ASSOCIATION OF VETERINARY ANAESTHETISTS

SPRING MEETING RIMINI, ITALY

23 April 2005

PROCEEDINGS
Resident and refresher day

Saturday 23 04 05
Ecva Residents Training Day Program

9.00- 10.30	Physiology of the heart Main diseases in SA, arrhythmias Radiology and echocardiography	Borgarelli M. (I)
11.00 –12.30	Physiology of the heart Main diseases in LA, arrhythmias (bovines and horses) Radiology and echocardiography	Marr C. (UK)
14.00-15.30	Anaesthesia for specific cardiovascular diseases in SA	Pascoe P. (USA)
16.00-17.30	Cardiovascular dysfunction during anaesthesia in large animals	Blissit K. (UK)

Keynotes resident day

PHYSIOLOGY OF THE HEART AND MAIN CARDIOLOGICAL DISEASES IN SMALL ANIMALS. USELFUNESS OF ECG RADIOLOGY AND ECHOCARDIOGRAPHY EXAMS

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Principles of heart physiology

The circulatory system serves to transport and distribute substances (i.e. essential elements and drugs) to the tissues and to remove by-products of metabolism. Essentially the heart is composed by two pumps (left and right ventricle) those work in series. The left ventricle (LV) propels blood into systemic circulation, the right ventricle (RV) propels blood into pulmonary circulation. Unidirectional flow through the heart is achieved by the appropriate arrangement of effective flap valves (mitral, tricuspidal, aortic, and pulmonary valves). One of the most important point is that LV can be considered as a pressure pump (it pumps blood in a high pressure system), whereas RV can be considered as a volume pump (it pumps blood in a low pressure system). According to this gross classification LV best tolerates pressure overload (i.e. subaortic stenosis or systemic hypertension) and RV best tolerates volume overload (i.e. tricuspid regurgitation). To best understand pathophysiology of heart diseases other important concepts to remind are represented by heart rate (HR), preload, afterload and contractility.
HR is determined by the rate of spontaneous sinoatrial node discharge. This in turns depends on existing autonomic efferent traffic. HR increases if sympathetic activity increases, HR decreases if parasympathetic activity increases. Under this point of view dogs could be considered parasympathetic whereas cats could be considered sympathetic animals. In normal heart increasing HR increases cardiac output, but in diseased hearts although increasing HR contribute to maintain cardiac output, this produces an increase in cardiac work, cardiac O_2 consumption and eventually contribute to worsen cardiac failure. **Preload** represents the quantity of blood returning to the heart. In normal heart according to Frank Starling mechanisms an increase in preload produce an increase in cardiac output. In diseased hearts increasing preload can contribute to maintain cardiac output, but eventually it leads to eccentric hypertrophy, cardiac remodelling and apoptosis and to a worsening of cardiac performance. **Afterload** may be described as the stress acting on ventricular wall myocetes after onset of shortening. It depends essentially, but not only, by peripheral resistances (i.e. degree of peripheral vasoconstriction). Any increasing in afterload is associated with a decrease of cardiac output. A chronic afterload increase leads, according to La Place law, to a concentric hypertrophy.

Main cardiac diseases in small animals

Cardiac diseases can be grossly divided in congenital and acquired. From the pathophysiological point of view they can also been divided in volume overload and pressure overload diseases.

Congenital heart diseases with volume overload.

The most common congenital diseases associated with volume overload in **dogs** are patent ductus arteriosus (PDA), interventricular defects (VSD) and mitral (MD)and tricuspid dysplasia (TD). Severity of PDA and VSD depends on diameter of the ductus or of the defect, and so on the volume of blood that shunts through them. PDA is a common congenital disease in female. The most common affected breed are German Sheperds, Poodles, Collies and Newfounland. VSD is more common in Bulldogs. MD is common in Bull terriers, Newfoundland and Great Danes. TD is more common in Labrador, Golden retrivier, Weimarainer. Volume overload congenital diseases may remain asymptomatic for a long time, and some animals, if affected by small defects, may also never require any treatment. PDA can be corrected both by surgical procedures or through coil occlusion. Both techniques present advantages or disadvantage. The most common volume overload congenital heart disease in **cats** is VSD.

