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# Anaesthesia in dogs and cats with cardiac disease – An impossible endeavour or a challenge with manageable risk?

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## SUMMARY

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

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## 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).

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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

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

| Drug | Heart rate | Inotropy | Cardiac output | Vascular resistance | Arterial blood pressure |
|---|---|---|---|---|---|
| Acepromazine | ↔ | ↔ | ↔ | ↓ | ↓ |
| Midazolam | ↔ | ↔ | ↔ | ↔ | ↔ |
| Diazepam | ↔ | ↔ | ↔ | ↔ | ↔ |
| Butorphanol | ↔ | ↔ | ↔ | ↔ | ↔ |
| Buprenorphine | ↔ | ↔ | ↔ | ↔ | ↔ |
| Methadone | ↓ | ↔ | ↔ | ↔ | ↔ |
| Fentanyl | ↓ | ↔ | ↔ | ↔ | ↔ |
| Xylazine<br>Medetomidine<br>Dexmedetomidine | ↓↓ | ↔ | ↓ | ↑ | ↑↓ |
| Ketamine | ↑ | ↑ | ↑ | ↑ | ↑ |
| Propofol | ↔ | ↓ | ↓ | ↓ | ↓ |
| Thiopental | ↑ | ↓ | ↓ | ↓ | ↓ |
| Alphaxalone | ↑ | ↓ | ↓ | ↓ | ↓ |
| Etomidate | ↔ | ↔ | ↔ | ↔ | ↔ |
| Isoflurane | ↑ | ↔ | ↔ | ↓ | ↓ |
| Sevoflurane | ↔ | ↔ | ↔ | ↓ | ↓ |

↓: 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 [LEMKE 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 [LEMKE u. 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 ERHARD, 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]. The 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 $CO^2$ ($ETCO^2$). However, patients with hypoxia or acidosis represent an exception: They should receive forced ventilation already at an end-tidal $CO^2$ of 45 mmHg or an oxygen saturation of less than 90 % [ERHARDT, 2004; CLUTTON, 2007].

## Perianaesthetic monitoring

Perianaesthetic 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 perianaesthetic 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

only in critically ill patients. For non-invasive blood pressure measuring, the oscillometric method and the Doppler ultrasound can be used. The so-called "High-Definition Oscillometry" HDO) provides results, which show a very high degree of concordance with results obtained by invasive methods. In contrast to the oscillometric method and the Doppler technique, HDO provides exact measurements even at low blood pressures (systolic blood pressure <60 mmHg). The Doppler technique is primarily used to evaluate blood flow, but can also measure blood pressure; it should be kept in mind, however, that in the anaesthetized patient it does not measure the systolic but the mean blood pressure [CAULKETT et al., 1998]. Capnography is a technique that measures the carbon dioxide concentrations in the exhaled air (ETCO2) and the respiratory frequency. Combined monitoring of respiratory frequency and heart rate together with the control of reflexes allows a good evaluation of the anaesthetic depth and helps identify apnoeic episodes. Measuring ETCO2 provides information on the ventilation (normocapnia or hypocapnia with hyperventilation and hypercapnia with hypoventilation, respectively). A decrease in ETCO2 is not only observed during hyperventilation, it also indicates that the transport of CO2 from the periphery to the alveoli is impaired due to a drop in blood pressure or a cardiac arrest. For that reason, it is of utmost importance to relate all results from instrument based monitoring to other diagnostic data obtained and to interpret them in connection with the clinical picture of the patient.
Pulse oximetry provides information on pulse frequency and oxygenation of the arterial blood. This technique 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 2: Receptor binding, mode of action and dosage guide for antihypotensive drugs*

| Drug | Receptor | Mode of action | Dose | Indication |
|---|---|---|---|---|
| Ephedrine | Alpha-1<br>Beta-1 | Release of norepinephrine<br>Vasoconstriction<br>Positive chronotropy | 0.03–0.1 mg/kg | Severe hypotension |
| Noradrenaline | Alpha-1 | Vasoconstriction | 0.01–0.05 µg/kg/min | Aortic or pulmonic stenosis<br>Hypertrophic cardiomyo-pathy |
| Phenylephrine | Alpha-1 | Vasoconstriction | 1-3 µg/kg/min | Aortic or pulmonic stenosis<br>Hypertrophic cardiomyo-pathy |
| Dopamine | Beta-1 | ositive inotropy and chronotropy,<br>Dose-dependent vasodilation | 1-20 µg/kg/min | Valvular insufficiency<br>PDilated cardiomyopathy |
| Dobutamine | Beta-1<br>Minor Alpha-1<br>and Beta-2 | Positive inotropy and chronotropy | 1-20 µg/kg/min | Valvular insufficiency<br>Dilated cardiomyopathy |

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 $\alpha$ (alpha)- and $\beta$ (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 $\alpha$- and $\beta$-adrenergic receptors. By stimulating the $\beta$1-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 $\beta$-adrenergic stimulation leading to dilation, higher doses stimulate the $\alpha$-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 vasodilatatory 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].

