxygen consumption of the myocardium. Etomidate does not induce any changes in heart rate or blood pressure, nor does it have any effect on the myocardium [NAGEL et al., 1979]. It is well suited for anaesthesia in patients with severe myocardial disease and cardiovascular instability [ROBERTSON, 1992]. Etomidate is available as a lipid emulsion preparation and in a formulation with propylene glycol as a solvent. At high doses, the etomidate preparation with propylene glycol may cause haemolysis. For that reason, it is preferable to use the lipid emulsion [KULKA et al., 1993; DOENICKE et al., 1997].
Alphaxalone is one of a group of steroid anaesthetics. When used at clinically relevant doses, alphaxalone has similar effects on the cardiovascular system as propofol [AMBROS et al., 2008]. In dogs, it induces a dose dependent decrease of arterial blood pressure and simultaneously an increase in heart rate so that the cardiac output is maintained [MUIR et al., 2008]. In the cat, it causes a dose-dependent decrease in heart rate, blood pressure and consequently in cardiac output [MUIR et al., 2009].

**Maintenance of anaesthesia**

All inhalation anaesthetics reduce the stroke volume by decreasing myocardial contractility [EGER, 1985; PAGEL et al., 1991; BOBAN et al., 1992; WARLTIER and PAGEL, 1992]. The strongest depression is caused by halothane [KLIDE, 1976; STEFFEY and HOWLAND, 1978, 1980; EGER, 1985]. At usual concentrations, isoflurane, sevoflurane and desflurane may maintain cardiac output at almost normal levels [WARLTIER and PAGEL, 1992; EGER, 1994; MALAN et al., 1995; STEFFEY et al., 2005]. Both vagal and preganglionic sympathetic activity is inhibited by inhalation anaesthetics, but vagal inhibition is more pronounced producing a slight increase in heart rate [PADDLEFORD and ERHARDT, 1992b]. While this increase is very small to non-existing in halothane anaesthesia, it is clearly present with isoflurane, sevoflurane und desflurane, as they possess stronger vasolytic activity [PICKER et al., 2001]. Inhalation anaesthetics produce a dose dependent decrease in blood pressure, which is based on the reduction of the stroke volume and on the vasodilatory effect of these drugs [STEFFEY and HOWLAND, 1977, 1978; MERIN et al., 1991; FRINK et al., 1992]. While the decrease in blood pressure caused by isoflurane, sevoflurane and desflurane is primarily based on the vasodilatory activity of the anaesthetic, halothane decreases blood pressure almost exclusively by reducing myocardial contractility and cardiac output [RIVENES et al., 2001].

Apart from inhalation anaesthetics, injectable anaesthetics (e.g. propofol or alphaxalone constant rate infusion) can be used for maintenance of anaesthesia. In terms of a balanced anaesthesia, fentanyl, ketamine or lidocaine can be administered by constant rate infusion to reduce the amount of the inhalation anaesthetic [MARTIN et al., 2001; PYPENDOP and ILKIW, 2005; VILLALBA et al., 2011]. In most cases, spontaneous breathing during anaesthesia means less stress for the cardiovascular system than forced mechanical ventilation. Forced ventilation may produce an increase in intrathoracic pressure followed by compression of venous vessels and reduction of venous return. Therefore, ventilation at low pressures (approx. 12 cmH2O) is recommended, which should only be initiated when there is a dramatic increase (>60 mmHg) of the end-tidal CO2 (ETCO2). However, patients with hypoxia or acidosis represent an exception: They should receive forced ventilation already at an end-tidal CO2 of 45 mmHg or an oxygen saturation of less than 90 % [ERHARDT, 2004; CLUTTON, 2007].

**Perianesthetic monitoring**

Perianesthetic monitoring is of great importance, not only, but above all in patients with cardiovascular disease. The objective of close monitoring of the anaesthetized patient is to ensure optimum anaesthetic depth with a minimum of physiological alterations [HASKINS, 2007]. Evaluation of the anaesthetic depth can be done by clinical monitoring (e.g. palpebral reflex, position of the bulbus, muscle tone of the jaws), supported by additional instrument based monitoring. In the cardiac patient, instrument based monitoring is required in addition to the assessment of clinical parameters to evaluate cardiovascular function (pulse frequency, colour of mucous membranes, capillary refill time). Performing an ECG provides information on both heart rate and cardiac rhythm. As the induction phase of general anaesthesia is particularly challenging for the cardiovascular system, close monitoring of cardiac patients from the very beginning of this stage by performing an ECG is extremely important. Many anaesthetics cause a decrease in blood pressure. If this adds to existing disease related cardiovascular dysfunction, severe consequences may result. In some cases, a perianesthetic increase in blood pressure may occur, which must immediately be diagnosed and treated. To monitor blood pressure, several invasive and non-invasive methods are available. The most precise results are obtained using the invasive technique (catheterization of a peripheral artery). Apart from exact blood pressure measuring, this technique offers the advantage of allowing sampling for arterial blood gas analysis. However, as with all invasive methods, the risk of potential infections should always be born in mind. It is therefore mandatory to perform catheterization of peripheral arteries under strictly aseptic conditions and
Anaesthesia in dogs and cats with cardiac disease