Congenital heart diseases with pressure overload

The most common heart diseases with pressure overload in **dogs** are subaortic (SAS) and pulmonic stenosis (PS), whereas these are sporadic diseases in **cats.** SAS represent the most common congenital disease in Boxer, Golden Retriever, Newfoundland, Rottweiler. PS is common in Airdale Terrier, Beagle, Bulldog, Schnautzer breed, Boxer. Severity of SAS and PS essentially depends on how narrow is the stenosis. It should be remind that dogs affected may be asymptomatic also with very severe disease. These patient may die suddenly. Valvular PS can be corrected using balloon valvuloplasty with very good results. Patients with moderate or severe diseases are susceptible to development of hypossia or arrhytmias during anesthesiological procedure.

Acquired heart diseases with volume overload in dogs

In **dogs** these include more than 70 % of heart diseases. The most common is chronic mitral valve disease or endocardiosis (CMVD) who affect around 30% of dogs of more 8 years of age. The second most common acquired heart disease in dogs is represented by dilated cardiomyopathy (DCM), that affects mostly large breed dogs and it is overrepresented in Dobermann Pincher, Great Danes and Newfoundland.
CMVD is a chronic disease of mitral valve usually characterize by the presence of a typical murmur localize on left side thorax. However affected dogs may remain asymptomatic and may not require any treatment for a long period (i.e. more than 2 years). At the time they decompensate therapy should be directed to remove lung fluid

accumulation and counteract neuro-hormonal mechanism of compensation (i.e. increase renin angiotensin aldosterone (ACE) system activity). Systolic dysfunction (i.e. decrease contractility) has been believed to occur late during the phase of the disease, although this concept is actually under review from some authors.
DCM is a primary disease of the heart muscle, characterized by a primary systolic dysfunction. The pre clinical phase can be last more than 2 years. During this period the only way to diagnose the disease is echocardiography. However diagnosis at the very beginning of the diseases can be difficult also with echocardiography and require appropriate trained cardiologists. Guideline for the diagnosis of DCM have been recently published. It should be underlined that dogs with asymptomatic disease present an high anesthesiological risk, as it has been demonstrated in Dobermann pincher. So echocardiographic screening in predisposed breed should be encourage whenever possible.

Acquire heart diseases in cats

The most common acquired heart disease in **cats** are represented by both hypertrophic and restrictive cardiomyopathies. Both diseases may present a long asymptomatic period. Asymptomatic affected patients are difficult to diagnose, as they have no or minimal clinical findings (i.e. presence of gallop rhythm). In these patients also echocardiographic examination may provide equivocal results. Patients with asymptomatic diseases may be develop hypossia or arrhythmias during anesthesia if not adequate O_2 support is granted.

ECG, THORAX RADIOGRAPHY AND ECHOCARDIOGRAPHIC USEFULENESS TO EVALUATE PATIENTS WITH CARDIAC DISEASES.

Electrocardiography (ECG)

Electrocardiography (ECG) may provide some usefulness information concerning heart rhythm and presence or absence of arrhythmias. It doesn't provide any information concerning compensate vs not compensated heart failure. Moreover it should be remind that may arrhythmias may be paroxysmal and consequently not detected through a regular ECG. In these cases, if patients present an anamnesis of lipothymic episode or syncope a 24 hours Holter monitoring exam could be a better choice.

Thorax radiographies

These exams provide a lot a very useful information. They can provide information concerning the degree of cardiac enlargement and which part of the heart is affected, presence and severity of pulmonary edema, and they presence of concomitant respiratory diseases (i.e. chronic bronchitis) with clinical signs similar to heart failure. According to our opinion thorax X-rays should be performed in all dogs with known cardiac disease who have to be submitted to an anesthesiological procedure whenever this would be possible

Echocardiography and echo Doppler

Echocardiographic exam represent the gold standard technique to provide the most accurate diagnosis of a cardiac disease. Moreover this exam may not invasively provide hemodynamic information such as evaluation of systolic function. More information can be obtained through an echo Doppler exam. This can provide not invasively information on severity of valvular stenosis and diastolic function.
It should be underlined that echocardiography and echo Doppler exams to be properly interpreted needs a deep knowledge in the mechanism of heart disease, otherwise it could be easy misinterpreted the results.