*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)*

| | |
|---|---|
| **Class I Asymptomatic animals** | |
| I A | No radiographical evidence of cardiomegaly |
| I B | Radiographical evidence of cardiac enlargement |
| **Class II Mild to moderate cardiac insufficiency** | |
| | Reduced exercise tolerance, mildly increased respiratory rate at rest, dyspneoa and coughing on physical exertion |
| **Class III Severe cardiac insufficiency** | |
| | dyspnoea and coughing at rest; frequent oedema |

## Anaesthesia in patients with mitral valve insufficiency

The exact degree of valve insufficiency should be assessed prior to anaesthesia. First signs of early decompensation 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 $\alpha$2-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

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 in case 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*

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

*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, $\alpha_1$-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

Perianaesthetic management of patients with pulmonic stenosis is the same is 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 $\alpha$2-agonists administered at low doses may increase the preload by inducing vasoconstriction, thus enhancing diastolic filling of the heart [PASCOE,

*Table 5: Anaesthesia protocols for cats with hypertrophic cardiomyopathy*

| Drugs | Dosage | Comments |
|---|---|---|
| **Premedication*** | | |
| Butorphanol | 0.1-0.4 mg/kg | Visceral analgesia |
| Methadone | 0.1-0.4 mg/kg | Good somatic analgesia |
| Midazolam | 0.1-0.5 mg/kg | Warning: paradoxical reactions in generally healthy patients |
| Medetomidine | 1-20 µg/kg | Increases vascular tone, decreases heart rate |
| Dexmedetomidine | 0.5-10 µg/kg | Increases vascular tone, decreases heart rate |
| **Induction** | | |
| Propofol | 2-6 mg/kg | Respiratory depression, hypotension |
| Etomidate | 1-2 mg/kg | Pharyngeal reflex is maintained, first-choice drug |
| Alphaxalone | 1-4 mg/kg | Possibly hypotension, dose-dependent respiratory depression |
| **Maintenance**** | | |
| Isoflurane/ Sevoflurane | | Concentration depending on chosen anaesthetic/analgesic; vasodilation, hypotension |
| Fentanyl (CRI) | 10-20 mg/kg/h | Possibly respiratory depression and bradycardia, good somatic analgesia |
| Propofol (CRI) | 6-12 mg/kg/h | Respiratory depression |

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

2005]. 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 $\alpha$1-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

AMBROS, B., DUKE-NOVAKOVSKI, T., PASLOSKE, K.S. (2008): Comparison of the anaesthetic efficacy and cardiopulmonary effects of continuous rate infusions of alfaxalone-2-hydroxypropyl-β-cyclodextrin and propofol in dogs. Am J Vet Res 69, 1391–1398.

ATKINS, C.E. (2007): An approach to asymptomatic acquired heart disease in
dogs. Proc. of the World Small Animals Veterinary Association, Sydney, Australia.

BEARDOW, A.W., BUCHANAN, J.W. (1993): Chronic mitral valve disease in Cavalier King Charles Spaniels: 95 cases (1987-1991). J Am Vet Med Assoc 203, 1023–1029.

BEDNARSKI, R.M. (1992): Anesthetic concerns for patients with cardiomyopathy. Vet Clin North Am: Small Anim Pract 22, 460–465.

BOBAN, M., STOWE, D.F., BULJUBASIC, N. (1992): Direct comparative effects of isoflurane and desflurane in isolated guinea pig hearts. Anesthesiol 76, 775–780.

BORGARELLI, M. (2005): Physiology of the heart and main cardiological diseases in small animals. Usefulness of ECG radiology and echocardiography exams. Proc. AVA Spring Meeting, Rimini, Italy, p. 12–15.

BORKON, A.M., JONES, M., BELL, J.H., PIERCE, J.E. (1982): Regional myocardial blood flow in left ventricular hypertrophy. An experimental investigation on Newfoundland dogs with congenital subaortic stenosis. J Thorac Cardiovasc Surg 84, 876–885.

BRAUNWALD, E. (1988): Valvular heart disease. In: BRAUNWALD, E. (ed.): Heart Disease: a Textbook of Cardiovascular Medicine. 3rd ed., Saunders, Philadelphia, p. 1052–1060.
BRAUNWALD, E. (1997): Valvular heart disease. In: BRAUNWALD, E. (Ed.): Heart Disease: a Textbook of Cardiovascular Medicine. 5th ed., Saunders, Philadelphia, p. 1007–1076.

BRODBELT, D.C., BLISSITT, K.J., HAMMOND, R.A., NEATH, P.J., YOUNG, L.E., PFEIFFER, D.U., WOOD, J.L. (2008): The risk of death: the confidential enquiry into perioperative small animal fatalities. Vet Anaesth Analg 35, 365–373.

BUBENHEIMER, P. (2007): Aortenstenose. In: ROSKAMM, H., NEUMANN, F.J., KALUSCHA, D., BESTEHORN, H.P. (Hrsg.): Herzkrankheiten. 5. Aufl., Springer, Heidelberg, S. 683–706.

BUCHANAN, J.W. (1977): Chronic valvular disease (endocardiosis) in dogs. Adv Vet Sci Comp Med 21, 75–106.

BUCHANAN, J. (1999): Prevalence of cardiovascular disorders. In: FOX, P.R., SISSON, D, MOISE, N. (eds.): Textbook of canine and feline cardiology. 2nd ed., Saunders, Philadelphia, p. 457–470.

CALVERT, C.A. (1986): Dilated congestive cardiomyopathy in Doberman Pinschers. Comp Cont Educ Pract Vet 8, 417–430.

