



비엔동물전문의료센터
BIEN ANIMAL MEDICAL CENTER

비엔 마취 세미나 : 심질환에 따른 마취 전략 (정석과 꿀팁)

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Previous seminar

Pain management

- ☐ Definition of pain
- ☐ Pain evaluation
- ☐ Classification of pain
- ☐ Duration of pain
- ☐ Type of analgesic drugs

Local anesthesia technique

- ☐ Infiltration
- ☐ Dental
 - ☐ Infra-orbital nerve block
 - ☐ Mental nerve block
 - ☐ Mandibular nerve block
- ☐ Forelimb
 - ☐ RUMM nerve block
- ☐ Hindlimb
 - ☐ Femoral and sciatic nerve block
- ☐ Epidural anesthesia
- ☐ Adjunctive of local-anesthesia

Consideration of cardiovascular disease patients

Before anesthesia

- ☐ ECG
- ☐ Thoracic radiography
- ☐ Echocardiography
- ☐ Blood-based cardiac markers

During anesthesia

- ☐ Background
- ☐ Drugs
- ☐ MMVD
- ☐ VSD and PDA
- ☐ HCM and RCM
- ☐ Pericardial effusion
- ☐ Tachycardia
- ☐ Bradycardia

Abbreviations

- ACEIs: angiotensin-converting enzyme inhibitors
- AF: atrial fibrillation
- AiVR: accelerated idioventricular rhythm
- AV: atrioventricular
- BP: blood pressure
- CHF: congestive heart failure
- CO: cardiac output
- CO₂: carbon dioxide
- DCM: dilated cardiomyopathy
- GDV: gastric dilation-volvulus
- HCM: hypertrophic cardiomyopathy
- HR: heart rate
- LV: left ventricle
- LVOT: left ventricular outflow tract
- MMVD: myxomatous mitral valve degeneration
- MR: mitral valve regurgitation
- NO: nitric oxide
- PDA: patent ductus arteriosus
- PEEP: positive end-expiratory pressure
- PGs: prostaglandin
- PHT: pulmonary hypertension
- PS: pulmonic stenosis
- PNS: parasympathetic nervous system
- PVR: pulmonary vascular resistance
- rPDA: reverse patent ductus arteriosus
- RV: right ventricle
- SAM: systolic anterior motion
- SAS: subaortic stenosis
- SH: systemic hypertension
- SNS: sympathetic nervous system
- SVR: systemic vascular resistance
- SVT: supraventricular tachycardia
- TEE: transesophageal echocardiography
- TF: tetralogy of Fallot
- VF: ventricular fibrillation
- VHS: vertebral heart size
- VPC: ventricular premature complex
- VT: ventricular tachycardia

Introduction

In animal with (**suspected**) **cardiac disease**, care must be taken both **prior to** and **during anesthesia to assess the heart's ability**

Why?

1. Provide adequate cardiac output and tissue perfusion
2. Maintain low venous pressures and prevent congestion
3. Avoid arrhythmia

How?

1. The medical history
2. Physical exam
 - ① Auscultation (cardiac and respiratory)
 - ② Inspection of the jugular vein
 - ③ Peripheral arterial pulse
3. Electrocardiography (ECG)
4. Thoracic radiography
5. Echocardiography
6. Non-invasive blood pressure measurement
7. Biomarker (NTproBNP and cTnI)

Pre-anesthetic evaluation

- ECG

ECG is the **gold standard** for the evaluation of **cardiac arrhythmia**
(not good at the absence of arrhythmia)

What?

1. Heart rate (HR)
2. Mean electrical axis (Fig 1.)
3. Rhythm
4. Criteria for cardiac chamber enlargement

Impact of anesthesia

1. Abnormalities of HR (brady- or tachy-cardia)
2. Criteria for cardiac chamber enlargement

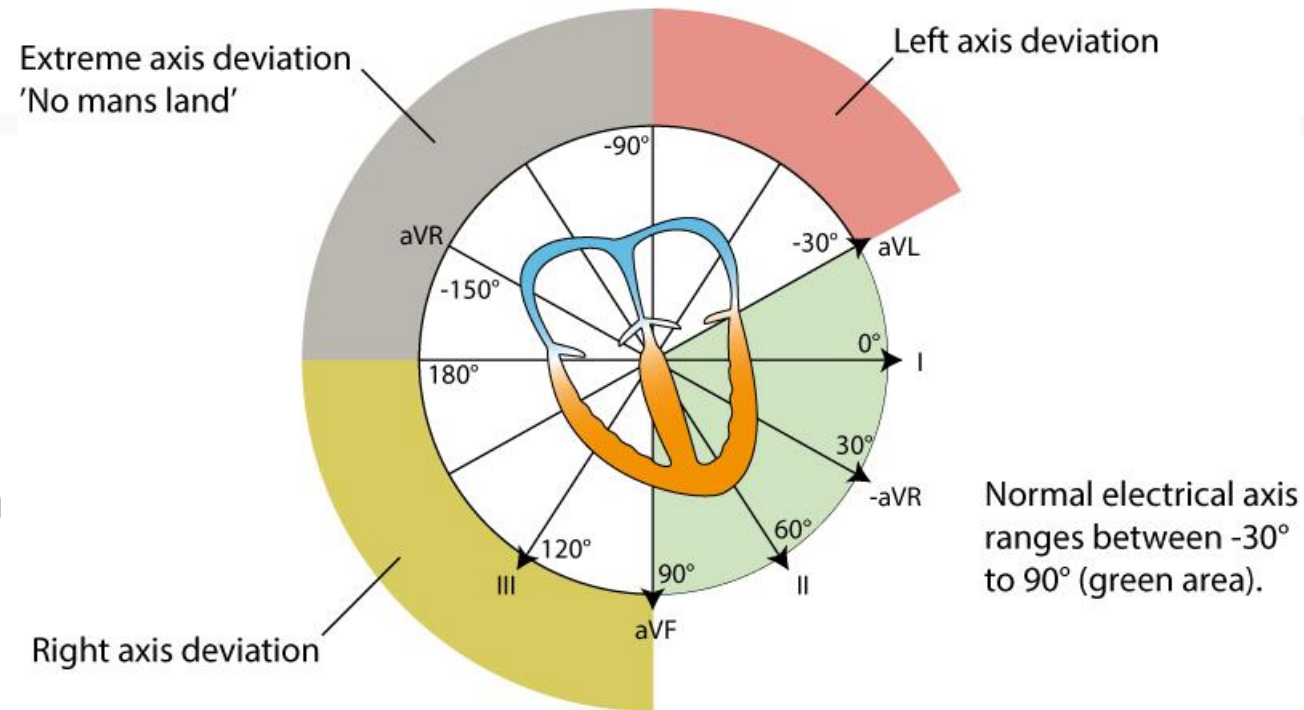


Fig. 1 The electrical axis of the heart

Pre-anesthetic evaluation

- ECG

Bradycardia

1. 2nd and 3rd-degree atrioventricular nodal (AV) block
2. Sinus arrest or block
3. Sinus bradycardia
4. Atrial standstill
5. Sick sinus syndrome

Treatment

1. **Vagolytic agents** (glycopyrrolate or atropine)
 - 1-1. Complete resolution
 - 1-2. Repeated administration as needed

-
- 2-1. Incomplete resolution
 - 2-2. Absence of response to vagolytic agents → often indicate injury or disease → Additional exams

Low risk for anesthesia

(vagally mediated cause)

1. Sinus block
2. Sinus bradycardia
3. Respiratory sinus arrhythmia

High risk for anesthesia

(primary cardiac disease or severe electrolyte disturbance)

1. High grade 2nd AV block
2. 3rd AV block
3. Atrial standstill

Parameter	Dog	Cat
Heart rate (beats/min)	70–160	140–240
P wave duration (seconds)	<0.04	<0.04
P wave amplitude (mV)	<0.4	<0.2
P–Q interval (seconds)	0.06–0.13	0.05–0.09
QRS duration (seconds)	<0.06	<0.04
R wave amplitude (mV)	<2.5 (<3.0 in giant breeds)	<0.9
T wave character	<25% QRS complex height, can be negative in lead II	
Q–T interval (seconds)	0.15–0.25	0.12–0.18

21.25

Reference intervals for electrocardiographic measurements in the dog and the cat.

BSAVA manual of canine and feline anaesthesia and analgesia, 3rd edition, BSAVA

Pre-anesthetic evaluation

- ECG

Tachycardia

1. Supraventricular tachycardia (SVT)
2. Ventricular tachycardia (VT)
3. Atrial fibrillation (AF)
1~3 → From primary cardiac disease (MMVD, cardiomyopathy; DCM, HCM)
4. Atrial flutter
5. Ventricular arrhythmia (AiVR)
5 → Extra-cardiac disease (splenic and liver neoplasia or GDV) → Need additional exam (abdominal)

What cause the sinus tachycardia?

1. Pain
2. Hyperthyroidism
3. Congestive heart failure
4. Drug/toxin (theophylline, terbutaline, and theobromine)

What tachycardia caused?