SUGGESTED READINGS

Bern RM, Levy MN "Cardiovascular physiology 6th ed.". Mosby, New York 1992

Fox P, Sisson D, Moise "Textbook of canine and feline cardiology 2nd ed." W.B. Saunders Philadelphia

Kittleson MD, Kienle RD "Small Animal Cardiovascular" Mosby, New York, 1998

LARGE ANIMAL CARDIOLOGY

Celia M Marr
Rossdales equine hospital, newmarket, uk

Common reasons for presentation for cardiovascular investigation in farm animals

- Failure to gain weight
- Weight loss
- Fever
- Respiratory signs
- Peripheral oedema

Common reasons for presentation for cardiovascular investigation in horses

- Cardiac murmur or arrhythmia detected incidentally during a routine examination (including pre-operative assessment)
- Poor performance or exercise intolerance
- Respiratory signs
- Peripheral oedema
- Collapse, Distress

Common equine clients' questions

- Is this disease affecting my horse's performance now?
- Will this disease affecting my horse's performance in future?
- Is it safe for me to ride my horse?
- Will this disease shorten my horse's life span?
- Can this problem be cured?
- Should I buy this horse?

SPECIFIC EXAMINATION OF THE CARDIOVASCULAR SYSTEM

MUCOUS MEMBRANES

- HORSES - normally slightly yellowish pink
- CATTLE - normally slightly pale

ARTERIAL PULSES

- HORSES - facial, transverse facial, digital
- CATTLE - brachial

VEINS

- Large animals normally have a visible jugular pulse in the distal third of the neck
- HORSES - jugular, lateral thoracic
- CATTLE - jugular, superficial epigastric

AUSCULTATION OF THE HEART

- Under triceps muscle mass, between level of point of shoulder and point of elbow
- COMMONEST ERROR LISTENING TOO FAR BACK AND TOO LOW DOWN

EQUINE HEART SOUNDS

First:

- closure of the atrioventricular valves
- opening of the semilunar valves
- all horses

Second:

- closure of the semilunar valves
- opening of the atrioventricular valves
- all horses

Third:

- End of rapid ventricular filling
- 30% of horses

Fourth:

- atrial systole
- 60% of horses

RUMINANT HEART SOUNDS

First:

- closure of the atrioventricular valves
- opening of the semilunar valves
- onset of systole

Second:

- closure of the semilunar valves
- opening of the atrioventricular valves

at onset of diastole

CLINICAL SIGNS OF CARDIAC DYSFUNCTION

LOW CARDIAC OUTPUT

- Tachycardia
- Generalised Weakness
- Pale mucous membranes
- Weak arterial pulses
- Cold extremities

LEFT HEART FAILURE

Acute left heart disease (rare)

- Alveolar oedema
- Frothy nasal discharge

Chronic left heart disease

- Interstitial oedema
- Raised respiratory rate
- Moist wheezes

RIGHT-SIDED FAILURE

- Ventral oedema:
- Brisket
- Muzzle and submandibular area
- Prepuce
- Hind-limbs
- Venous distension and pulsation
- Pleural effusion - areas of ventral dullness
- Ascites - difficult to appreciate on clinical exam

Causes of murmurs in horses

- Physiological flow murmurs
    - most common and occur in over 65% of fit Thoroughbreds and Standardbreds
    - forward flow through valves that are open during the phase of the cardiac cycle in which the murmur occurs
- Physiological regurgitation
- Acquired Valvular disease
    - causes insufficiency but very rarely stenosis
    - backward flow through valves that are should be shut during the phase of the cardiac cycle in which the murmur occurs therefore when the murmur can be localised to a specific valve area and a specific phase of the cardiac cycle, physiological murmurs are readily distinguished from murmurs of valvular insufficiency
- Congenital Cardiac Disease
    - particularly Ventricular Septal Defect

Causes of murmurs in cattle

- Acquired Valvular disease
    - Most common form is bacterial endocarditis of the tricuspid and/or pulmonic valves
- Congenital Cardiac Disease
    - Ventricular Septal Defect and Tetralogy of Fallot are relatively common in cattle

BACTERIAL ENDOCARDITIS

AETIOPATHOGENESIS:

CATTLE

- Bacteraemia
- chronic septic focus at a distant site - e.g. liver abscess, traumatic reticulitis, metritis, mastitis, omphalophelbitis, musculoskeletal infection
    - *Enterococci* spp. Ipha-haemolytic *Streptococcus, Actinomyces (Corynebacterium) pyogenes*

HORSES

- Bacteraemia
    - *Pasteurella/Actinobacillus* spp., *Streptococcus* spp., *Rhodococcus equi*
- Site of chronic sepsis may not be identified
- May occur in association with septic jugular thrombophelbitis

PIGS

- Erysipelothrix rhusiopathiae (erysipelas), Staph aureus, Actinobacillus equuli and suis
- Left valves more commonly affected
- Diagnosed on post-mortem exam and usually involves lesions in other systems