CALVERT, C.A., CHAPMAN, W.L., TOAL, R.L. (1982): Congestive cardiomyopathy in Doberman Pinscher dogs. J Am Vet Med Assoc 181, 598–602.

CARR, A.P., DUKE, T., EGNER, B. (2008): Blood pressure in small animals - part I: hypertension and hypotension and an update on technology. Europ J Comp Anim Pract 18, 135–142.

CAULKETT, N.A., CANTWELL, S.L., HOUSTON, D.N. (1998): A comparison of indirect blood pressure monitoring techniques in the anesthetized cat. J Vet Surg 27, 370–377.

CHETBOUL, V., LEFEBVRE, H.P., SAMPEDRANO, C.C., GOUNI, V., SAPONARO, V., SERRES, F., CONCORDAT, D., NICOLLE, A.P., POUCHELON, J.-L. (2007): Comparative adverse cardiac effects of pimobendan and benazepril monotherapy in dogs with mild degenerative mitral valve disease: a prospective, controlled, blinded, and randomized study. J Vet Internal Med 21, 742–753.

CLUTTON, E. (2007): Cardiaovascular disease. In: SEYMOR, C., DUKE-NOVAKOVSKI, T. (eds.): BSAVA manual of canine and feline anaesthesia and analgesia. 2nd ed., BSAVA, Gloucester, p. 200–219.

DAMBACH, D.M., LANNON, A., SLEEPER, M.M., BUCHANAN, J. (1999): Familial dilated cardiomyopathy of young Portuguese Water Dogs. J Vet Internal Med 13, 65–71.

DARKE, P.G.G. (1987): Valvular incompetence in Cavalier King Charles Spaniels. Vet Rec 120, 365–366.

DAS, K.M., TASHJIAN, R.J. (1965): Chronic mitral valve disease in the dog. Vet Med, Small Animal Clin 60, 1209–1216.

DAY, T. (2002): Anesthesia of patients with cardiac disease. In: GREENE, S.A. (eds.): Veterinary anesthesia and pain management secrets. 2nd ed., Hanley and Belfus, Philadelphia, p. 157–164.

DETWEILER, D.K., PATTERSON, D.F. (1965): The prevalence and types of cardiovascular disease in dogs. Ann New York Acad Sci 127, 481–516.

DiFILIPPO, S.M., NORBERG, P., SUSON, U.D., SAVINO, A.M., REIM, D.A. (2004): A comparison of xylazine and medetomidine in an anesthetic combination in New Zealand White rabbits. Contemp Topics 43, 32–43.

DOENICKE, A., ROIZEN, M.F., HOERNECKE, R., MAYER, M., OSTWALD, P., FOSS, J. (1997): Haemolysis after etomidate: comparison of propylene glycol and lipid formulations. Br J Anaesth 79, 386–288.

EBERSPÄCHER, E., BAUMGARTNER, C., HENKE, J., ERHARDT, W. (2005): Invasive Blutdruckmessung nach intramuskulärer Verabreichung von Acepromazin als Narkoseprämedikation beim Hund. Tierärztl Praxis 33, 27–31.

EBERT, T.J., MUZI, M. (1994): Propofol and autonomic reflex functions in humans. Anest Anal 78, 369–375.

EBERT, T.J., MUZI, M., BERENS, R., GOFF, D., KAMPINE, J.P. (1992): Sympathetic response to induction of anesthesia in humans with propofol or etomidate. Anesthesiol 76, 725–733.

EGER, E.I. (1985): Isoflurane (Forane): a compendium and reference. 2nd ed., Ohio Medical Products, Madison, p. 79–90.

EGER, E.I. (1994): New inhaled anesthetics. Anesthesiol 80, 906–922.

ENGELMANN, L. (2009): Bedeutung und Einsatz der Beatmung bei akut dekompensierter Herzinsuffizienz. Intensivmed Notfallmed 46, 391–398.

ERHARDT, W. (2004): Tiere mit bestehenden kardiovaskulären Problemen. In: ERHARDT, W., HENKE, J., HABERSTROH, J. (Hrsg.): Anästhesie und Analgesie beim Klein- und Heimtier. Schattauer, Stuttgart, S. 432–453.

ERHARDT, W., HENKE, J., KROKER, P. (2004): Pharmaka im Rahmen der Anästhesie und der perioperativen Schmerzlinderung. In: ERHARDT, W., HENKE, J., HABERSTROH, J. (Hrsg.): Anästhesie und Analgesie beim Klein- und Heimtier. Schattauer, Stuttgart, S. 15–138.

ETTINGER, S.J., SUTER, P.F. (1970): Acquired and congenital heart disease. In: ETTINGER, S.J., SUTER, P.F. (Eds.): Canine Cardiology. Saunders, Philadelphia, p. 528–541.

EVANS, A.T. (1992): Anesthesia for severe mitral and tricuspid regurgitation. Vet Clin North Am: Small Anim Pract 22, 465-466.

FARVER, T.B., HASKINS, S.C., PATZ, J.D. (1986): Cardiopulmonary effects of acepromazine and of subsequent administration of ketamine in the dog. Am J Vet Res 47, 631–635.

FAYYAZ, S., KERR, C.L., DYSON, D.H., MIRAKHUR, K.K. (2009): The cardiopulmonary effects of anesthetic induction with isoflurane, ketamine-diazepam or propofol-diazepam in the hypovolemic dog. Vet Anaesth Analg 36, 110–123.