: Decrease diastolic filling time and cardiac output (CO)

Rhythm	Underlying cause	Treatment	Dose and dosing interval	Potential adverse effects
Bradyarrhythmias				
Sinus bradycardia	Anaesthetic drugs Anaesthetic overdose	Reduce or antagonize causative agent Consider treatment with anticholinergics	Atropine: 0.02–0.04 mg/kg i.v. Glycopyrronium: 0.005–0.01 mg/kg i.v. Repeat after 10–15 minutes if no response	Possibility that depth of anaesthesia will lighten Reduced efficacy with hypothermia Low doses may increase severity of bradycardia in the short term
	Hypertension (particularly sudden onset) Pain or noxious stimuli, vasoconstricting agents or phaeochromocytoma	Provide appropriate analgesia Administer vasodilators	Acepromazine: 0.005–0.01 mg/kg i.v. Phentolamine: 0.025–0.1 mg/kg i.v. Repeat phentolamine every 5–20 minutes if needed	Some analgesic agents (e.g. opioids, alpha-2 adrenoceptor agonists) may reduce heart rate further May reduce blood pressure without a concurrent increase in heart rate
	Hypothermia	Use external warming devices and consider warm lavage fluids to re-warm the patient	Continuous warming from anaesthetic induction is recommended. Minimize anaesthetic length to reduce degree of hypothermia	Avoid excessive heat to prevent burns and use equipment according to manufacturers' instructions
	Vagal reflex	Identify cause of stimulation and cease Consider treatment with anticholinergics	Atropine: 0.02–0.04 mg/kg i.v. Glycopyrronium: 0.005–0.01 mg/kg i.v. Repeat after 10–15 minutes if no response	Reduced efficacy with hypothermia Low doses may increase severity of bradycardia in the short term
	Hyperkalaemia	Calcium gluconate 10% Dextrose Dextrose ± insulin Sodium bicarbonate	Calcium gluconate: 50–100 mg/kg (0.5–1.0 ml/kg of 10% solution) i.v. over 10 minutes Dextrose: 0.5–1.0 g/kg i.v. over 5 minutes Regular insulin, 0.25–0.5 IU/kg i.v. with dextrose, 1–2 g per unit of insulin (2–4 ml of 50% dextrose, diluted into 8–16 ml of fluids, respectively, to make a 10% solution) Sodium bicarbonate: 1–2 mEq/kg i.v. over 15–30 minutes	Calcium may induce arrhythmias if administered rapidly Possible hypoglycaemia with insulin Electrolyte imbalances and volume overload with sodium bicarbonate
	Hypoglycaemia	Dextrose	0.5–1.0 g/kg i.v. over 5 minutes 2.5–5% dextrose i.v. infusion if prolonged hypoglycaemia is expected	Administration of concentrated (>10%) dextrose in peripheral veins can result in phlebitis
	Severe acidosis (pH <7.0)	Sodium bicarbonate	1–2 mEq/kg i.v. over 15–30 minutes	Electrolyte imbalances and volume overload with sodium bicarbonate
	Severe hypoxia	Increase inspired oxygen concentration and consider ventilation	Use 100% oxygen initially Reduce inspired concentration for long-term use	May not respond if severe right-to-left shunting is present. High inspiratory pressures may worsen haemodynamics
	Raised intracranial pressure	Mannitol Hypertonic saline (7.5%)	Mannitol: 0.2–2.0 g/kg i.v. over 30 minutes Hypertonic saline: 2–4 ml/kg i.v. over 20 minutes Repeat after 4–8 hours if required	Volume overload, electrolyte imbalances and acidosis
Second-degree AV block	High vagal tone	Anticholinergics	Atropine: 0.02–0.04 mg/kg i.v. Glycopyrronium: 0.005–0.01 mg/kg i.v. Repeat after 10–15 minutes if no response	Anticholinergics administered after alpha-2 adrenoceptor agonists can increase myocardial workload and oxygen consumption Reduced efficacy with hypothermia Low doses may increase severity of bradycardia in the short term
	Anaesthetic drugs: Opioids	Anticholinergics	Atropine: 0.02–0.04 mg/kg i.v. Glycopyrronium: 0.005–0.01 mg/kg i.v. Repeat after 10–15 minutes if no response	
	Alpha-2 adrenoceptor agonists	Antagonize alpha-2 agonists (atipamezole)	Atipamezole: use 0.25–0.5 the dose that would have been used to antagonize the administered dose of medetomidine i.m.	

Rhythm	Underlying cause	Treatment	Dose and dosing interval	Potential adverse effects
Bradycarrhythmias continued				
Third-degree AV block	Inflammatory or degenerative AV nodal disease	Pacemaker	Pacemaker placement generally recommended for lifelong support	
Sick sinus syndrome	Unknown, although often associated with DMVD and sinus node fibrosis with vascular changes often seen	Anticholinergics Pacemaker	Atropine: 0.02–0.04 mg/kg i.v. Glycopyrronium: 0.005–0.01 mg/kg i.v. Pacemaker placement generally recommended	Reduced efficacy with hypothermia Low doses may increase severity of bradycardia in the short term
Asystole and pulseless electrical activity	Many possible causes, including those listed for sinus bradycardia	Initiate cardiopulmonary resuscitation immediately Attempt to identify and treat potential underlying causes	Perform basic life support continuously until there is a positive response or there has been no response for 10 minutes If possible, perform advanced life support once basic life support has been initiated	Treatment may not be successful Success rates of treating anaesthetic cardiac arrests are higher than for non-anaesthetic cardiac arrests if treatment is instigated rapidly
Tachycarrhythmias				
Sinus tachycardia	Inadequate anaesthesia or analgesia	Increase depth of anaesthesia Provide more analgesia	Depends on anaesthetic technique being employed Consider providing additional opioids or alternative analgesia	
	Hypovolaemia or hypotension	Volume resuscitation with i.v. crystalloid or colloid therapy Vasopressors or inotropes for hypotension without hypovolaemia	Volumes of intravenous fluid therapy required depend upon degree of hypovolaemia (see Chapter 18) For doses and dosing intervals of vasopressors and inotropes, see Figure 21.13	Take care to avoid volume overload in patients in cardiac failure
	Hypercapnia	Initiate or increase positive pressure ventilation Decrease depth of anaesthesia	Positive pressure ventilation may be required for the remainder of anaesthesia A decreased depth of anaesthesia may reduce the degree of hypoventilation	High inspiratory pressures may worsen haemodynamics
	Electrolyte disturbances: Hypokalaemia Hypercalcaemia Hypomagnesaemia	Assess plasma electrolytes and then supplement or instigate appropriate treatment to correct the disturbance	Hypokalaemia: potassium supplementation with fluid therapy, not to exceed 0.5 mEq/kg/h Hypercalcaemia: diuresis with 0.9% sodium chloride, salmon calcitonin 4–6 IU/kg s.c. q8–12 hours Hypomagnesaemia: magnesium supplementation at 0.15–0.3 mEq/kg i.v. over 5–15 minutes (equivalent to 19–37 mg/kg magnesium sulphate i.v. or 16–32 mg/kg magnesium chloride i.v.)	Care should be taken with the rate of K ⁺ supplementation to avoid inadvertent hyperkalaemia Volume overload may occur with large volumes of i.v. fluid for diuresis in patients with cardiac disease
	Hypoxia or hypoxaemia	Increase inspired oxygen concentration and consider ventilation	Use 100% oxygen initially Reduce inspired concentration for long-term use	May not respond if severe right-to-left shunting is present. High inspiratory pressures may worsen haemodynamics
	Anaemia	Whole blood, packed red cells or Oxyglobin®	Chronic anaemia, if affecting haemodynamics, should be treated before anaesthesia to increase the PCV >20% Acute haemorrhage will primarily cause hypovolaemia, but support of oxygen-carrying capacity may be required secondarily Volumes of blood products or Oxyglobin® will depend upon the degree of anaemia and on the volume status of the patient	See Chapter 18 regarding potential adverse effects of blood products