PATHOLOGY

- Vegetative lesions on affected valves
- Emboli shed to distant sites such as lungs (from right heart), kidneys and joints (from left heart)

VALVES AFFECTED

CATTLE

- Tricuspid, pulmonic, right ventricular wall

HORSES & PIGS

- Mitral and aortic - most common
- Tricuspid and pulmonic - occurs following septic thrombophelbitis

CLINICAL PRESENTATION

CATTLE

- Early signs: recurrent fever, anorexia, weight loss, poor production, lameness or stiffness
- These non-specific signs progress to
- Cardiac murmurs and tachycardia
- Right-sided failure

HORSES

- Early signs: recurrent fever, anorexia, and weight loss
- Shifting lameness or stiffness
- These non-specific signs progress to
- Cardiac murmurs and tachycardia
- Left or right-sided failure depending on the valve affected

DIAGNOSIS

- Echocardiography
- Blood culture
- False negative results are common
- At least three samples from aseptically prepared sites
- Avoid sampling from catheters
- Sampling during febrile episodes may improve likelihood of positive culture

TREATMENT

- Broad-spectrum antibiotics, if possible based on culture and sensitivity results

PROGNOSIS

- Guarded - even if it is possible to clear the infection, there may be permanent structural damage to valve

DISRUPTION OF THE VALVE APPARATUS

- Ruptured chordae tendineae
- Can arise spontaneously or secondary to inflammatory or degenerative changes in the chordae
- Severe regurgitation with a rapid change in haemodynamic status
- Sudden death or signs of acute cardiac failure

EQUINE MITRAL INSUFFICIENCY

- Mitral Insufficiency is the most common cause of clinical signs of cardiac disease
- **Can be caused by acquired degenerative lesions (middle-aged and older horses)**
- May be related to hypertrophy in athletes
- Occasionally due to bacterial endocarditis or rupture of a chordae tendineae (acute, severe onset)

MURMUR CHARACTERISTICS

- Grade 1 - 6/6, Early, mid, holo or pan-systolic, band-shaped
- Left fifth intercostal space, radiating caudodorsally

CLINICAL PRESENTATION

- All age groups
- Incidental finding
- Poor Performance
- Atrial fibrillation
- Acute onset left-sided failure
- Right-sided failure
- Collapse, fainting or sudden death

INDICATIONS FOR FURTHER INVESTIGATION

- Grade $\geq$ 3/6
- Loud third heart sound
- Resting tachycardia
- Fever
- Poor performance
- Arrhythmias

TRICUSPID INSUFFICIENCY

- Common in racing Thoroughbreds and Standardbreds
- May be related to hypertrophy
- Generally well tolerated
- Less common in other breeds
- Bacterial endocarditis - secondary to septic jugular thrombosis

MURMUR CHARACTERISTICS

- Grade 1 - 6/6, Holo or pansystolic, Crescendo-decrescendo or Band-shaped
- Right fourth intercostal space, Radiating craniodorsally

INDICATIONS FOR FURTHER INVESTIGATION

- Grade $\geq$ 4/6 in racehorses
- Grade $\geq$ 3/6 in other types
- Jugular pulsation and distension
- Ventral and peripheral oedema
- Poor performance
- Atrial fibrillation

AORTIC INSUFFICIENCY

DEGENERATIVE

- Commonest form
- Middle-aged or older horses - typically not severe until late teens
- Frequently an incidental finding

INFLAMMATORY VALVULITIS

- Any age group
- Incidental or poor performance

BACTERIAL ENDOCARDITIS

- Fever, vague malaise, shifting lameness
- Congestive heart failure

MURMUR CHARACTERISTICS

- Grade 1 - 6/6, Early or holodiastolic, Decrescendo, Sometimes creaking
- Left heart base, Radiating caudoventrally

CONSEQUENCES

- With progressive dilation, the mitral valve annulus dilates and becomes incompetent

- There is a higher prevalence of ventricular arrhythmias in horses with aortic insufficiency compared with other forms of valvular insufficiency

INDICATIONS FOR FURTHER INVESTIGATION

- Hyperkinetic pulses
- Murmur grade $\geq$ 3/6
- Young horse
- Old horse still in work
- Concurrent murmurs or arrhythmias
- Poor performance
- Fever
- Congestive heart failure

PULMONIC INSUFFICIENCY

- Both valvular pathology and clinical disease associated with pulmonic insufficiency are uncommon