FLACKE, W., FLACKE, J., BLOOR B., McINTEE, D.F., SAGAN, M. (1993): Effects of dexmedetomidine on systemic and coronary haemodynamics in the anesthetized dog. J Cardiothorac Vasc Anesth 7, 41–49.

FOX, P.R., SISSON, D., MOISE, N.S. (1999): International Small Animal Cardiac Health Council 1999: recommendations for diagnosis of heart disease and treatment of heart failure in small animals. In: FOX, P.R., SISSON, D., MOISE, N.S. (Eds.): Textbook of canine and feline cardiology. 2nd ed., Saunders, Philadelphia, Appendix A.

FREEMAN, L.M., MICHEL, K.E., BROWN, D.J., KAPLAN, P.M., STAMOULIS, M.E., ROSENTHAL, S.L., KEENE, B.W., RUSH, J.E. (1996): Idiopathic dilated cardiomyopathy in Dalmatians: nine cases (1990-1995). J Am Vet Med Assoc 209, 1592–1596.

FRIEDMAN, W. (1988): Congenital heart disease in infancy and childhood. In: BRAUNWALD, E. (ed.): Heart Disease: A Textbook of Cardiovascular Medicine. 3rd ed., Saunders, Philadelphia, p. 932–938.

FRINK, E.J., MORGAN, S.E., COETZEE, A., CONZEN, P.F., BROWN, B.R. (1992): The effects of sevoflurane, halothane, enflurane, and isoflurane on hepatic blood flow and oxygenation in chronically instrumented greyhound dogs. Anesthesiol 76, 85–90.

FUENTES, V.L., CORCORAN, B., FRENCH, A., SCHOBER, K.E., KLEEMANN, R., JUSTUS, C. (2002): A double blind, randomized, placebo-controlled study of pimobendan in dogs with dilated cardiomyopathy. J Vet Internal Med 16, 255–261.

GELISSEN, H.P., EPEMA, A., HENNING, R.H., KRIJNEN, H.J., HENNIS, P.J., DEN HERTOG, A. (1996): Inotropic effects of propofol, thiopental, midazolam, etomidate, and ketamine on isolated human atrial muscle. Anesthesiol 84, 397–403.

GILBERT, E.M., ANDERSON, J.L., DEITCHMAN, D., YANOWITH, F.G., CONNELL, J.B., RENLUND, D.G., BARTHOLOMEW, M., MEALEY, P.C., LARRABEE, P., BRISTOW, M.R. (1999): Long-term α-blocker vasodilator therapy improves cardiac function in idiopathic dilated cardiomyopathy: a double blind randomized study of bucindolol versus placebo. Am J Med 88, 223–229.

GORDON, S.G., MILLER, M.W., BAIG, S. (2002): A retrospective review of balloon valvuloplastiy for the treatment of pulmonic stenosis in 50 dogs. J Vet Internal Med 16, 341.

HÄGGSTRÖM, J., HAMLIN, R.L., HANSSON, K., KVART, C. (1996): Heart-rate variability in relation to severity of mitral regurgitation in the Cavalier King Charles Spaniel. J Small Anim Pract 37, 69–75.

HÄGGSTRÖM, J., HANSSON, K., KVART, C., SWENSON, L. (1992): Chronic valvular disease in the Cavalier King Charles Spaniel in Sweden. Vet Rec 131, 549–553.

HÄGGSTRÖM, J., KVART, C., PEDERSEN, H.D. (2005): Acquired valvular heart disease. In: ETTINGER, S.J., FELDMAN, E.C. (eds.): Textbook of Veterinary Internal Medicine, 6th ed., Elsevier Saunders, Philadelphia, p. 1022–1039.

HARPSTER, N.K. (1983): Boxer cardiomyopathy. In: KIRK, R.E. (ed.): Current Veterinary Therapy. 8th ed., Saunders, Philadelphia, p. 329–337.

HARVEY, R.C., ETTINGER, S.J. (2007): Cardiovascular Disease. In: TRANQUILLI, W.J., THURMON, J.C., GRIMM, K.A. (eds.): Lumb and Jones´ Veterinary Anesthesia and Analgesia. 4th ed., Blackwell Publishing, Ames, p. 891–898.

HASKIN, S.C. (2007): Monitoring anesthetized patients. In: TRANQUILLI, W.J., THURMON, J.C., GRIMM, K.A. (eds.): Lumb and Jones´ Veterinary Anesthesia and Analgesia. 4th ed., Blackwell Publishing, Ames, p. 533–558.

HASKINS, S.C., PATZ, J.D., FARVER, T.B. (1986): Xylazine and xylazine-ketamine in dogs. Am J Vet Res 47, 636–641.

HAYASHI, Y., SUMIKAWA, K., MAZE, M. (1991): Dexmedetomidine prevents epinephrine induced arrhythmias through stimulation of central alpha-2 adrenoceptors in halothane anaesthetized dogs. Anesthesiol 75, 113–117.

HENDRIX, P.K., ROBINSON, E.P. (1997): Effects of a selective and a nonselective muscarinic cholinergic antagonist on heart rate and intestinal motility in dogs. J Vet Pharmacol Therapeut 20, 387–395.

HOSSACK, K.F. (1987): Cardiovascular response to dynamic exercise. Cardiol Clin 5, 147–156.