Pulse oximetry helps identify pulse deficits (difference between the simultaneously counted heart rate [ECG] and the pulse rate) and hypoxaemia. Therefore, pulse oximetry does not only allow an evaluation of the cardiovascular system, but also of the respiratory system.

Another technique to monitor the cardiovascular system is measuring the central venous pressure (e.g. by means of a catheter introduced in the jugular vein). It provides information about the blood volume returning to the heart and the hearts’ capacity to pump it into the arterial system. However, this method is only rarely used in veterinary medicine. There are several other methods to measure cardiac output (e.g. the thermodilution method using a cold thermal indicator; LiDCO technique), but these are rarely used in the routine clinical setting due to their invasiveness and high costs.

Emergency cardiac drugs

Anticholinergic drugs (e.g. atropine, glycopyrrolate) have a parasympatholytic effect, with parasympathetic inhibition of the cardiovascular and gastrointestinal system [LEMKE, 2007]. In perioperative medication, anticholinergics are primarily used for prevention or therapy of bradycardia. In many cases, administration of these drugs produces sinus tachycardia. As this results in increased myocardial work and decreased myocardial perfusion, parasympatholytics should be used with caution in cardiac patients. At clinically relevant doses (0.02–0.04 mg/kg IV/IM), atropine increases both heart rate by 30-40 % for about 30 minutes and atrial contractility [HENDRIX and ROBINSON, 1997]. At

<table>
<thead>
<tr>
<th>Drug</th>
<th>Receptor</th>
<th>Mode of action</th>
<th>Dose</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ephedrine</td>
<td>Alpha-1, Beta-1</td>
<td>Release of norepinephrine Vasoconstriction</td>
<td>0.03–0.1 mg/kg</td>
<td>Severe hypotension</td>
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<td></td>
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<td>Positive chronotropy</td>
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<tr>
<td>Noradrenaline</td>
<td>Alpha-1</td>
<td>Vasoconstriction</td>
<td>0.01–0.05 μg/kg/min</td>
<td>Aortic or pulmonic stenosis Hypertrophic cardiomyopathy</td>
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<tr>
<td>Phenylephrine</td>
<td>Alpha-1</td>
<td>Vasoconstriction</td>
<td>1-3 μg/kg/min</td>
<td>Aortic or pulmonic stenosis Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Beta-1</td>
<td>Positive inotropy and chronotropy, Dose-dependent vasodilation</td>
<td>1-20 μg/kg/min</td>
<td>Valvular insufficiency Dilated cardiomyopathy</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Beta-1, Minor Alpha-1 and Beta-2</td>
<td>Positive inotropy and chronotropy</td>
<td>1-20 μg/kg/min</td>
<td>Valvular insufficiency Dilated cardiomyopathy</td>
</tr>
</tbody>
</table>
lower doses, a drop in heart rate and AV blocks might occur [KANTELIP et al., 1985; RICHARDS et al., 1989]. Glycopyrrolate has similar cardiovascular effects, which last about one hour. While in human patients, tachycardia caused by glycopyrrolate was less pronounced than that induced by atropine, this could not be confirmed by studies in animals [SHORT et al., 1974; RICHARDS et al., 1989]. At therapeutic doses (0.005–0.01 mg/kg IM/IV), an increase in heart rate and atrial contractility occur, while lower doses may also induce a drop in heart rate. Sympathomimetics (see Table 2) have a stimulant effect on the sympathetic nervous system, leading to an increase in heart rate and blood pressure. Dopamine is a positive inotropic drug inducing an increase in heart rate. The effect on the vascular system is dose-dependent. While low doses (0.5–2 μg (micrograms) /kg) lead to vasodilation, intermediate dose levels (2–10 μg/kg) do not produce any vasomotor changes, but organ perfusion improves due to the positive inotropic effect [KELLY and SMITH, 1996; PLUMP, 2008]. High doses of dopamine (10–12 μg /kg) cause peripheral vasoconstriction leading to an increase in blood pressure [KELLY and SMITH, 1996; MARKS and ABBOTT, 1998]. Dobutamine has a positive inotropic and chronotropic effect. Increased myocardial contractility leads to an increase in cardiac output, which indirectly raises the blood pressure. Ephedrine indirectly stimulates the sympathetic nervous system due to the release of noradrenaline. Additional sympathomimetic properties are based upon the effect on α-(alpha)- and β-(beta)-receptors, which leads to positive inotropy and vasoconstriction. Due to the increase in blood pressure, reflex bradycardia occurs [WAGNER et al., 1993]. Adrenaline acts on both α- and β-adrenergic receptors. By stimulating the β1-receptors of the heart, it induces an increase in heart rate and contractility [LÖSCHER, 2003]. The effect on the vascular system is dose dependent. While low doses (< 1 μg/kg) cause a predominantly β-adrenergic stimulation leading to dilation, higher doses stimulate the α-receptors and induce vasoconstriction [LÖSCHER, 2003]. The local anaesthetic lidocaine is one of the Class IB antiarrhythmics (sodium channel blockers). Due to the lidocaine induced conduction slowing, it is often used to treat ventricular arrhythmias (e.g. premature ventricular contractions). As all antiarrhythmics of Class I, lidocaine has a negative inotropic and vasodilatory effect [SCHÜTZ, 1998]. Beta-blockers (e.g. esmolol, atenolol, propranolol) are used for the treatment of tachycardia and hypertension, as they inhibit the effect of the endogenous catecholamines adrenaline and noradrenaline [SCHÜTZ, 1998].