Rhythm	Underlying cause	Treatment	Dose and dosing interval	Potential adverse effects
Tachyarrhythmias continued				
	Pre-existing disease, e.g. hyperthyroidism or phaeochromocytoma	Attempt to reduce catecholamine release (storms) Vagomimetics such as beta blockers or opioids may be useful	Beta blockers: see Figure 21.13 Short acting opioids: Fentanyl: 1–2 µg/kg i.v. Alfentanil: 1–2 µg/kg i.v. Remifentanyl: 0.5–1.0 µg/kg i.v.	Bronchospasm may occur with propranolol
	Drug therapy, e.g. beta-1 adrenoceptor agonists	Cease causative drug therapy		
Idioventricular rhythm	Cardiac or extracardiac disease	Usually requires no specific treatment and usually self-resolves Lidocaine may be administered, although this is rarely successful	Lidocaine i.v. bolus: 1–2 mg/kg (dogs), 0.5 mg/kg (cats) Lidocaine i.v. infusion: 25–100 µg/kg/min (dogs)	Lidocaine boluses may cause some cardiovascular depression, especially with repeated doses and any dose in cats Maximum dose of 8 mg/kg (dogs) and 2 mg/kg (cats) over 10 minutes should be administered
Atrial fibrillation	Vagotonic in large or giant breeds (especially GSD). Atrial dilation – may be present in structurally normal hearts in giant-breed dogs	Usually requires no specific treatment in the peri-anaesthetic period unless newly developed due to anaesthesia; in this case, lidocaine should be used Diltiazem or beta blockers may be useful to control heart rate in pre-existing atrial fibrillation Electrical cardioversion may be performed	Lidocaine 2 mg/kg i.v. bolus, repeated every 10 minutes up to a total dose of 8 mg/kg Beta blockers: see Figure 21.13 Diltiazem: 0.1–0.25 mg/kg i.v. bolus slowly over 1–2 minutes, repeat after 5–10 minutes up to 0.75 mg/kg Diltiazem i.v. infusion: 0.1–0.3 mg/kg/h	Lidocaine boluses may cause some cardiovascular depression, especially with repeated doses and any dose in cats Bronchospasm may occur with propranolol Diltiazem may cause a mild reduction in systolic function
Ventricular tachycardia	Cardiac or extracardiac disease	Lidocaine Magnesium may be attempted for refractory ventricular tachycardia	Lidocaine i.v. bolus: 1–2 mg/kg (dogs), 0.5 mg/kg (cats) Lidocaine i.v. infusion: 25–100 µg/kg/min (dogs) Magnesium i.v. bolus: 30 mg/kg over 10 minutes Magnesium i.v. infusion: 0.5 mmol/kg/24 hours i.v. (equivalent to 5.2 mg/kg/h)	Lidocaine boluses may cause some cardiovascular depression, especially with repeated doses and any dose in cats Maximum dose of lidocaine of 8 mg/kg (dogs) and 2 mg/kg (cats) over 10 minutes should be administered Magnesium should be given slowly i.v. and an ECG is recommended for monitoring
Ventricular fibrillation	Many possible causes, including those listed for sinus tachycardia	Initiate cardiopulmonary resuscitation immediately Attempt to identify and treat potential underlying causes Instigate electrical defibrillation if available; otherwise attempt a precordial thump	Perform basic life support continuously until there is a positive response or there has been no response for 10 minutes If possible, perform advanced life support once basic life support has been initiated External electrical defibrillation: 4–6 J/kg Internal electrical defibrillation: 0.5–1 J/kg	Treatment may not be successful Success rates of treating anaesthetic cardiac arrests are higher than for non-anaesthetic cardiac arrests if treatment is instigated quickly

Pre-anesthetic evaluation

- Thoracic radiography

Thoracic radiography is an extremely useful tool in **assessing patients with cardiac disease**.

Cardiac disease caused by neurohormonal responses



Increase cardiac preload (renal retention of fluid and sodium)



Eccentric cardiac hypertrophy



Increased radiographic heart size (VHS)



Severity and risk of **congestive** heart failure



Assess the risk of induced or spontaneous CHF

- Ventral to the carina of the trachea
- Ventral border of the caudal vena cava
- Apex of the heart
- Width of the silhouette (widest portion)
- The fourth thoracic vertebra

Normal range of VHS in dogs

- Low risk: <11.0-11.5
- High risk: >11.5-12.0

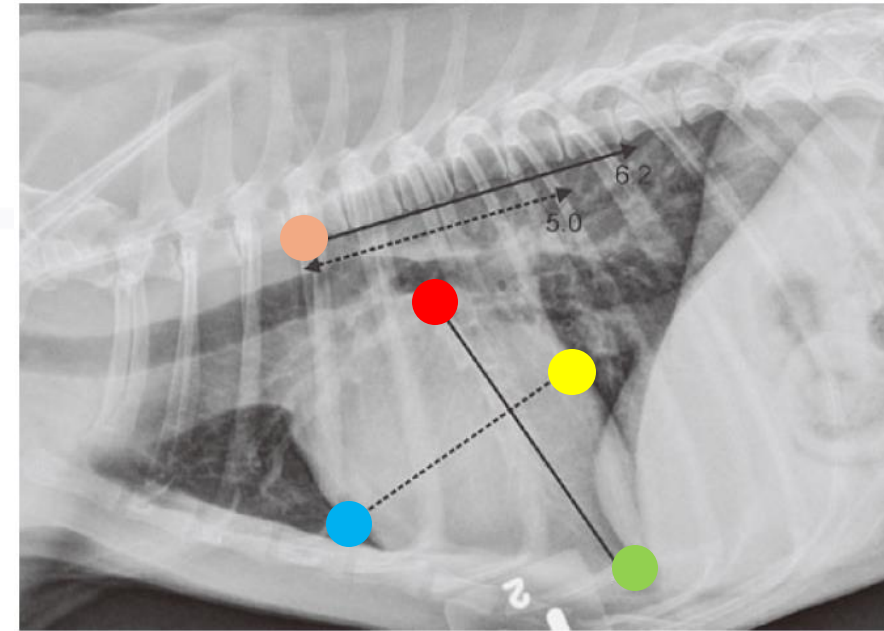


Figure 36.1 Right lateral thoracic radiograph from a 13-year-old female spayed Toy Poodle demonstrating the vertebral heart size (VHS) measurement technique. The length of the cardiac silhouette is measured from a point ventral to the carina of the trachea to the apex of the heart and the width of the silhouette is measured along the widest portion of the heart perpendicular to the long axis. The ventral border of the caudal vena cava is often used as the starting point for the width measurement. The length and width measurements are compared with the number of vertebral bodies starting from the cranial aspect of the fourth vertebral body. In this instance, the length of the silhouette is 6.2 vertebra and the width is 5.0 vertebra, yielding an overall VHS of 11.2.

Pre-anesthetic evaluation

- Echocardiography

Echocardiography: gold standard for the clinical evaluation of **cardiac structure and function** (less useful than thoracic radiography in determining the presence or likelihood CHF; pulmonary edema)

Providing data

- Ventricular and atrial **chamber dimensions**
- **Pattern of blood flow** through the heart and proximal portions of great vessel
- **Thickness** of the ventricular walls and interventricular septum
- Myocardial **contractility**
- Morphology of **valve structure**

Table 36.3 Upper and lower bounds of the 95th percentile confidence interval (CI) for indexed echocardiographic formula constants in healthy dogs. The constants multiplied by the body weight (BW in kg) raised to an exponential power provide a single reference range across all body weights for the indicated measurements.

Measurement	95% CI boundary	Formula (BW in kg)
iLVIDd	1.35–1.73	$iLVIDd = LVIDd/BW^{0.294}$
iLVIDs	0.79–1.14	$iLVIDs = LVIDs/BW^{0.315}$
iIVSd	0.33–0.52	$iIVSd = IVSd/BW^{0.241}$
iIVSs	0.48–0.71	$iIVSs = IVSs/BW^{0.240}$
iLVPWd	0.33–0.53	$iLVPWd = LVPWd/BW^{0.232}$
iLVPWs	0.53–0.78	$iLVPWs = LVPWs/BW^{0.222}$
iLAD	0.64–0.90	$iLAD = LAD/BW^{0.345}$
iAoD	0.68–0.89	$iAoD = AoD/BW^{0.341}$

Table 36.2 Common echocardiographic measurements and calculations.

Measurement	Commonly used abbreviation(s)
Left ventricle at end-diastole	
Left ventricular end-diastolic dimension; values that are indexed or normalized to the animal's body weight commonly utilize an "i" or "n" (i.e., iLVEDD or LVIDdN)	LVEDD, LVIDd, LVd
Thickness of the left ventricular posterior wall	LVPWd
Thickness of the interventricular septum	IVSd
Left ventricle at end-systole	
Left ventricular end-systolic dimension; values that are indexed or normalized to the animal's body weight commonly utilize an "i" or "n" (i.e., iLVESD or LVIDsN)	LVESD, LVIDs, LVs
Thickness of the left ventricular posterior wall	LVPWs
Thickness of the interventricular septum	IVSs
Diameter of the aortic root	AoD
Diameter of the left atrium	LAD
Ratio of the aortic to left atrium diameter	LA:Ao, LAD:AoD
Fractional shortening $[(LVEDD - LVESD)/LVEDD] \times 100\%$	FS%

Pre-anesthetic evaluation

- Echocardiography

Echocardiography: gold standard for the clinical evaluation of **cardiac structure and function**
(less useful than thoracic radiography in determining the presence or likelihood CHF; pulmonary edema)

Providing data

- Ventricular and atrial **chamber dimensions**
- **Pattern of blood flow** through the heart and proximal portions of great vessel
- **Thickness** of the ventricular walls and interventricular septum
- Myocardial **contractility**
- Morphology of **valve structure**

Example

- MMVD, DCM, PDA = volume overload
→ **Increased diastolic chamber dimension**
- HCM, SH, SAS = increased wall thickness
→ **Increased diastolic and systolic ventricular wall thickness**
- DCM = decreased contractility
→ **Increased systolic chamber dimension**

Table 36.2 Common echocardiographic measurements and calculations.