ILKIW, J.E., PASCOW, P.J., HASKINS, S.C., PATZ, J.D. (1992): Cardiovascular and respiratory effects of propofol administration in hypovolemic dogs. Am J Vet Res 52, 2323–2327.

JONES, D.J., STEHLING, L.C., ZAUDER, H.L. (1979): Cardiovascular response to diazepam and midazolam maleat in the dog. Anesthesiol 51, 430–434.

KANNO, N., KUSE, H., KAWASAKI, M., HARA, A., KANO, R., SASAKI, Y. (2007): Effects of pimobendan for mitral valve regurgitation in dogs. J Vet Sci 69, 373–377.

KANTELIP, J.P., ALATIENNE, M. GUEORGUIEV, G., DUCHENE-MARULLAZ, P. (1985): Chronotropic and dromotropic effects of atropine and hyoscine methobromide in unanaesthetized dogs. Br J Anaesth 57, 214–219.

KELLY, R.A., SMITH, T.W. (1996): Pharmacological treatment of heart failure. In: GOODMAN, L.S., GILMAN, A.G. (eds.): The Pharmacological Basis of Therapeutics. 9th ed., Macmillan, New York, p. 809–838.

KERR, D.D., JONES, E.W., HOLBERT, D., HUGGINS, K. (1972): Sedative and other effects of xylazine given intravenously to horses. Am J Vet Res 33, 525–532.

KITTLESON, M. (2005): Feline myocardial disease. In: ETTINGER, S.J., FELDMAN, E.C. (eds.): Textbook of Veterinary Internal Medicine. 6th ed., Elsevier Saunders, Philadelphia, p. 1082–1104.

KITTLESON, M., KIENLE, R.D. (1998): Congential pulmonic stenosis. In: KITTLESON, M., KIENLE, R.D. (eds.): Small Animal Cardiovascular Medicine, Mosby, St. Louis, p. 248–250.

KLIDE, A.M. (1976): Cardiovascular effects of enflurane and isoflurane in the dog. Am J Vet Res 37, 127–131.

KLIDE, A.M., CALDERWOOD, H.W., SOMA, L.R. (1975): Cardiopulmonary effects of xylazine in dogs. Am J Vet Res 36, 931–935.

KULKA, P.J., BREMER, F., SCHUTTLER, J. (1993): Narkoseeinleitung mit Etomidat in Lipidemulsion. Anästhesist 42, 205–209.

KUUSELA, E., VAINIO, O., SHORT, C.E. (2003): A comparison of propofol infusion and propofol/isoflurane anesthesia in dexmedetomidine premedicated dogs. J Vet Pharmacol Therapeut 26, 199–204.

LAMONT, L.A., MATHEWS, K.A. (2007): Opioids, nonsteroidal anti-inflammatories, and analgetic adjuvants. In: TRANQUILLI, W.J., THURMON, J.C., GRIMM, K.A. (eds.): Lumb and Jones´ Veterinary Anesthesia and Analgesia. 4th ed., Blackwell Publishing, Ames, p. 241–271.

LEMKE, K.A. (2007): Anticholinergics and sedatives. In: TRANQUILLI, W.J., THURMON, J.C., GRIMM, K.A. (eds.): Lumb and Jones´ Veterinary Anesthesia and Analgesia. 4th ed., Blackwell Publishing, Ames, p. 203–239.

LEMKE, K.A., TRANQUILLI, W.J. (1994): Anesthetics, arrhythmias, and myocardial sensitization to epinephrine. J Am Med Assoc 205, 1679–1684.

LIN, H.-C. (2007): Dissociative anesthetics. In: TRANQUILLI, W.J., THURMON, J.C., GRIMM, K.A. (eds.): Lumb and Jones´ Veterinary Anesthesia and Analgesia. 4th ed., Blackwell Publishing, Ames, p. 301-353.

LOMBARD, C.W., JÖNS, O., BUSSADORI, C.M. (2006): Clinical efficacy of pimobendan versus benazepril for the treatment of acquired atrioventricular valvular disease in dogs. J Am Anim Hosp Assoc 42, 249–261.

LORD, P., ERIKSSON, A., HÄGGSTRÖM, J., JÄRVINEN, A.K., KVART, C., HANSSON, K, MARIPUU, E., MÄKELÄ, O. (2003): Increased pulmonary transit times in asymptomatic dogs with mitral regurgitation indicate decreased pump function before failure. J Vet Internal Med 17, 824–829.

LOWE, D., HETTRICK, D.A., PAGEL, P.S., WARLTIER, D.C. (1996): Propofol alters left ventricular afterload as evaluated by aortic input impedance in dogs. Anesthesiol 84, 368–376.

LÖSCHER, W. (2003a): Pharmaka mit Wirkung auf das autonome (vegetative) Nervensystem. In: LÖSCHER, W., UNGEMACH, F.R., KROKER, R. (Hrsg.): Pharmakotherapie bei Haus- und Nutztieren. 6. Aufl., Parey, Berlin, S. 27–47.

LÖSCHER, W. (2003b): Pharmaka mit Wirkung auf das Zentralnervensystem. In: LÖSCHER, W., UNGEMACH, F.R., KROKER, R. (Hrsg.): Pharmakotherapie bei Haus- und Nutztieren. 6. Aufl., Parey, Berlin, S. 55–108.