**Recovery phase**

Duration of general anaesthesia and the stress it causes to the cardiovascular system should be as short as possible. The use of antagonisable or short-acting agents is recommended. If long-acting drugs are administered, lower doses should be used [CLUTTON, 2007]. Patients should be allowed to awake in a quiet room. Additional oxygen supply using e.g. an oxygen mask improves myocardial oxygenation during the recovery phase. However, some animals do not tolerate the mask and come under stress. In those cases, the produced stress for the patients’ heart must be balanced against the advantages of additional oxygen supply. Depending on the size of the animal, an oxygen box can be used, if a mask is really unacceptable. Monitoring of cardiovascular parameters (pulse quality, capillary refill time; if necessary, blood pressure and ECG) and regular auscultation of heart and lungs should be continued during the recovery phase, as decompensation might occur and lead to fatal outcome. This has been proved by a study, which found out that 45–60 % of perioperative deaths occurred during the recovery phase after general anaesthesia [BRODBELT et al., 2008].

**Cardiac disease in the dog**

**Mitral valve insufficiency**

Mitral valve insufficiency is the most common cardiac disease in the dog accounting for 75–80 % of all cardiac disorders. Although it is typically found in older dogs, there is a certain breed predisposition in Papillons, Poodles, Chihuahuas, Dachshunds and Cavalier King Charles Spaniels [DAS and TASHJIAN, 1965; DETWEILER and PATTERSON, 1965; BUCHANAN, 1977; THRUSFIELD et al., 1985; THRUSFIELD, 1986; DARKE, 1987; HÄGGSTRÖM et al., 1992; BEARDOW and BUCHANAN, 1993; BUCHANAN, 1999; SISSON and KVART, 1999]. In the course of the disease, the left atrioventricular valve progressively thickens and degenerates in a way that correct closure of the valve becomes impossible. The functional consequence of this degenerative process is mitral valve regurgitation, where blood leaks back from the
ventricle into the left atrium. Depending on the degree of pathological alterations of valvular structures, the condition is classified as mild, moderate or severe mitral valve insufficiency. Clinical consequences depend on the extent of regurgitation and its sequelae. Mild mitral valve regurgitation does not cause any changes in heart function and size. Even with progressive regurgitation, the forward stroke volume is compensated by increasing the preload and occasionally by increasing the heart rate [HÄGGSTRÖM et al., 1996; LORD et al., 2003]. In fact, even patients with severe mitral valve insufficiency can survive several years [BRAUNWALD, 1997; SISSON and KVART, 1999]. However, pathological alterations like increase in cardiac size and hypercontractility develop and, in the later course of the disease, a slowly progressing decrease in myocardial contractility [URABE et al., 1992; SISSON and KVART, 1999; LORD et al. 2003]. Due to increased venous return and the related volume increase in the left atrium, the pulmonary veins get congested and pulmonary oedema develops. In larger dog breeds, the disease usually progresses faster and takes a more dramatic course [HÄGGSTRÖM et al., 2005]. The most common clinical signs, which do not become manifest until the valve insufficiency has reached its severe form, include coughing (compression of the mainstem bronchus due to volume increase in the left atrium), particularly in the second half of the night until early morning, as well as dyspnoea (pulmonary oedema due to increased pressure in the pulmonary veins). Weakness and reduced stamina may also be present as a consequence of the reduced left-ventricular ejection. In rare cases, secondary right heart failure may develop due to mitral valve insufficiency. Permanent pulmonary hypertension causes dilation and dysfunction of the right ventricle, leading to dilation of the tricuspid valve annulus and, as a consequence, to valvular insufficiency [SHIRAN and SAGIE, 2009]. Typical clinical signs of right heart failure are fluid accumulation in the thorax and ascites. The mild and moderate forms of mitral valve insufficiency hardly cause any clinical symptoms [ISACHC 1(-2)], see Table 3, and in most cases, coughing is the only sign [HÄGGSTRÖM et al., 2005].