MYOCARDIAL DISEASE & ARRHYTHMIAS

PATHOGENESIS OF MYOCARDIAL DISEASE AND DYSFUNCTION IN LARGE ANIMALS

PRIMARY

- Viral
- Bacterial
- Parasitic
- Toxic
- Nutritional
- Cardiomyopathy
- Neoplasia
- Immune-mediated
- Idiopathic

SECONDARY

- Hypoxia
- Endotoxaemia
- Electrolyte disturbances
- Acid-base disturbances
- Catecholamine-induced
- Vagally-induced

VIRAL

- Equine Influenza, Equine Herpes Virus, Equine Viral Arteritis
- Foot and mouth disease, African Horse sickness, Equine Infectious Anaemia

BACTERIAL

- Staphylococcus aureus, Clostridium chauvoei, Mycobacterium spp.
- Strep. equi equi., Actinobacillus spp, Rhodococcus equi

PARASITIC

- Strongyles, onchocerca, toxoplasma, cysticerca, sarcocysta, Borrelia burgdorferi (Lymes).
- **Myocardial Toxins**

DRUGS

- e.g. halothane, erythromycin, digoxin, dobutamine
- anti-arrhythmic agents

IONOPHORE ANTIBIOTICS

- Used as coccidiostats in poultry and growth promoters in cattle
- Horses and pigs are more sensitive to cardiac side-effects
- In recent years, most reported episodes have been associated with accidental contamination of feed at feedmills

NUTRITIONAL MYODEGENERATION (WHITE MUSCLE DISEASE)

- Ruminants and, less commonly, equines grazing selenium deficient pastures

IDIOPATHIC CARDIOMYOPATHY

CATTLE

- Inherited
- Linked to the red Holstein gene in Holstein-Friesians
- Associated with curly hair coat in polled Herefords

HORSES

- Occurs sporadically, causes unknown

CARDIAC NEOPLASIA

CATTLE

- Right atrial lesions extending into the remainder of the heart and heart base area
- Adult form Enzootic Bovine Leukaemia

HORSES

- Lymphosarcoma, haemangiosacroma and other neoplastic conditions occur sporadically

INFLAMMATORY LESIONS AND FIBROSIS

- Focal or generalised
- Aetiology unknown
- Immune-mediated?
- Poorly investigated and information extrapolated from human cardiology

SECONDARY MYOCARDIAL DISEASE AND DYSFUNCTION

- Cattle and Horses with severe gastrointestinal disease and horses with upper airway obstruction during exercise may develop arrhythmias
- Fairly common, and often unrecognised, in equine intensive care patients

PHYSIOLOGICAL BRADYARRHYTHMIAS

Control of heart rate at rest

- Equine resting heart rate is low: 25 - 42 bpm
- Horses have very little or no sympathetic tone at rest
- Parasympathetic tone is high
- The vagus innervates both the sinoatrial and the atrioventricular nodes
- The horse can reduce its heart rate by delaying conduction at the atrioventricular node

Arrhythmias at rest

- Sinus arrhythmia
- Sinus pause / block
- Atrioventricular block

ATRIOVENTRICULAR BLOCK

First degree

- Delayed conduction through the AV node
- Slow, slightly variable heart rate

Second degree

- Intermittent block of conduction through the AV node
- Slow heart rate with pauses at regular intervals
- Fourth heart sound audible
- Disappears when vagal tone reduced and/or sympathetic tone increased

Third degree

- Complete block of conduction
- Due to pathology at the AV node
- Very slow heart rate, syncope, weakness

Cardiac responses during and after strenuous exercise

During exercise

- Heart rate increases to 200 - 230 bpm
- Stroke volume increases about 20 - 25%
- Cardiac output increases by 5 - 10 fold

After exercise

- both second degree AV block and sinus arrhythmia are common

PATHOLOGICAL BRADYARRHYMIAS

ADVANCED SECOND DEGREE AND THIRD DEGREE ATRIOVENTRICULAR BLOCK

- occur very occasionally
- drugs (e.g. halothane overdose)
- hyperkalaemia (e.g. uroperitoneum, renal failure)
- inflammation or fibrosis at AV node

Treatment

- address underlying cause
- pacemakers have been used successfully in horses

SUPRAVENTRICULAR PREMATURE DEPOLARISATIONS

- SVP may or may not be conducted into ventricle because of horse's superior ability to control conduction through the AV node
- rarely require specific anti-arrhythmic therapy because ventricular rate is not increased
- identify, investigate and treat underlying cause (see list above)
- idiopathic - corticosteroids and rest are used in the hope that this may be effective in a sub-population with an inflammatory pathogenesis