MALAN, T.P., DiNARDO, J.A., ISNER J. (1995): Cardiovascular effects of sevoflurane compared with those of isoflurane in volunteers. Anesthesiol 83, 918–928.

MARKS, S.L., ABBOTT, J.A. (1998): Critical care cardiology. Vet Clin North Am: Small Anim Pract 28, 1567–1593.

MARTIN, M.F., LIMA, J.R., EZQUERRA, L.J., CARRASCO, M.S., USON-GARGALLO, J. (2001): Prolonged anesthesia with desflurane and fentanyl in dogs during conventional and laparoscopic surgery. J Am Med Assoc 219, 941–945.

MARTINEZ, E.A., HARTSFIELD, S.M., MELENDEZ, L.P., MATTHEWS,
N.S., SLATER, M.R. (1997): Cardiovascular effects of buprenorphine in anesthetized dogs. Am J Vet Res 58, 1280–1284.

MERIN, R.G., BERNARD, J.M., DOURSOUT, M.F., COHEN, M., CHELLY, J.E. (1991): Comparison of the effects of isoflurane and desflurane on cardiovascular dynamics and regional blood flow in the chronically instrumented dog. Anesthesiol 74, 568–574.

MEURS, K.M. (2005): Primary myocardial disease in the dog. In: ETTINGER, S.J., FELDMAN, E.C. (eds.): Textbook of Veterinary Internal Medicine. 6th ed., Elsevier Saunders, Philadelphia, p. 1077–1082.

MONNET, E., ORTON, C.E., SALMAN, M., BOON, J. (1995): Idiopathic dilated cardiomyopathy in dogs: survival and prognostic indicators. J Vet Internal Med 9, 12–17.

MUIR, W., LERCHE, P., WIESE, A., NELSON, L., PASLOSKE, K., WHITTEM, T. (2008): Cardiorespiratory and anesthetic effects of clinical and supraclinical doses of alfaxalone in dogs. Vet Anaesth Analg 35, 451–462.

MUIR, W., LERCHE, P., WIESE, A., NELSON, L., PASLOSKE, K., WHITTEM, T. (2009): The cardiorespiratory and anesthetic effects of clinical and supraclinical doses of alfaxalone in cats. Vet Anaesth Analg 36, 42–54.

MUIR, W.W., SKARDA, R.T., SHEEHAN, W. (1979): Hemodynamic and respiratory effects of a xylazine-acetylpromazine drug combination in horses. Am J Vet Res 40, 1518–1522.

MUIR, W.W., WERNER, L.L., HAMLIN. R.L. (1975): Effects of xylazine and acetylpromazine upon induced ventricular fibrillation in dogs anesthetized with thiamylal and halothane. Am J Vet Res 36, 1299–1303.

MUZI, M., BERENS, R.A., KAMPINE, J.P., EBERT, T.J. (1992): Venodilation contributes to propofol-mediated hypotension in humans. Anesth Analg 74, 877–883.

NAGEL, M.L., MUIR, W.W., NGUYEN, K. (1979): Comparison of the cardiopulmonary effects of etomidate and thiamylal in dogs. Am J Vet Res 40, 193–196.

NAKAMURA, R.K., RISHNIW, M., KING, M.K., SAMMARCO, C.D. (2011): Prevalence of echocardiographic evidence of cardiac disease in apparently healthy cats. J Feline Med Surg 13, 266–271.

O´GRADY, M.R., HOLMBERG, D.L., MILLER, C.W., COCKSHUTT, J.R. (1989): Canine congenital aortic stenosis: a review of the literature and commentary. Can Vet J 30, 811–815.

OPIE, L.H., DIMARCO, J.P., GERSH, B.J. (2009): Antiarrhythmic drugs and strategies. In: OPIE, L.H., GERSH, B.J. (eds.): Drugs for the heart. 7th ed., Saunders Elsevier, Philadelphia, p. 235–292.

PADDLEFORD, R., ERHARDT, W. (1992a): Anticholinergika und Anästhetika zur Prämedikation von Allgemeinanästhesie. In: PADDLEFORD, R., ERHARDT, W. (Hrsg.): Anästhesie bei Kleintieren. Schattauer, Stuttgart, S. 19–33.

PADDLEFORD, R., ERHARDT, W. (1992b): Allgemeinanästhesie. In: PADDLEFORD, R., ERHARDT, W. (Hrsg.): Anästhesie bei Kleintieren, Schattauer, Stuttgart, S. 37–87.

PAGEL, P.S., KAMPINE, J.P., SCHMELING, W.T., WARLTIER, D.C. (1991): Influence of volatile anesthetics on myocardial contractility in vivo: desflurane versus isoflurane. Anesthesiol 74, 900–907.

PASCOE, P.J. (2005): Anaesthesia for patients with cardiovascular disease. Proc. AVA Spring meeting, Rimini, Italy, p. 32–41.

PATTERSON, D.F. (1968): Epidemiologic and genetic studies of congenital heart disease in the dog. Circulation Res 23, 171–203.

PICKER, O., SCHEEREN, T.W., ARNDT, J. (2001): Inhalation anaesthetics increase heart rate by decreasing cardiac vagal activity in dogs. Br J Anaesth 87, 748–754.

PLUMB, D.C. (2005): In: PLUMP, D.C. (ed.): Plumb´s Veterinary Drug Handbook. 5th ed., Blackwell Publishing, Ames, p. 752.