**Anaesthesia in patients with mitral valve insufficiency**

The exact degree of valve insufficiency should be assessed prior to anaesthesia. First signs of early decomposition are, apart from clinical symptoms like cough, radiological or sonographic evidence of congestion [HARVEY and ETTINGER, 2007; ENGELMANN, 2009]. In case of decompensated valvular disease, the afterload should be reduced administering ACE inhibitors and furosemide [PASCOE, 2005]. In addition, a calcium sensitizer (e.g. pimobendan) may be used to support inotropy [KANNO et al., 2007]. However, pimobendan should only be used in patients with marked clinical symptoms. While it reduces clinical signs in these patients, it may have negative morphological and functional effects in asymptomatic patients [LOMBARD et al., 2006; CHETBOUL et al., 2007]. Heart rate and blood pressure should be assessed already during preanaesthetic examination, in order to obtain reference values for intraoperative monitoring of these parameters [HARVEY and ETTINGER, 2007].

During anaesthesia, an increase of regurgitation must be avoided. Therefore, no centrally effective α2-agonists or massive infusion therapy should be given to avoid any increase in afterload [PASCOE, 2005; HARVEY and ETTINGER, 2007]. In these patients, a significant decrease in heart rate also leads to increased regurgitation as the increased ventricular filling enhances contractility [EVANS, 1992]. Any drugs, which induce an increase in vascular tone and, consequently, in afterload, like dopamine (in vasoconstrictive doses) and ephedrine, should also be avoided [PASCOE, 2005]. Reducing the systemic vascular resistance by administering very small doses of acepromazine as a premedication in order to reduce the afterload is beneficial, as it reduces regurgitation and increases cardiac output despite reduced contractility [PASCOE, 2005; HARVEY and ETTINGER, 2007]. Excessive vasodilation, however, causes a drop in blood pressure, which in most cases can hardly be compensated for by the patient. Opioids like methadone or butorphanol, in combination with

<table>
<thead>
<tr>
<th>Class</th>
<th>Asymptomatic animals</th>
<th>MILD to moderate cardiac insufficiency</th>
<th>Severe cardiac insufficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>I A</td>
<td>No radiographical evidence of cardiomegaly</td>
<td>Reduced exercise tolerance, mildly increased respiratory rate at rest, dyspnoea and coughing on physical exertion</td>
<td>dyspnoea and coughing at rest; frequent oedema</td>
</tr>
<tr>
<td>I B</td>
<td>Radiographical evidence of cardiac enlargement</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Table 3: ISACHC classification (International Small Animal Cardiac Health Council): Classification of the degrees of severity of cardiac disease in small animals (FOX et al., 1999)*
acepromazine, produce adequate sedation and, in addition, analgesia [CLUTTON, 2007]. As opioids, above all μ-agonists (e.g. methadone), can reduce the heart rate if administered at higher doses, an anticholinergic drug (like atropine or glycopyrrolate) should always be at hand when μ-agonists are used, in order to be prepared if a drop in heart rate should occur [HARVEY and ETTINGER, 2007]. Whenever possible, induction of anaesthesia should be performed under complete monitoring and good preoxygenation. In severe cases, etomidate is a good choice as it has minimum cardiovascular side effects. In stable patients, low doses of ketamine can be used as an alternative, together with benzodiazepines or low doses of propofol [HARVEY and ETTINGER, 2007; FAYYAZ et al., 2009]. Negative inotropic drugs like propofol at high doses and thiopental can increase the regurgitation fraction in patients with severe valvular disease due to reduced forward propulsion of the blood and should therefore be used with caution [PASCOE, 2005].