SUPRAVENTRICULAR TACHYCARDIA

- Rarely occurs except in association with quinidine toxicity

VENTRICULAR PREMATURE DEPOLARISATIONS

- Identify, Investigate and treat underlying cause (see list above)
- Idiopathic - corticosteroids and rest are used in the hope that anti-inflammatories may remove underlying cause
- Ventricular arrhythmias are more likely than other arrhythmiasto progress into fatal arrhythmias
- Establish if the arrhythmia occurs
    - at rest only and not during exercise
    - at rest and during exercise
    - during exercise only

VENTRICULAR PREMATURE DEPOLARISATIONS AND TACHYCARDIA

- Identify, Investigate and treat underlying cause (see list above)
- Assess likelihood that the rhythm will destabilise to ventricular fibrillation

CONSIDER ANTI-ARRHYTHMIC THERAPY IF:

- there are clinical signs of low cardiac output
- ventricular rate is greater than 100
- polymorphic
- R-on-T phenomenon

ANTI-ARRHYTHMICS USED TO TREAT VENTRICULAR ARRHYTHMIAS IN HORSES

Procainamide

- drug of first choice in conscious horses

Quinidine gluconate

- USA only, not available in UK

Lignocaine
- seizures, drug of first choice under anaesthesia

Propanolol
- rarely effective in horses

Magnesium sulphate (physiological calcium channel blocker)
- readily available and inexpensive but has variable efficacy

ATRIAL FIBRILLATION

PATHOGENESIS

VARIABLE REFRACTORY PERIODS PREDISPOSE TO ONSET OF AF:
- High vagal tone
- normal horses
- cattle with GI disease
- Myocardial pathology
- Electrolyte disturbances

CRITICAL ATRIAL MASS SUSTAINS AF:
- Normal horses
- Atrial enlargement
- Mitral or tricuspid insufficiency
- Congenital heart disease

HAEMODYNAMIC IMPACT
- The atria contribute to around 15% of ventricular filling
- In horses, with no other cardiac disease, atrial fibrillation will only causes signs of exercise intolerance if the animal is engaged in vigorous exercise (Racehorses. Eventers, some hunters)
- In many types of horses it can be an incidental finding (Breeding stock, hacks, some show jumpers)

PRESENTING SIGNS
- Exercise intolerance/poor performance
- Reluctance to participate in exercise
- Irregularly-irregular cardiac rhythm
- intermittent long pauses interspersed with rapid runs
- no fourth heart sound
- Variable pulse quality
- Variable intensity of heart sounds
- Exercise-induced pulmonary haemorrhage

PAROXYSMAL ATRIAL FIBRILLATION
- Atrial fibrillation may spontaneously resolve

- exercise-induced
- horses and cattle with gastrointestinal disease

DIAGNOSTIC APPROACH

Goal

- identify and characterise any underlying cardiac disease
- clinical exam
- echocardiography
- clinical pathology

ATRIAL FIBRILLATION IS AN EFFECT AND NOT A CAUSE OF CONGESTIVE HEART FAILURE

Clinical findings that might indicate underlying heart disease

- tachycardia
- heart rate > 50 bpm
- cardiac murmurs
- particularly murmurs of AV regurgitation
- venous distension and pulsation
- peripheral oedema

TREATMENT OPTIONS

Well-tolerated – consider no treatment
Congestive Cardiac Failure – manage CHF, do not use quindine
Exercise limitation – quinidine sulphate or electrocardioversion

ELECTROCARDIOVERSION
Currently being evaluated at various centres

QUINIDINE

- prolongs the effective refractory period
- vagolytic
- alpha-adrenergic antagonist
- proarrhythmic
- gastrointestinal ulceration

PHARMACOKINETICS OF QUINIDINE SULPHATE

- peak concentration 131 minutes
- rapid plasma to tissue distribution
- elimination half-life 3.3 - 12.5 hours (mean 6.6)
- therapeutic plasma concentrations 2 - 5 g/ml
- toxic plasma concentration >5 g/ml

- Monitor for increased QRS duration before each dose (do not give if >25% pre-treatment value)

- Administer by nasogastric tube
- 22 mg/kg every 2 hours until:
- reach therapeutic plasma concentrations
- or have given four to six doses (in absence of ability to measure quinidine concentrations promptly)
- Then continue at 6 hour dosing intervals
- Continue treatment until:
- normal sinus rhythm is restored
- side-effects are observed