POLIAC, L.C., BARRON, M., MARON, B.J. (2006): Hypertrophic cardiomyopathy. Anesthesiol 104, 183–192.

POSNER, L.P. (2007): Pre-anaesthetic assessment. In: SEYMOR, C., DUKE-NOVAKOVSKI, T. (eds.): BSAVA Manual of Canine and Feline Anaesthesia and Analgesia. 2nd ed., BSAVA, Gloucester, p. 6–11.

PYLE, R.L. (1983): Congenital heart disease. IN: ETTINGER, S.J. (ed.). Textbook of Veterinary Internal Medicine, 2nd ed., Saunders, Philadelphia, p. 940–944.

PYLE, R.L., PATTERSON, D.F., CHACKO, S. (1976): The genetic and pathology of discrete subaortic stenosis in the Newfoundland dog. Am Heart J 92, 324–334.

PYPENDOP, B.H., ILKIW, J.E. (2005): The effects of intravenous lidocaine administration on the minimal alveolar concentration of isoflurane in cats. Anesth Analg 100, 97–101.

PYPENDOP, B., VERSTEGEN, J. (1998): Hemodynamic effects of medetomidine in the dog: a dose titration study. J Vet Surg 27, 612–622.

RENDONDO, J.I., GOMEZ-VILLAMANDOS, R.J., SANTISTEBAN, J.M., DOMINGUEZ, J.M., RUIZ, I., AVILA, I. (1999): Romifidine, medetomidine, or xylazine before propofol-halothane-N2O anesthesia in dogs. Can J Vet Res 63, 31–36.

RICHARDS, D.L., CLUTTON, R.E., BOYD, C. (1989): Electrocardiographic findings following intravenous glycopyrrolate to sedated dogs: a comparison with atropine. J Assoc Vet Anaesth 16, 46–50.

RICHARDSON, P., McKENNA, W., BRISTOW, M., MAISCH, B., MAUTNER, B., O´CONNELL, J., OLSEN, E., THIENE, G. (1996): Cardiovascular News. Circulation 93, 841–842.

RIVENES, S., LEWIN, M.B., STAYER, S.A., BENT, S.T., SCHOENIG, H.M., McKENZIE, E.P., FRASER, C.D., ANDROPOULOS, D.B. (2001): Cardiovascular effects of sevoflurane, isoflurane, halothane, and fentanyl-midazolam in children with congenital heart disease: an echocardiographic study of myocardial contractility and hemodynamics. Anesthesiol 94, 223–229.

ROBERTSON, S. (1992): Advantages of etomidate use as an anesthetic agent. Vet Clin North Am: Small Anim Pract 22, 277–280.

SABBAH, H.N., SHIMOYAMA, H., KONO, T., GUPTA, R.C., SHAROV, V.G., SCICLI, G., LEVINE, T.B., GOLDSTEIN, S. (1994): Effects of long-term monotherapy with enalpril, metoprolol, and digoxin on the progression of left ventricular dysfunction and dilation in dogs with reduced ejection fraction. Circulation 89, 2852–2859.

SAVOLA, J. (1989): Cardiovascular actions of medetomidine and their reversal by atipamezole. Acta Anaesthesiol Scand 85, 39–47.

SCHÜTZ, W. (1998): Pharmakologie des kardiovaskulären Systems: Das Herz. In: FORTH, W., HENSCHLER, D., RUMMEL, U. (Hrsg.): Allgemeine und spezielle Pharmakologie und Toxikologie. 7. Aufl., Spektrum akademischer Verlag, Heidelberg, S. 363–406.

SCHWARTZ, L.S., GOLDFISCHER, J., SPRAGUE, G.H., SCHWARTZ, S.P. (1969): Syncope and sudden death in aortic stenosis. Am J Cardiol 23, 647–658.

SELLGREN, J., EJNELL, H., ELAM, M., PONTEN, J., WALLIN, B.G. (1994): Sympathetic muscle nerve activity, peripheral blood flows, and baroreceptor reflexes in humans during propofol anesthesia and surgery. Anesthesiol 80, 534–544.

SHIRAN, A., SAGIE, A. (2009): Tricuspid regurgitation in mitral valve disease: incidence, prognostic implications, mechanism, and management. J Am Coll Cardiol 53, 401–408.

SHORT, C.E., PADDLEFORD, R.R., CLOYD, G.D. (1974): Glycopyrrolate for prevention of pulmonary complications during anaesthesia. Modern Vet Pract 55, 194–196.

SISSON, D., O´GRADY, M.R., CALVERT, C.A. (1999): Myocardial diseases in dogs. In: FOX, P.R., SISSON, D., MOISE, N. (eds.): Textbook of Canine and Feline Cardiology. 2nd ed., Saunders, Philadelphia, p. 581–619.

SISSON, D., KVART, C.P. (1999): Acquired valvular heart disease in dogs and cats. In: FOX, P.R., SISSON, D., MOISE, N. (eds.): Textbook of Canine and Feline Cardiology. 2nd ed., Saunders, Philadelphia, p. 536–555.

SISSON, D., THOMAS, W., KEEN, B. (2000): Primary myocardial disease in the dog. In: ETTINGER, S.J., FELDMAN, E. (eds.): Textbook of Veterinary Internal Medicine. 5th ed., Saunders, Philadelphia, p. 874–895.