To maintain anaesthesia, inhalation anaesthetics can be used at concentrations that should be as low as possible. Another possibility is a partial or total intravenous anaesthesia using propofol, fentanyl or ketamine combinations (subanaesthetic doses) [PASCOE, 2005]. Table 4 summarises dose suggestions for the mentioned drugs for premedication, induction and maintenance of general anaesthesia. In case hypotension and bradycardia should occur, these can be treated by administration of anticholinergics. In doing so, the target heart rate should lie within the preanaesthetic range or slightly above [DAY, 2002]. Should hypotension not be accompanied by bradycardia and not return to normal levels after reducing the concentration of the inhalant, positive inotropic drugs like dobutamine should preferably be administered [PASCOE, 2005]. The most important properties and dosages of antihypotensive drugs are listed in Table 2.

Table 4: Anaesthesia protocols for dogs with cardiac disease

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indication</th>
<th>Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Premedication</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acepromazine</td>
<td>Valvular insufficiency, DCM</td>
<td>5–20 μg/kg</td>
<td></td>
</tr>
<tr>
<td>Butorphanol</td>
<td>All cardiac conditions</td>
<td>0.1-0.4 mg/kg</td>
<td>Visceral analgesia</td>
</tr>
<tr>
<td>Methadone</td>
<td>All cardiac conditions</td>
<td>0.1-0.4 mg/kg</td>
<td>Good somatic analgesia</td>
</tr>
<tr>
<td>Midazolam</td>
<td>All cardiac conditions</td>
<td>0.1-0.5 mg/kg</td>
<td>Warning: paradoxical reactions in generally healthy patients</td>
</tr>
<tr>
<td><strong>Induction</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>Mild valvular insufficiency, mild DCM</td>
<td>2-6 mg/kg</td>
<td>Respiratory depression, hypotension Long maintenance of pharyngeal reflex</td>
</tr>
<tr>
<td>Etomidate</td>
<td>All cardiac conditions</td>
<td>1-2 mg/kg</td>
<td>Not to be used with adrenal disease Good somatic analgesia</td>
</tr>
<tr>
<td>Alphaxalone</td>
<td>Mild valvular insufficiency, DCM</td>
<td>1-2 mg/kg</td>
<td>Possibly hypotension and respiratory depression</td>
</tr>
<tr>
<td>Ketamine</td>
<td>Valvular insufficiency</td>
<td>1-5 mg/kg</td>
<td>Combination with benzodiazepines or low-dosed propofol possible</td>
</tr>
<tr>
<td><strong>Maintenance</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isoflurane/ Sevoflurane</td>
<td>All cardiac conditions</td>
<td>Concentration depending on chosen anaesthetic/ analgesic; vasodilation, hypotension; keep doses low Not to be used with adrenal disease Good somatic analgesia</td>
<td></td>
</tr>
<tr>
<td>Propofol (CRI)</td>
<td>All cardiac conditions</td>
<td>6–12 mg/kg/h</td>
<td>Possibly respiratory depression</td>
</tr>
<tr>
<td>Fentanyl (CRI)</td>
<td>All cardiac conditions</td>
<td>10-20 μg/kg/h</td>
<td>Possibly respiratory depression and bradycardia, somatic analgesia</td>
</tr>
<tr>
<td>Ketamine (CRI)</td>
<td>All cardiac conditions</td>
<td>0.3-0.6 mg/kg/h</td>
<td>Analgetic dose</td>
</tr>
</tbody>
</table>

*Combinations with premedication in same doses possible (except methadone with butorphanol). **Combinations can be freely chosen
Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is the most frequently diagnosed myocardial condition in dogs (SISSON et al., 1999). Dilated cardiomyopathy is characterized by cardiomegaly and reduced systolic function of one or both ventricles [RICHARDSON et al., 1996]. Particularly medium-sized to large canine breeds are affected by DCM. With certain regional differences, the condition is most frequently diagnosed in Doberman Pinschers, Irish Wolfhounds, Great Danes, Cocker Spaniels, Airedale Terriers, Newfoundlands, Boxers, English Cocker Spaniels, Portuguese Water Dogs and Dalmatians [HARPSTER, 1983; MONNET et al., 1995; FREEMAN et al., 1996; TIDHOLM and JÖNSSON, 1997; SISSON et al., 2000]. With the exception of the Portuguese Water Dog, cardiac symptoms do not become manifest until the dogs have reached adulthood [SLEEPER et al., 2002]. DCM takes a progressive course with gradually increasing exercise intolerance and reduction of body mass [DAMBACH et al., 1999]. Due to myocardial insufficiency and the resulting systolic dysfunction, emptying of the chamber is incomplete. Filling of the ventricle during diastole produces an increased end-diastolic pressure, which leads to dilatation of the ventricle. In the further course of the disease, ventricular dilation causes geometric distortion of the atrioventricular valve apparatus, leading to insufficiency and atrial dilation [MEURS, 2005]. Clinical signs of DCM-related congestive heart failure are primarily due to the left heart failure and include coughing, dyspnoea and tachypnoea [CALVERT et al., 1982; CALVERT, 1986]. Only occasionally, ascites is observed as a symptom of right heart failure.