Measurement	Commonly used abbreviation(s)
Left ventricle at end-diastole	
Left ventricular end-diastolic dimension; values that are indexed or normalized to the animal's body weight commonly utilize an "i" or "n" (i.e., iLVEDD or LVIDdN)	● LVEDD, LVIDd, LVd
Thickness of the left ventricular posterior wall	● LVPWd
Thickness of the interventricular septum	● IVSd
Left ventricle at end-systole	
Left ventricular end-systolic dimension; values that are indexed or normalized to the animal's body weight commonly utilize an "i" or "n" (i.e., iLVESD or LVIDsN)	● LVESD, LVIDs, LVs
Thickness of the left ventricular posterior wall	● LVPWs
Thickness of the interventricular septum	● IVSs
Diameter of the aortic root	AoD
Diameter of the left atrium	LAD
Ratio of the aortic to left atrium diameter	LA:Ao, LAD:AoD
Fractional shortening $[(LVEDD - LVESD)/LVEDD] \times 100\%$	FS%

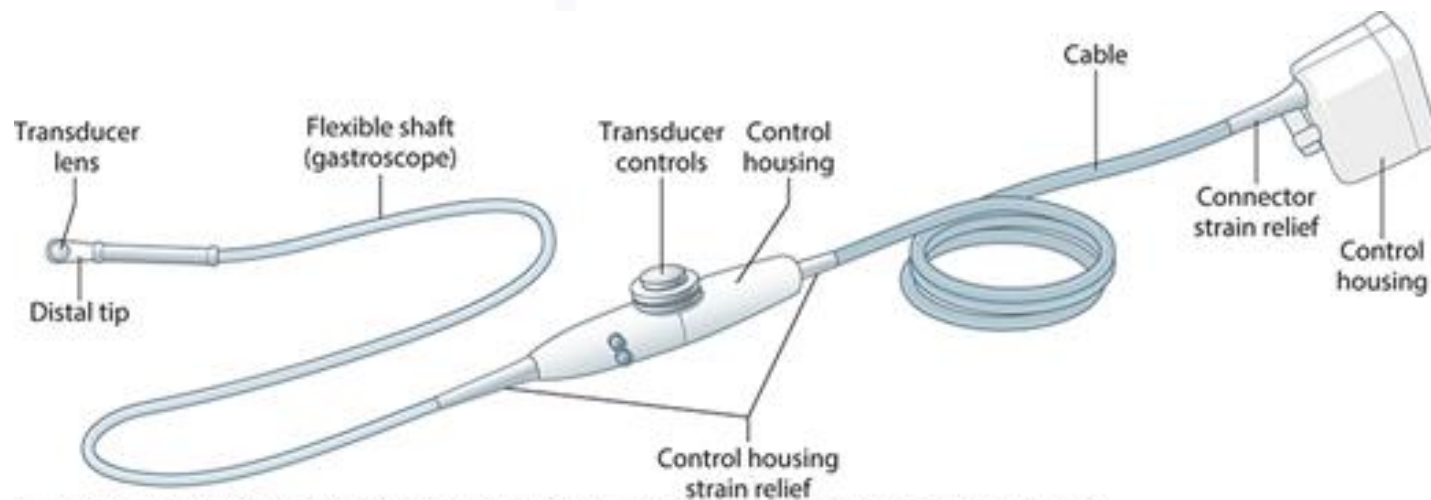
Pre-anesthetic evaluation

- Echocardiography

- **Objective measurement should align with the trained echocardiographer's subjective assessment** of cardiac morphology and function.
- **Subjective estimation** of cardiac function by experienced echocardiographers **have a better correlation** with angiographic evaluation than objective calculations.

Transesophageal echocardiography (TEE)

- Used when chest-conformation and body weight make poor-quality transthoracic image.
- Facilitate cardiac catheterization procedure (PDA occlusion, balloon valvuloplasty of PS)
- Biplane (switch two transducer) and multiplane (rotating the probe)



Source: Joseph P. Mathew, Chakib M. Ayoub, Alina Nicoara, and Madhav Swaminathan: *Clinical Manual and Review of Transesophageal Echocardiography*, 3e
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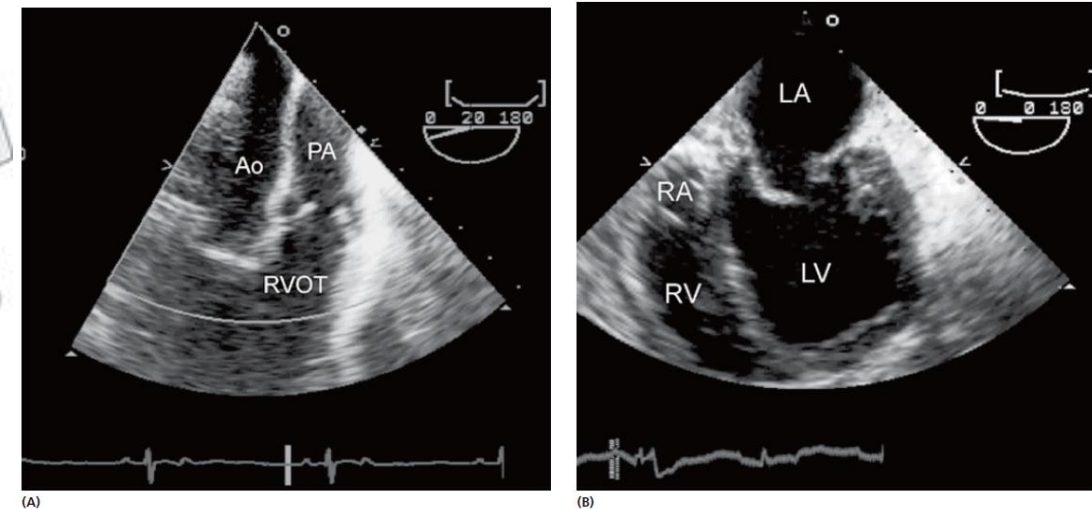


Figure 36.2 Transesophageal echocardiography (TEE) in dogs. A. Dog with congenital pulmonic stenosis showing the incomplete opening and abnormal thickness of the pulmonic valve leaflets from the cranial transverse probe position (RVOT, right ventricular outflow tract; Ao, aorta; PA, pulmonary artery). B. Dog with congenital mitral stenosis showing incomplete opening of the mitral valve leaflets and from the middle transverse position (LA, left atrium; LV, left ventricle; RV, right ventricle; RA, right atrium).

Pre-anesthetic evaluation

- **Blood-based cardiac markers** = **Warrant additional diagnostics** 

The heart is an active endocrine organ.

Sympathomimetic and RAAS → The heart (end-organ **target of neurohormonal activity**)

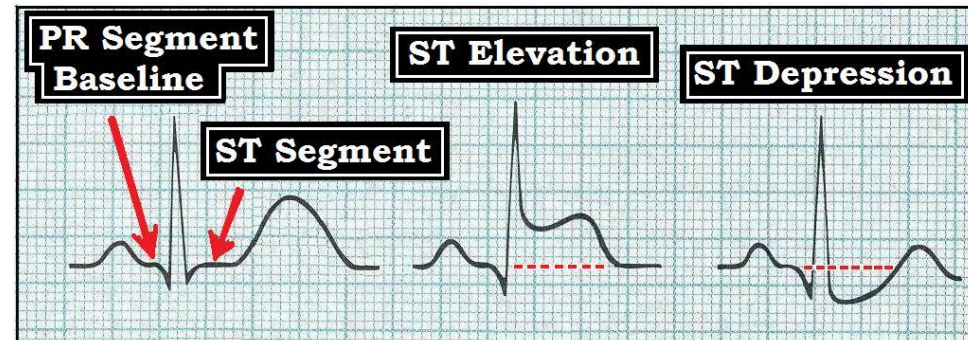
- Atrial and B-type natriuretic peptide (ANP, BNP): **source of neurohormonal production**
 - **NTproBNP**: a precursor of BNP
- Help **differentiate** cardiac versus respiratory etiology of respiratory signs.



- Murmurs
- Gallop
- Arrhythmia
- NTproBNP elevation

→ High risk for cardiomyopathy → cTnI elevation

- cTnI: markers of cardiac tissue injury (+ ST segment changes)



Background

Aim!

= **Maintaining CO and tissue perfusion** is a primary goal.

How?