SKARDA, R.T., BEDNARSKI, R.M., MUIR, W.W., HUBBELL, J.A.E., MASON, D.E. (1995a): Sedation und Narkose bei Hund und Katze mit Herzkreislaufkrankheit 1. Teil: Narkoseplanung nach Risikobeurteilung, hämodynamische Wirkung der Pharmaka, Monitoring. Schweiz Arch Tierheilk 137, 312–321.

SKARDA, R.T., BEDNARSKI, R.M., MUIR, W.W., HUBBELL, J.A.E., MASON, D.E. (1995b): Sedation und Narkose bei Hund und Katze mit Herzkreislaufkrankheit 2. Teil: Narkoseplanung anhand der Pathophysiologie, Herzarrhythmien. Schweiz Arch Tierheilk 137, 543–551.

SLEEPER, M., HENTHORN, P., VIJAYASARATHY, C., DAMBACH, D., BOWERS, T., TIJSKENS, P., ARMSTRONG, C., LANKFORD, E. (2002): Dilated cardiomyopathy in juvenile Portuguese Water Dogs. J Vet Internal Med 16, 52–62.

STAFFORD JOHNSON, M., MARTIN, M. (2004): Results of balloon valvuloplasty in 40 dogs with pulmonic stenosis. J Small Anim Pract 45, 148–153.

STEFFEY, E.P., HOWLAND, D. (1977): Isoflurane potency in the dog and cat. Am J Vet Res 38, 1833–1836.

STEFFEY, E.P., HOWLAND, D. (1978): Potency of enflurane in dogs: Comparison with halothane und isoflurane. Am J Vet Res 39, 673–677.

STEFFEY, E.P., HOWLAND, D. (1980): Comparison of circulatory and respiratory effects of isoflurane and halothane anesthesia in horses. Am J Vet Res 41, 821–825.

STEFFEY, E.P., MAMA, K.R., FRANK, D.G., PUSCHNER, B., WOLINER, M.J. (2005): Effects of sevoflurane dose and mode of ventilation on cardiopulmonary function and blood biochemical variables in horses. Am J Vet Res 66, 606–614.

THRUSFIELD, M.V. (1986): Epidemiologic studies using computerized data bases. III. Various examples of epidemiological studies using computerized data bases. Tijdschrift Diergeneesk 111, 384–388.

THRUSFIELD, M.V. AITKEN, C.G., DARKER, G. (1985): Observations on breed and sex in relation to canine heart valve incompetence. J Small Anim Pract 26, 709–717.

TIDHOLM, A. (1997): Retrospective study of congenital heart defects in 151 dogs. J Small Anim Pract 38, 94–98.

TIDHOLM, A., JÖNSSON, L. (1997): A retrospective study of canine dilated cardiomyopathy (189 cases). J Am Anim Hosp Assoc 33, 544-550.

TRAUTVETTER, E., NEU, H., SCHNEIDER, M., SKRODZKI, M., HOLMBERG, D.L. (2007): Kongenitale Herzerkrankungen. In: GRÜNBAUM, E., SCHIMKE, E. (Hrsg.): Klinik der Hundekrankheiten. 3. Aufl., Enke, Stuttgart, S. 373–442.

TRANQUILLI, W., THURMAN, J., BENSON, G. (1988): Alterations in epinephrine-induced arrhythmogenesis after xylazine and subsequent yohimbine administration in isoflurane anaesthetized dogs. Am J Vet Res 49, 1072–1075.

URABE, Y., MANN, D.L., KENT, R.L., NAKANO, K., TOMANEK, R.J., CARABELLO, B.A., COOPER, G. (1992): Cellular and ventricular contractile dysfunction in experimental canine mitral regurgitation. Circulation Res 70, 131–147.

VAINIO, O., PALMU, L. (1989): Cardiovascular and respiratory effects of medetomidine in dogs and influence of anticholinergics. Acta Vet Scand 30, 401–408.

VICKERY, R., SHERIDAN, B., SEGAL, I. (1988): Anesthetic and haemodynamic effects of the stereoisomeres of medetomidine, an alpha 2 adrenergic agonist, in halothane anesthetized dogs. Anesth Analg 67, 611–615.

VILLALBA, M., SANTIAGO, I., GOMEZ DE SEGURA, A. (2011): Effects of constant rate infusion of lidocaine and ketamine, with or without morphine, on isoflurane MAC in horses. Equine Vet J, doi: 10.111/j.2042-3306.2011.00355.x.

WAGNER, A.E., DUNLOP, C.I., CHAPMAN, P.L. (1993): Effects of ephedrine on cardiovascular function and oxygen delivery in isoflurane-anesthetized dogs. Am J Vet Res 54, 1917–1922.

WARE, W.A. (2006): Kardiovaskuläre Erkrankungen. In: NELSON, R.W., COUTO, C.G. (Hrsg.): Innere Medizin der Kleintiere. 6. Aufl., Elsevier, München, S. 159–178.

WARLTIER, D.C., PAGEL, P.S. (1992): Cardiovascular and respiratory actions of desflurane: is desflurane different from isoflurane? Anesth Analg 75, 17–31.

ZSIGMOND, E.K., KELSCH, R.C., KOTHARY, S.P. (1974): Rise in plasma free-norepinephrine during anesthetic induction with ketamine. Behav Neuropsychiatry 6, 81–84.