Anaesthesia in patients with dilated cardiomyopathy

In all dogs with DCM, just as in dogs with valvular insufficiency, the degree of severity of the condition should be assessed prior to anaesthesia. As sinus tachycardia, atrial fibrillation or ventricular tachyarrhythmias are to be expected in the late form of dilated cardiomyopathy, it is recommended to perform not only a radiographic and sonographic examination, but also an electrocardiographic examination and blood pressure measurement in order to identify and evaluate the haemodynamic effects of the cardiac disease [MEURS, 2005]. In patients with retrograde congestion in the pulmonary and systemic circulation, vasodilatory support with loop diuretics and ACE inhibitors is also indicated. As inotropy is particularly compromised in patients with DCM, inotropic therapy using pimobendan represents a cornerstone of preanaesthetic stabilization [FUENTES et al., 2002]. If supraventricular tachycardia occurs, therapy can be complemented by administration of digoxin or beta-blockers [ABBAH et al., 1994; GILBERT et al., 1999; ATKINS, 2007]. For premature ventricular contractions, Class IB antiarrhythmics (e.g., mexiletine) are recommended [OPIE et al., 2009]. For the anaesthesia protocol, the same principles apply as for valvular insufficiency. However, with DCM, it is even more important to avoid negative inotropy. Mild vasodilation reduces the afterload and increases contraction effectivity [PASCOE, 2005]. Eligible drugs for induction of anaesthesia are etomidate and alphaxalone [HARVEY and ETTINGER, 2007]. Perioperative monitoring using ECG and blood pressure measurement is indispensable for patients suffering from dilated cardiomyopathy. In patients with advanced disease, invasive blood pressure measurement is recommended [SKARDA et al., 1995b]. Should this not be possible, non-invasive methods like oscillometry or Doppler technique should be used. For emergencies in DCM patients, lidocaine and esmolol for treatment of ventricular and supraventricular tachyarrhythmias should be kept at hand apart from glycopyrrolate for treating bradycardia.

Aortic stenosis

Depending on the site of stenosis, there are three types of aortic stenosis: subvalvular, valvular and supravalvular. In the vast majority of dogs, the aortic stenosis is located in the subvalvular region [ETTINGER and SUTER, 1970]. Breeds like Newfoundland, Golden Retriever, German Shepherd, Boxer, Bouvier, Rottweiler and Bull Terrier are predisposed to aortic stenosis [PATTERSON, 1968; ETTINGER and SUTER, 1970; O’GRADY et al., 1989; TIDHOLM and JÖNSSON, 1997]. As a consequence of the increased myocardial strain caused by the outflow obstruction during systole, symmetrical left ventricular hypertrophy develops [ETTINGER and SUTER, 1970; PYLE et al., 1976; BRAUNWALD, 1988; FRIEDMAN, 1988]. Due to the concentric hypertrophy, myocardial ischaemia occurs leading to ventricular arrhythmias [SCHWARTZ et al., 1969; PYLE et al., 1976; BORKON et al., 1982; BRAUNWALD, 1988; FRIEDMAN, 1988]. In the healthy heart, physical strain reduces the arterial tone and increases the stroke volume. However, with aortic stenosis, this physiological reaction is possible to only a limited extent, as the increase in stroke volume is limited by the obstruction of the left ventricular...
outflow tract [HOSSACK, 1987; FRIEDMAN, 1988]. Cardiac output, which depends on both stroke volume and heart rate, becomes increasingly dependent on the heart rate only, as volume capacity and ventricular elasticity are reduced. As the diseased heart is not able to increase the stroke volume, weakness (reduced oxygen supply to the muscles), syncope (reduced oxygen supply to the brain) and/or ventricular arrhythmias (myocardial hypoxia) develop [SCHWARTZ et al., 1969; PYLE, 1983; BRAUNWALD, 1988; FRIEDMAN, 1988]. In rare cases only there are signs of a gradual development of congestive heart failure. Pulmonary oedema only develops if simultaneous mitral valve insufficiency or an extremely severe form of aortic stenosis is present [BRAUNWALD, 1988; O’GRADY et al., 1989].