= To achieve the best results, it **depends on your underlying heart condition.**

$$BP = CO \times SVR$$

$$BP = HR \times SV \times SVR$$

$$HR \sim SNS, PNS$$

$$SV \sim \text{Preload, afterload, contractility}$$

$$SVR \sim CO_2, NO, PGs, \text{histamine, vasopressin, angiotensin II, SNS}$$

Example

: **Fast heart rates** (AF, VF, VT etc.) → **Decreased diastolic filling times** → **Decreased ventricular filling**

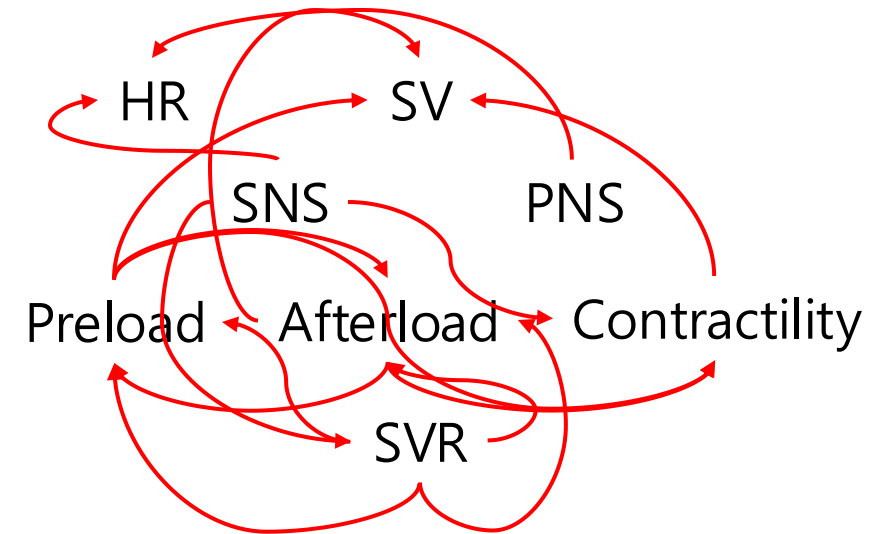


Table 8-1 Causes of Hypotension and Recommended Treatment

Cause	Sample Diseases	Treatments
Reduction in Preload		
Hypovolemia	Hemorrhage Severe dehydration Edema/cavitary effusions	Address underlying problem. Provide fluid resuscitation.
Obstructive	Gastric dilation-volvulus Mesenteric volvulus Caval/portal venous occlusion Pericardial effusion Severe pleural space disease Pulmonary thromboembolism	Relieve the obstruction if possible, with surgery, pericardiocentesis, or thoracentesis; administration of thrombolytics; or thrombectomy as needed. Provide fluid resuscitation.
Reduction in Cardiac Function		
Primary	Cardiomyopathy Valvular disease Tachyarrhythmia or bradyarrhythmia	Administer positive inotrope. Administer antiarrhythmics. Provide supportive measures for congestive heart failure
Secondary	Systemic inflammatory response syndrome/sepsis Electrolyte abnormalities Severe hypoxia Severe acidosis or alkalosis	Address the underlying cause. Administer positive inotrope.
Reduction in Systemic Vascular Resistance		
	SIRS/sepsis Electrolyte abnormalities Severe hypoxia Severe acidosis or alkalosis Drug or toxins	Address the underlying cause. Provide fluid resuscitation. Administer vasopressors.

Drugs

Table 36.4 Inotropic and vasopressor drugs, adrenergic receptor binding, and standard dose range for dogs and cats.

Drug	α_1	α_2	β_1	β_2	Dose	Comments
Epinephrine	5+	3+	4+	2+	0.01–1 $\mu\text{g/kg/min}$ 0.02–0.2 mg/kg bolus	Primarily β effects at lower doses; increasing α effects at higher doses For cardiac arrest
Norepinephrine	5+	5+	3+	0/1+	0.01–0.1 $\mu\text{g/kg/min}$	β_2 effect not evident clinically
Dobutamine	0/1+	0	4+	2+	5–15 $\mu\text{g/kg/min}$	Positive inotropic effects, β_2 effect decreases afterload
Phenylephrine	5+	2+	0	0	0.2–2 $\mu\text{g/kg/min}$	
Dopamine	1–5+	2+	3+	2+	1–3 $\mu\text{g/kg/min}$	Actions are dependent on dose: Acts primarily on dopamine-1 receptors in renal and splanchnic vasculature to produce vasodilation
					5–10 $\mu\text{g/kg/min}$	Primarily β effects
					> 10 $\mu\text{g/kg/min}$	Primarily α effects
Ephedrine	2+	1+	3+	1+	0.05–0.5 mg/kg	Primary action by an indirect effect; direct and indirect effects (norepinephrine release)

Medication therapy

: Drugs are selected based on the patient's condition and underlying hemodynamic disturbance

- ① HR (chronotropism)
- ② Contractility (inotropism)
- ③ Myocardial conduction velocity (dromotropism)
- ④ Rhythm
- ⑤ Peripheral vasodilation or vasoconstriction → Influences preload and afterload

Anesthesia for cardiovascular disease patients

- Conditions associated with volume overload

1. Mitral valve insufficiency/regurgitation

“MR begets further MR”

Mitral valve regurgitation (MR)

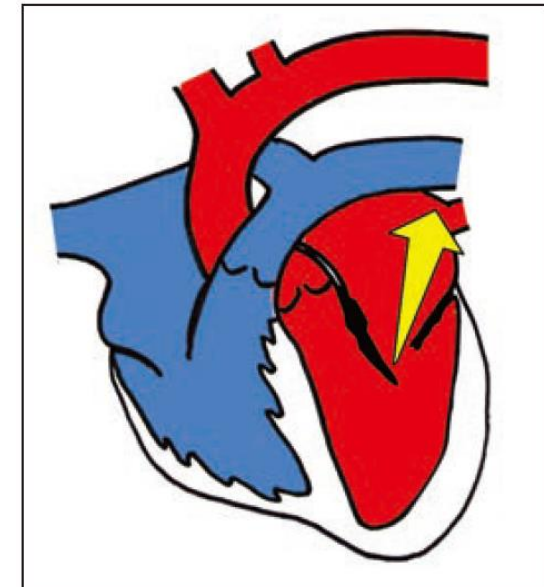
- Primary: incompetence of **the valve** leaflets (MMVD; 30% in small breed dog over 10 y) (e.g., myxomatous mitral valve degeneration, bacterial endocarditis, congenital malformation)
- Secondarily: dilation of **the annular ring** in diseases (more common in large breed dog) (e.g., DCM).

Mechanism

- High-pressure/low-compliance forward outflow (the aorta and arterial tree)
- Low-pressure/high-compliance backward outflow (incompetent mitral valve into the left atrium)

Chronic medical therapy

- Reduction in preload (diuretics such as furosemide and spironolactone)
- Reduction in arterial afterload (vasodilators such as ACEIs and pimobendan)
- Maintenance of adequate systolic contractility (positive inotropes such as pimobendan).



21.15 Schematic diagram illustrating major haemodynamic abnormalities in degenerative mitral valve disease: increased left atrial volume and pressure due to mitral insufficiency.

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Anesthesia for cardiovascular disease patients

- Conditions associated with volume overload

1. Mitral valve insufficiency/regurgitation

Anesthetic considerations

Goal: reducing afterload and optimizing forward flow

HR = Normal or slight above normal

- Smaller LV volume and minimize MR
- Bradycardia → increase systolic time for regurgitation

SVR = Decreased

Preload = Overzealous fluid administration should be avoided

- Fluid: 2-4 mL/kg/hr recommended

Contractility = Significant MR may benefit from inotropics.

Anticholinergics ← Induction → Dobutamine

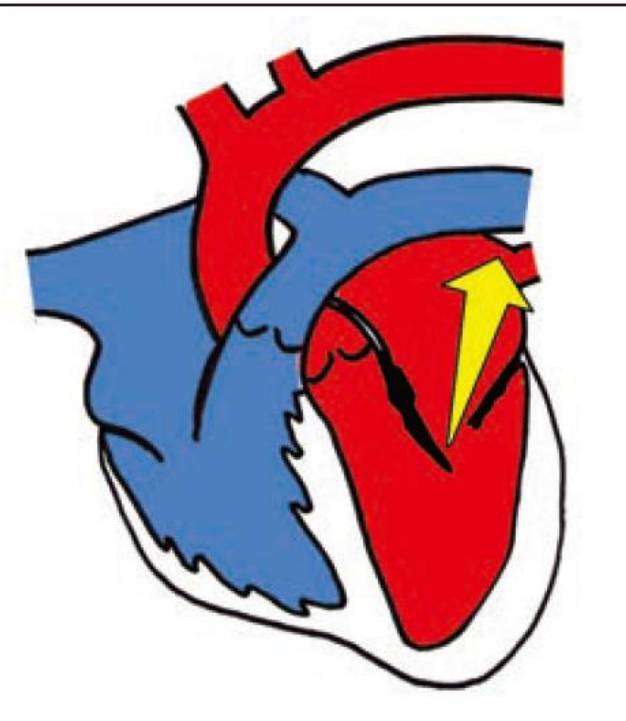
Box 36.1 Hemodynamic goals in patients with mitral valve regurgitation.

Preload: usually increased; some reduction may help decrease regurgitant flow

Afterload: decreases help minimize regurgitant flow

Contractility: unrecognized myocardial depression is possible

Rate: modest increases help maintain smaller left ventricular volume



21.15 Schematic diagram illustrating major haemodynamic abnormalities in degenerative mitral valve disease: increased left atrial volume and pressure due to mitral insufficiency.