SIDE EFFECTS
Extra-cardiac
- diarrhoea
- colic
- flatulence
- respiratory stridor
- penile protrusion
- ataxia
- tend to be seen in horses that require prolonged treatment
- GI effects are the main cause of treatment failure

Cardiovascular
- hypotension
- rapid supraventricular tachycardia
- ventricular arrhythmias

- potentially fatal arrhythmias occur in around 4% of cases, independent of plasma concentrations, monitor the ECG continuously

HYPOTENSION
- ALPHA ADRENERGIC ANTAGONIST
- keep the horse calm
- do not allow any form of exercise during treatment
- iv fluids may be necessary

RAPID SUPRAVENTRICULAR TACHYCARDIA
- VAGOLYTIC EFFECT
- Emergency treatment required if ventricular rate > 100 /min
- digoxin to slow conduction through the AV node
- bicarbonate to increase protein binding to reduce effective plasma concentration
- intravenous fluids to support blood pressure

VENTRICULAR ARRHYTHMIAS
- PROARRHYTHMIC EFFECT
- Magnesium sulphate

- Propanolol
- Lignocaine
- NOT PROCAINAMIDE (similar mechanism of action as quinidine)

MANAGEMENT AFTER TREATMENT
- Normal 24 hour ECG
- return to training
- Supraventricular premature depolarisations
- Rest?
- Corticosteroids?
- Digoxin?

PROGNOSIS
Depends on
- degree of underlying cardiac disease & duration prior to treatment
- less than 3 months: recurrence rate 15%
- greater than three months: recurrence rate 60%
- **higher prevalence of side-effects associated with prolonged duration of treatment**

ARE HORSES WITH AF "SAFE" TO RIDE
- Probably but may be a slight increased risk of developing exercise-induced ventricular arrhythmias compared to other horses.
- Risk has not been quantified.
- Recommend exercising ECG

ATRIAL FIBRILLATION & ANAESTHESIA
- Can occur during GA – no specific interventions are necessary provided blood pressure is maintained
- When detected during a pre-operative assessment: must consider its significance in light of proposed surgical procedure.
- Emergency GA: procede with no specific intervention provided blood pressure is maintained
- Elective GA: no evidence that AF increases the risk of anaesthetic death. May need to evaluate the need for elective surgery in light of impact of AF on horse's future use.

PERICARDIAL DISEASE IN LARGE ANIMALS
Traumatic / Septic
- most common from in cattle

Idiopathic
- most common from in horses

Bacterial
- most common form in pigs

Neoplastic

- uncommon in large animals

Viral

- uncommon in large animals

TRAUMATIC PERICARDITIS IN CATTLE

PATHOGENESIS

- A manifestation of hardware disease (traumatic reticulitoperitonitis)
- ingested wires migrate through the wall of the reticulum, into the peritoneal cavity through the diaphragm into the pericardial sac
- accumulation of septic fluid and gas within the pericardial sac

CLINICAL SIGNS

- Early signs: non-specific
- fever, anorexia, depression
- stand with elbows abducted, or with forequarters elevated
- reluctant to move
- positive grunt test
- Later signs: right-sided heart failure
- venous congestion, peripheral oedema
- tachycardia
- muffling heart sounds
- splashing "washing machine" murmurs
- venous distension
- weak pulses

TREATMENT

- the vast majority of affected cattle are culled
- surgical procedures to strip out the pericardium and remove septic debris have been described

PREVENTION:

- **magnets**

PERICARDITIS IN HORSES

AETIOLOGY AND PATHOLOGY

- the majority of cases are idiopathic
- equine viral arteritis, equine influenza
- Strep. Pneumoniae
- tend to develop fibrinous effusion

CLINICAL SIGNS

- venous distension
- ventral oedema
- muffled heart sounds

- pericardial friction rubs
  (triphasic sounds in time with heart)
- pleural effusion

DIAGNOSIS
- Echocardiography: fluid and fibrin in pericardial sac, compression of cardiac chambers
- Electrocardiography: small complexes, main differential is obesity
- Cytology of pericardial fluid

TREATMENT
- Pericardial drainage and lavage are indicated if the right atrium is collapsing (i.e. cardiac tamponade is present)

PROGNOSIS
- good provided treatment is early and aggressive
- constrictive disease may occur in chronic cases