Anaesthesia in patients with aortic stenosis

Taking into account that patients with aortic stenosis are not able to compensate for cardiovascular alterations, it is important to keep any influences on the cardiovascular parameters to a minimum. Bradycardia reduces cardiac output, because it is impossible to increase the stroke volume. Tachycardia increases the myocardial oxygen consumption reducing at the same time coronary perfusion. Vasodilatory drugs (e.g. acepromazine) should be avoided as the organ tries to counteract hypotension by tachycardia [PASCOE, 2005]. Positive inotropic drugs should not be used either, because the increased contractility would only worsen the outflow obstruction [BUBENHEIMER, 2007].

Premedication with a benzodiazepine and an opioid seems to be a promising option for patients affected by aortic stenosis, as they only have little effect on inotropy and vascular tone. Induction and maintenance of general anaesthesia should, whenever possible, only have a minimum negative inotropic effect. For this reason, it is recommended to use etomidate to induce anaesthesia. For maintenance of anaesthesia, an opioid (e.g. fentanyl) administered as a constant rate infusion may be used in combination with an inhalant (at the lowest possible concentration) or with a constant rate infusion of propofol. In hypotensive patients, 1-agonists (with vasoconstrictive effect) like phenylephrine should be preferred to positive inotropic or chronotropic drugs (dopamine, dobutamine) [CLUTTON, 2007].

Pulmonic stenosis

Also this form of vascular stenosis can be located either in the subvalvular, valvular or supravalvular region. The most common form in the dog is valvular stenosis [TRAUTVETTER et al., 2007]. The condition is frequently diagnosed in Beagles, Samoyeds, Chihuahuas, English Bulldogs, Miniature Schnauzers, Labrador Retrievers, Mastiffs, Chow Chows, Newfoundlands, Boxers, Basset Hounds, Fox Terriers, West Highland White Terrier and other Terrier and Cocker breeds [STAFFORD JOHNSON and MARTIN, 2004; WARE, 2006; TRAUTVETTER et al., 2007]. As a consequence of the pulmonic stenosis, concentric right ventricular hypertrophy develops. Hypertrophy of the myocardium leads to hypoperfusion with hypoxia and ventricular arrhythmias. Due to the outflow obstruction, the filling pressure in the right ventricle increases, leading to tricuspid valve regurgitation and, as a consequence, to right heart failure [KITTLESON and KIENLE, 1998; STAFFORD JOHNSON and MARTIN, 2004]. Most dogs suffering from pulmonic stenosis do not show any clinical signs of disease (WARE, 2006).

In about 35 % of dogs with severe pulmonic stenosis, exercise intolerance, syncopes or ascites may be observed [GORDON et al., 2002].

Anaesthesia in patients with pulmonic stenosis

Peri-anesthetic management of patients with pulmonic stenosis is the same as with aortic stenosis. Also in these patients, tachycardia may increase myocardial oxygen consumption without increasing the pulmonary arterial pressure. Appropriate premedication should therefore provide good sedation without inducing an increase in heart rate. In most cases, this is only possible using opioids [PASCOE, 2005]. For induction of general anaesthesia, etomidate is particularly suitable, perhaps together with simultaneous administration of a benzodiazepine. For maintenance of general anaesthesia, the dose of the inhalation anaesthetic should be reduced as much as possible by providing a partial intravenous anaesthesia using a constant rate infusion of an opioid [PASCOE, 2005].

Cardiac disease in the cat

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is the most frequently diagnosed cardiac disease in cats. A breed predisposition for the primary form has been reported for Maine Coons, Persians, British Shorthairs, Norwegian Forest Cats, Ragdolls, Turkish Vans and Scottish Folds [KITTLESON, 2005]. With the secondary form of the
condition, concentric hypertrophy develops due to increased myocardial strain caused by aortic stenosis, systemic hypertension or hyperthyroidism [KITTLESON, 2005]. The disease causes mild to severe thickening of the ventricular myocardium, above all in the region of the free wall of the left ventricle, reducing diastolic filling capacity. Hypertrophy of the chamber musculature leads to insufficient blood supply of the myocardium and, as a consequence, to ischaemia, hypoxia, myocardial necrosis and fibrosis. The circulatory system tries to maintain cardiac output, leading to a compensatory increase in heart rate. In the more advanced stages of hypertrophic cardiomyopathy, the left atrium becomes increasingly dilated. Due to the resultant blood stasis in the atrium, thrombi may form and be flushed into the blood stream. Increased intraventricular pressure and reduced filling volume of the ventricle lead to chronic heart failure with pulmonary oedema and/or fluid accumulation in the thorax. Many cats with hypertrophic cardiomyopathy also suffer from compensatory tachycardia. Due to that increase in heart rate, diastolic dysfunction is further aggravated due to diastolic shortening.