Anesthesia for cardiovascular disease patients

- Conditions associated with volume overload

1. Mitral valve insufficiency/regurgitation

*It is **always prudent to have inotropic and venoconstrictor agents** immediately available for the management of intraoperative hypotension since **fluid administration is often contraindicated**.*

Anesthetic considerations

Premedication

- Opioid: dose-dependent HR ↓ via vagal nerve
- α_2 -adrenergic receptor agonists: HR ↓, SVR ↑
- Benzodiazepine: HR -, SVR -, Contractility -
- Phenothiazine: SVR ↓, anti-arrhythmogenic

Induction

- **No one best technique exists**
- High dose opioid + Benzodiazepine ± Induction agents
- Etomidate > Alfaxalone > Propofol
 - Etomidate: Contractility -, SVR - Am J Vet Res 1992; 53: 2178–2182
 - Alfaxalone: Compensatory HR ↑
 - Propofol: Compensatory HR - Front. 2024; 11: 1442670

Maintenance

- Isoflurane: dose-dependent myocardial depression
- Etomidate: hemolysis, adrenocortical suppression

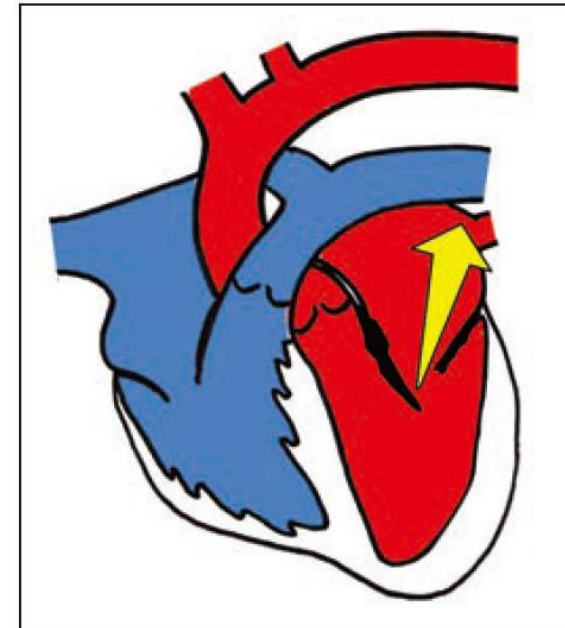
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21.15 Schematic diagram

illustrating major haemodynamic abnormalities in degenerative mitral valve disease: increased left atrial volume and pressure due to mitral insufficiency.

Anesthesia for cardiovascular disease patients

- Conditions associated with volume overload

2. Dilated cardiomyopathy

Dilated cardiomyopathy (DCM)

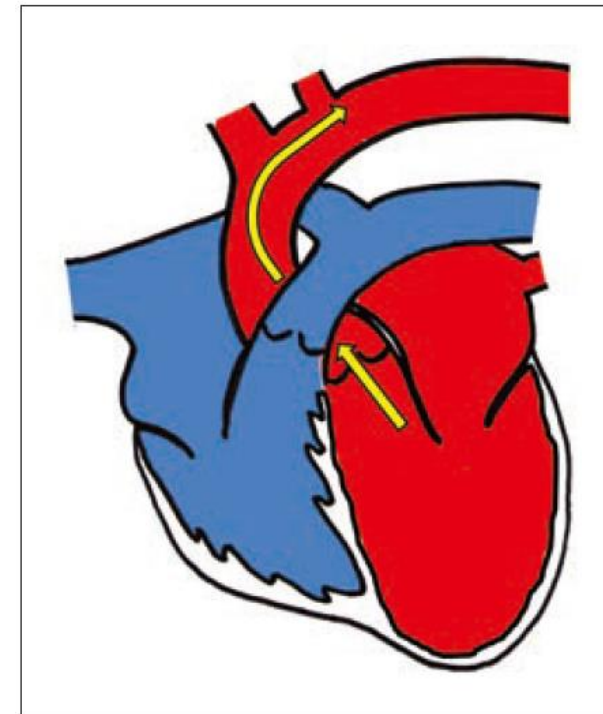
: defined as idiopathic **systolic dysfunction** and accompanied by **eccentric dilation and volume overload** (Doberman Pinscher, Irish Wolfhound, Great Dane, uncommon in cats, taurine deficiency in Cocker Spaniel)

Predispose

- CHF
 - Most, Left-sided (pulmonary edema)
 - Occasionally, right-sided (ascites, pleural effusion)
- **Ventricular arrhythmias** (VPC, AF)

Chronic medical therapy

- Reduction in preload (diuretics such as furosemide and spironolactone)
- Reduction in arterial afterload (vasodilators such as ACEIs and pimobendan)
- Maintenance of adequate systolic contractility (positive inotropes such as pimobendane and digoxin)
- Reduce AV conduction (lidocaine, sotalol, amiodarone, β -blockers)



21.17 Schematic diagram illustrating major haemodynamic abnormalities in dilated cardiomyopathy: poor systolic function, reduced cardiac output and eccentric left ventricular hypertrophy.

Anesthesia for cardiovascular disease patients

- Conditions associated with volume overload

2. Dilated cardiomyopathy

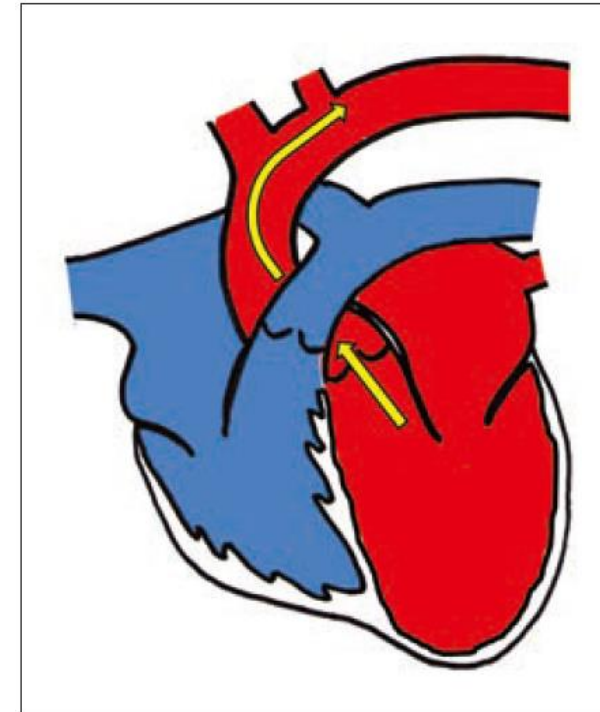
Anesthetic considerations

Goals

- **Like** those of animals with **MR**
- **Increasing systolic function** (may be severe)
- Caution for CHF
- Management of cardiac arrhythmias.

*Secondary means of monitoring CO

- Blood pressure monitoring (direct or indirect)
- Monitoring of end-tidal CO₂



21.17 Schematic diagram illustrating major haemodynamic abnormalities in dilated cardiomyopathy: poor systolic function, reduced cardiac output and eccentric left ventricular hypertrophy.

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Anesthesia for cardiovascular disease patients

- **Conditions associated with volume overload**

2. Dilated cardiomyopathy

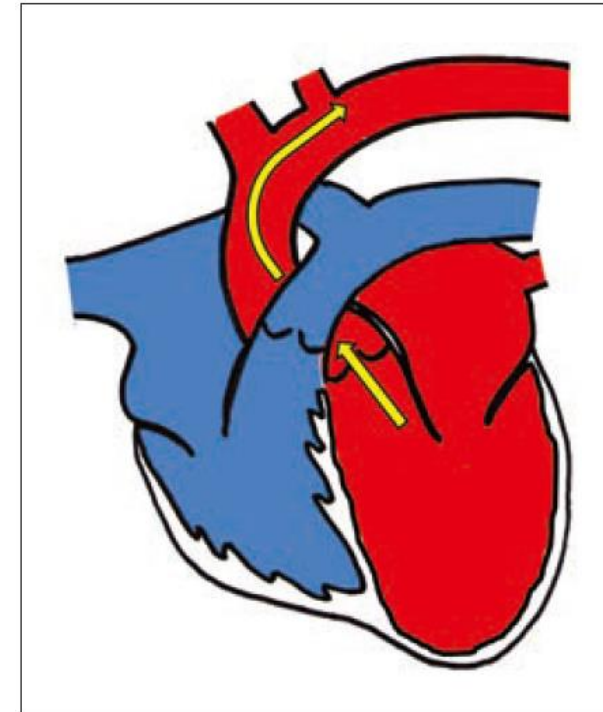
Anesthetic considerations

Contractility

- Dogs with **cardiomyopathy, down-regulation of β -adrenergic receptors in the heart**, although receptor affinity does not change
 - More resistant to treatment with a positive inotrope

Cardiac arrhythmias

- Lidocaine, for cases, the abnormal beats → BP and CO ↓
- Esmolol, a short-acting β -blocker → VPC by SNS ↑
- AF → resynchronization (cardioversion 0.5-2 J/kg)
- With AF → Anticholinergics is not useful
- Without AF → Anticholinergics is useful



21.17 Schematic diagram illustrating major haemodynamic abnormalities in dilated cardiomyopathy: poor systolic function, reduced cardiac output and eccentric left ventricular hypertrophy.

Anesthesia for cardiovascular disease patients

- Conditions associated with volume overload

3. Left-to-right shunting congenital defects (ventricular septal defect and patent ductus arteriosus)

Ventricular septal defect (VSD) and patent ductus arteriosus (PDA)

: produces **volume overload of the pulmonary circulation** and left heart.

→ Pulmonary hypertension (PHT) may be present

Goals

- Reduce shunt volume → Diastolic pressure ↑ (cardiovascular)
- Prevent reverse flow → Hypoxemia and polycythemia (respiratory)

Anesthetic consideration

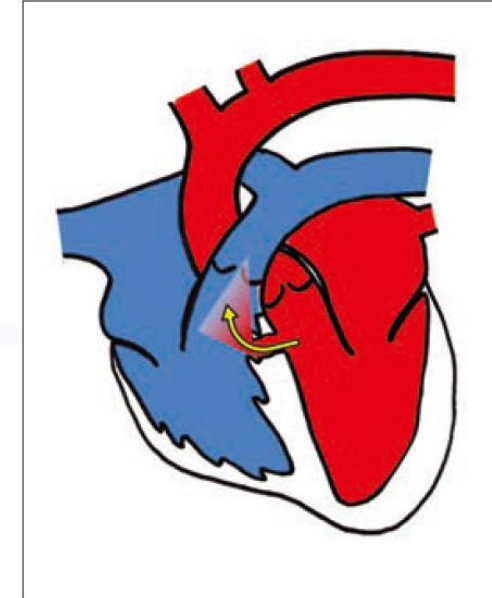
- **SVR ↓, PVR ↑ → Reduce shunt volume**
- **SVR ↓, PVR ↑ ↑ ↑ (with PHT) → Reverse flow**
- Maintain HR, contractility

Neonatal physiology (in puppies)

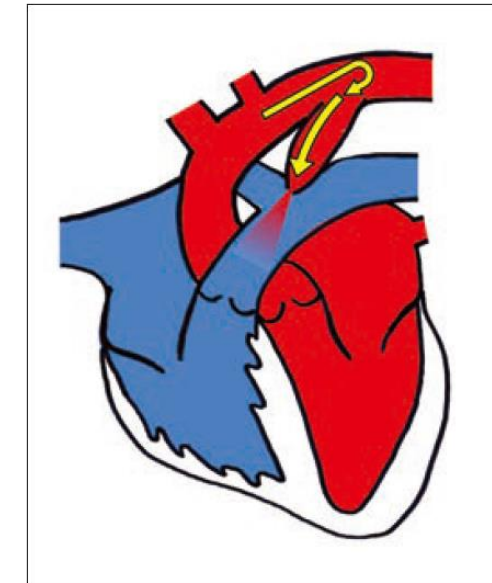
- Good responsiveness (< 1 week)
 - ① **Chronotropic**
- Bad responsiveness (< 8 week)
 - ① **Adrenergic vasoconstrictor**
 - ② **Inotropic response**

*PVR ↑

- Hypercapnia
- Acidosis
- Mechanical ventilation
- PEEP



21.21 Schematic diagram illustrating major haemodynamic abnormalities in ventricular septal defect: left-to-right flow due to low pulmonary vascular resistance.



21.20 Schematic diagram illustrating major haemodynamic abnormalities in patent ductus arteriosus: systemic-to-pulmonary flow due to low pulmonary vascular pressure relative to systemic pressure.

Anesthesia for cardiovascular disease patients

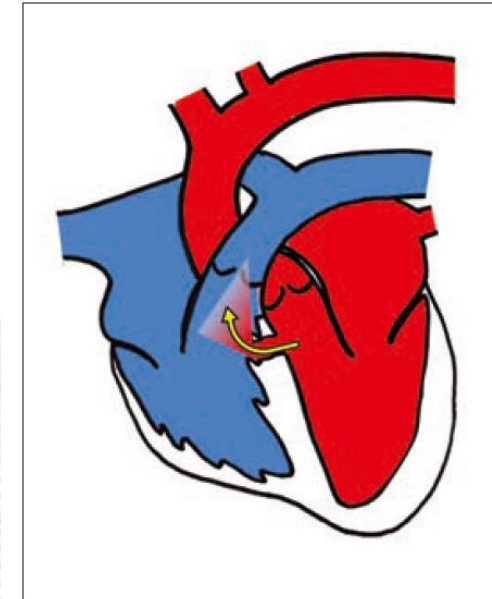
- Conditions associated with volume overload

3. Left-to-right shunting congenital defects (ventricular septal defect and patent ductus arteriosus)

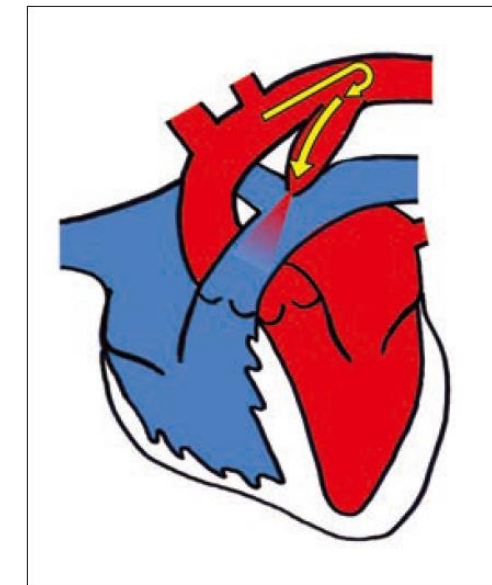
PDA occlusion, appearing dicrotic notch on arterial line



Figure 36.3 Output from a patient monitor during occlusion of a patent ductus arteriosus (PDA) showing the electrocardiogram and arterial pressure waveform. Notice the rapid increase in diastolic arterial blood pressure and the appearance of a dicrotic (reflected) wave during diastole immediately following occlusion (blue arrow). These changes are characteristic of decreased flow through the PDA and can be used to help identify the vessel during surgery.



21.21 Schematic diagram illustrating major haemodynamic abnormalities in ventricular septal defect: left-to-right flow due to low pulmonary vascular resistance.



21.20 Schematic diagram illustrating major haemodynamic abnormalities in patent ductus arteriosus: systemic-to-pulmonary flow due to low pulmonary vascular pressure relative to systemic pressure.

Anesthesia for cardiovascular disease patients

- Diastolic dysfunction-associated diseases

1. Hypertrophic cardiomyopathy and restrictive cardiomyopathy

Hypertrophic cardiomyopathy (HCM) and restrictive cardiomyopathy (RCM)

: Diastolic dysfunction → Inability of the ventricle to properly relax during diastole

Goals

- Optimizing diastolic filling
- Maintaining relatively low heart rates
- Avoiding increase cardiac contractility
- Maintain or increase SVR → LVOT ↓ and SAM ↓

Anesthetic considerations

- Ketamine is contraindicated.
- Vasoconstrictor could be useful.
 - α_1 -adrenergic receptor agonist with little or no β -activity

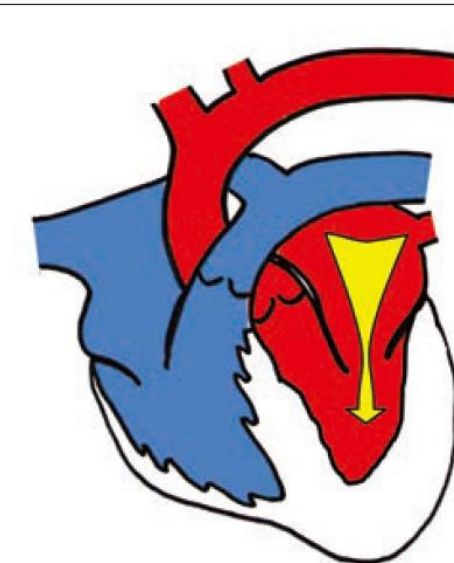
Box 36.3 Hemodynamic goals in patients with hypertrophic cardiomyopathy.

Preload: optimize intravascular volume to maintain filling of non-compliant ventricle

Afterload: maintain or augment, manage hypotension with vasoconstrictor titrated to effect

Contractility: reduction may be helpful

Rate: avoid tachycardia, modest reductions in rate preferred



21.16 Schematic diagram illustrating major haemodynamic abnormalities in hypertrophic cardiomyopathy: increased left atrial pressure due to reduced left ventricular compliance and volume.