Anaesthesia in patients with hypertrophic cardiomyopathy

When performing a preanaesthetic examination of cats with HCM, a Hyperthyroidism or chronic kidney disease should be ruled out and, if present, be treated prior to surgery. Therefore, blood pressure measurement is mandatory, because in the primary form of the condition hypotension is predominantly present, while the secondary form of hypertrophic cardiomyopathy is characterized by hypertension. In addition, sonographic and radiographic examinations should be performed to identify possible pulmonary congestion. As diastolic dysfunction typically occurs with the condition, tachycardia should be identified as soon as possible and, if present, be treated with beta-blockers, as any increase in heart rate inevitably leads to elevated myocardial oxygen consumption and reduced perfusion of the coronary arteries during diastole [BEDNARSKI, 1992].

Drugs, which reduce the preload by inducing vasodilation (e.g. acepromazine), should be avoided. In contrast, centrally effective α2-agonists administered at low doses may increase the preload by inducing vasoconstriction, thus enhancing diastolic filling of the heart [PASCOE,

<table>
<thead>
<tr>
<th>Table 5: Anaesthesia protocols for cats with hypertrophic cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drugs</strong></td>
</tr>
<tr>
<td>----------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Premedication</strong>*</td>
</tr>
<tr>
<td>Butorphanol</td>
</tr>
<tr>
<td>Methadone</td>
</tr>
<tr>
<td>Midazolam</td>
</tr>
<tr>
<td>Medetomidine</td>
</tr>
<tr>
<td>Dexametomidine</td>
</tr>
<tr>
<td><strong>Induction</strong></td>
</tr>
<tr>
<td>Propofol</td>
</tr>
<tr>
<td>Etomidate</td>
</tr>
<tr>
<td>Alphaxalone</td>
</tr>
<tr>
<td><strong>Maintenance</strong>*</td>
</tr>
<tr>
<td>Isoflurane/ Sevoflurane</td>
</tr>
<tr>
<td>Fentanyl (CRI)</td>
</tr>
<tr>
<td>Propofol (CRI)</td>
</tr>
</tbody>
</table>

*Combinations with premedication in same doses possible (except methadone with butorphanol). **Combinations can be freely chosen
Positive inotropic drugs should be avoided due to the risk of outflow obstruction caused by increased contractility of the hypertrophic myocardium. This might also occur when reducing the afterload. For that reason, all should be done to maintain the afterload at physiological levels or to slightly increase it. Table 5 contains a list of drugs suitable for premedication, together with recommended dosages.

The ideal drug for induction of general anaesthesia in cats with hypertrophic cardiomyopathy is etomidate. Propofol at low doses may be used as an alternative. Thiopental and ketamine should, if possible, not be used due to their arrhythmogenic and positive inotropic effect; in addition, they would induce an increase in heart rate. Inhalation anaesthetics are well suited for maintenance of anaesthesia in patients with HCM as they provide myocardial depression [POLIAC et al., 2006]. In case hypotension should occur, an α1-agonist like phenylephrine or noradrenaline can be given to produce vasoconstriction [PASCOE, 2005].

**Conclusion**

Before performing surgery under general anaesthesia in cardiac patients, the type of disease must be identified and the degree of compensation assessed. In decompensated cardiac disease, the anaesthetic risk can be reduced by providing specific medical therapy prior to anaesthesia.

The entire perianaesthetic period should be as stress free as possible for the patient. Establishing a venous line, intubating the patient and supplying oxygen are indispensable measures to be taken before performing anaesthesia in cardiac patients. For most animals with cardiac disease, premedication with an opioid and, where indicated, with a benzodiazepine, and induction of anaesthesia with etomidate are a good option. Monitoring of anaesthesia should include clinical monitoring, ECG, blood pressure measurement, capnography and pulse oxymetry. Emergency drugs should be at hand at all times in case any complications should present.

**References**


Anaesthesia in dogs and cats with cardiac disease ...  


Anaesthesia in dogs and cats with cardiac disease …