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Anesthesia for cardiovascular disease patients

- Diastolic dysfunction-associated diseases

2. Pericardial effusion

Pericardial effusion

: the abnormal accumulation of large volumes of fluid within the pericardial space

Cardiac tamponade → Diastolic dysfunction

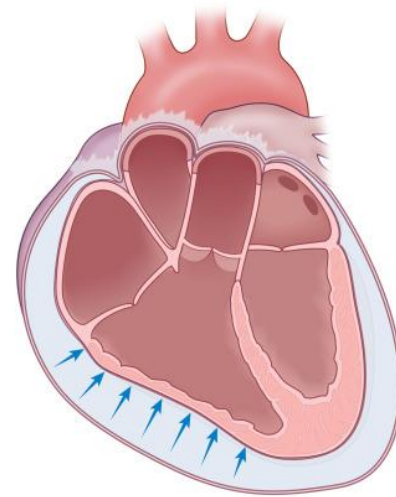
: **Volume of pericardial effusion is sufficient to decrease cardiac filling**, leading to poor CO and shock

Common causes

- ① Cardiac neoplasia
- ② Idiopathic pericarditis in dogs
- ③ CHF
- ④ Feline infectious peritonitis in cats

Common clinical findings

- ① Muffled heart sound, dyspnea, weakness, collapse etc.
- ② Low amplitude QRS complex
- ③ Globoid cardiac silhouette



<https://els-jbs-prod-cdn.jbs.elsevierhealth.com/cms/attachment/b85d68b2-daa3-4238-a227-cde55139510c/gr2.jpg>

Anesthesia for cardiovascular disease patients

- Diastolic dysfunction-associated diseases

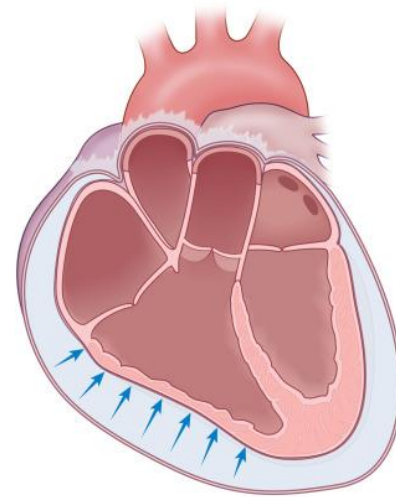
2. Pericardial effusion

Goals

- Preserve compensatory mechanism and **maintain forward flow**
- **Optimize preload** and maintain cardiac-filling pressures

Anesthetic considerations

- **Pericardiocentesis** indicated prior to induction of anesthesia
- **Small bolus** (2-5 mL/kg IV) of crystalloid or colloids at induction
→ Avoiding worsening PE than longer-term fluid therapy
- **Ketamine** helpful in preserving HR and BP (sympathomimetics)
- Mechanical ventilation or PEEP → Preload ↓ → CO ↓



Anesthesia for cardiovascular disease patients

- Diseases associated with arrhythmias

1. Arrhythmogenic diseases resulting in tachycardia

Common tachycardias

- ① VT (Boxer ARVC, feline cardiomyopathy, DCM, SAS)
- ② AF (MMVD and DCM)
- ③ SVT (MMVD, DCM and accessory pathway-mediated SVT) → Carotid sinus massage

Journal of Electrocardiology 1996; 29 (4): 327-332

Cardiovascular changes

- ① **Myocardial oxygen ↑**
- ② **Diastolic filling time ↓**

Differentiation

- **Origin of the tachycardia** (i.e., sinus versus ectopic)
- Species and breed (primarily related to body size)
- Underlying systolic and diastolic function of the heart
- Presence of eccentric or concentric hypertrophy

Indication of intervention

- **Ectopic HR > 250 beats/min in cats and small-breed dogs**
- **Ectopic HR > 200 beats/min in large-breed dogs**

Anesthesia for cardiovascular disease patients

- Diseases associated with arrhythmias

1. Arrhythmogenic diseases resulting in tachycardia

Medication

- ① **Lidocaine**: A sodium channel blocker (Class 1B)
 - inhibits sodium influx into cardiac cells
 - shortening the action potential and helping to control ventricular arrhythmias
- ② **Procainamide**: Another sodium channel blocker (Class 1A)
 - slowing down electrical conduction and prolonging the action potential duration
 - treating both ventricular and supraventricular arrhythmias
- ③ **Esmolol**: A short-acting β -blocker (Class II)
 - decreases heart rate and conduction
 - AF and SVT
- ④ **Diltiazem**: A calcium channel blocker (Class IV)
 - slows down the heart rate
 - AF and SVT
- ⑤ **Sotalol**: A beta-blocker (Class III)
 - blocks potassium channels, prolonging repolarization and the action potential
 - chronic ventricular arrhythmias and AF
- ⑥ **Mexiletine**: A sodium channel blocker (Class 1B) similar to lidocaine, but for chronic
- ⑦ **Digoxin**: Increases vagal tone (parasympathetic activity)
 - slowing conduction through the atrioventricular node

Anesthesia for cardiovascular disease patients

- Diseases associated with arrhythmias

2. Arrhythmogenic diseases resulting in bradycardia

Common bradycardias

- ① 2nd and 3rd degree AV nodal block (large breed dog)
 - ② SSS (Miniature Schnauzers)
 - ③ Atrial standstill
- *** Uncommon in cat

Differentiation

- 1. Primary (the cardiac conduction system)
 - ① Idiopathic degeneration of the sinus node, AV node, and other conduction tissue
 - ② Acute inflammatory injury to the heart (i.e., myocarditis)
- 2. Secondary
 - ① Electrolyte abnormalities (**severe hyperkalemia**)
 - Renal disease, uncontrolled hyperadrenocorticism, soft tissue injury, and other conditions

Normal HR?

= HR during sleep may be a better indicator of the patient's acceptable heart rate during anesthesia

Anesthesia for cardiovascular disease patients

- Diseases associated with arrhythmias

2. Arrhythmogenic diseases resulting in bradycardia

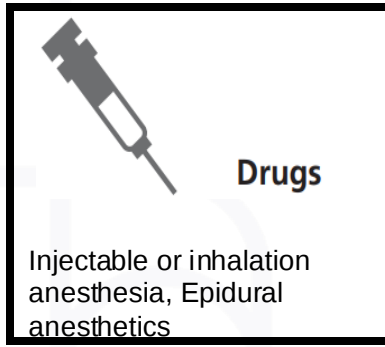
Therapy

1. Correction of underlying electrolyte abnormalities
2. Support of heart rate
 - ① Parasympatholytics (e.g., atropine, glycopyrrolate, and probanthine bromide)
 - ② Sympathomimetics (e.g., isoproterenol, terbutaline, and theophylline)
3. Artificial pacing → poor responsive to medical therapy

Anesthetic considerations

- Correct electrolyte abnormalities prior to anesthesia
- Acepromazine → Anti-arrhythmogenic effect
- Bradycardia and ectopic rhythm by hyperkalemia → Contraindicate using lidocaine
- Tachycardia → Ketamine usually avoided
- SVT → Vagomimetic drugs (fentanyl and remifentanyl) be useful → Anticholinergics usually avoided
- SSS
 - High and repeated dose of anticholinergics
 - Contraindicate α_2 -adrenergic receptor agonist
 - Prepare artificial pacemaker (poor response at medication)
 - Prepare sympathomimetics if you don't have artificial pacemaker

Balance between anesthesia and cardiovascular disease



Injectable

- Alfaxalone
- Propofol

Inhalational

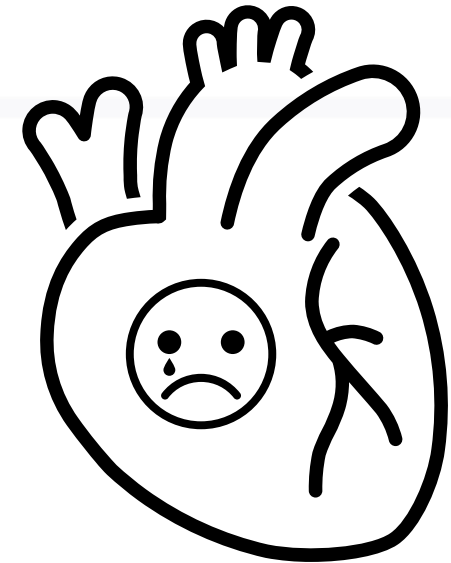
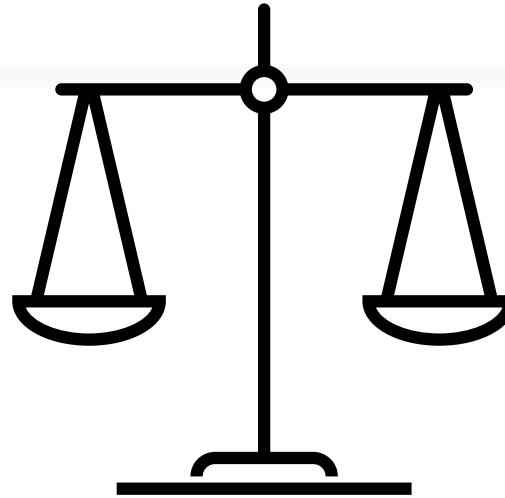
- Isoflurane

Sympathetic nervous systems ↓

Heart rate ↓

Vessel resistance ↓

Blood pressure ↓





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