ANAESTHESIA FOR PATIENTS WITH CARDIOVASCULAR DISEASE

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Introduction

When looking at a patient with cardiopulmonary changes it is important to define the nature and the severity of the changes that have occurred. An animal with an aortic stenosis may respond poorly to a positive inotrope whereas an animal with dilated cardiomyopathy may need such treatment in order to improve cardiac output. It is best to treat any cardiac disease before the procedure if it is thought that such treatment will stabilize or improve the condition of the animal. An animal with hypertrophic cardiomyopathy might be treated with a beta blocker and/or a calcium channel blocker for 1-2 weeks prior to the procedure in order to control the arrhythmias and decrease myocardial wall stress. If pulmonary oedema is present the animal should not be anaesthetized unless the condition requiring therapy is life threatening. These animals often respond to diuretics and the pulmonary oedema may be significantly decreased within a few hours. If the patient has been on diuretics for more than a few days the clinician should do a careful evaluation of hydration status and also check to see if the serum potassium concentration is low.
In dogs the most common cause for a murmur is mitral insufficiency, especially in older small-breed dogs. It is well known that the loudness of the murmur does not necessarily correlate with the degree of pathology and so it is appropriate to try to define the condition before anaesthetizing the animal.
In cats we often detect heart murmurs which are diagnosed as a right ventricular outflow obstruction. In most instances this appears to be a benign change and there are no other changes in the heart indicative of a cardiomyopathy. On the other hand gallop rhythms, even in the absence of a murmur, are often indicative of a cardiomyopathy and should be investigated carefully.
If the heart is failing it may not tolerate an increased fluid load and because of the change in the Frank-Starling curve an increased preload may not result in an increased cardiac output. On the other hand, even a failing heart will not function optimally if preload is allowed to decrease too much. In a prospective human study it was found that the incidence of postoperative cardiac failure was highest in those patients who had received less than 500 ml/hr of fluids intraoperatively. The most common cause of congestive heart failure in dogs is mitral insufficiency. This is characterized by an excessive retrograde flow with an increasing volume load on the heart. Treatment for this condition often involves the use of peripheral vasodilators (e.g. nitroglycerin, hydralazine and ACE inhibitors) to decrease afterload, diuretics and salt restriction to decrease the circulating volume so these patients certainly have the potential to be hypovolemic. The diagnosis of a relative hypovolemia in these patients is based on clinical signs such as mucous membrane hydration, capillary refill time and jugular venous distension. An evaluation of renal function may assist in deciding whether the animal is adequately hydrated. Chest radiographs can be used as a rough indicator of pulmonary venous distension (lack of distension implies lower left atrial pressure and hence lack of preload). The most useful measurement in these cases would be pulmonary capillary wedge pressure (PCWP) - obtained by inserting a balloon tipped catheter into the pulmonary vein

from either the jugular or femoral vein. This kind of aggressive monitoring is certainly warranted in some of these cases and will provide the best guide to fluid therapy. If the animal is in right heart failure then monitoring central venous pressure will give similar information. In the above mentioned human study the use of CVP or PCWP was associated with more aggressive fluid therapy (>500ml/hr) which was associated with a lower risk for postoperative congestive heart failure. In the past it has been recommended that fluids containing low sodium be administered to these patients (solutions such as 0.45% saline/2.5% dextrose or even 0.225%saline/3.75% dextrose). Most of these patients have an increase in total body sodium as well as an increase in total body water and the latter tends to exceed the former so they appear to be hyponatremic. Thus it would seem illogical to give a solution which contains even more free water. If such a patient is hypovolemic then it would be more appropriate to use a balanced electrolyte solution and if it is in its "normal" state it already has an increased volume and fluids may not be needed.
In other myocardial diseases it is also important to assess the patient carefully preoperatively paying particular attention to signs of dehydration and also signs indicative of heart failure (distended jugular veins, slow jugular emptying, jugular pulses, ascites, pulmonary oedema, pleural effusion). Aggressive monitoring as indicated above may be needed in order to optimize fluid therapy throughout the anaesthesia and surgery. Although, rheologically, blood may flow best at a hematocrit of 25-30% it may be necessary to maintain higher values than this in order to maintain tissue oxygenation. If an animal in heart failure is also anemic consideration should be given to preloading the animal with some red cells in order to optimize oxygen delivery.

Anaesthetic management

Many of these patients are on medications to slow the progression of their disease or to minimize the effects that the condition may be having on the animal. We generally do not discontinue these medications and try to give the morning dose(s) of these drugs prior to anaesthesia. If the animal has any significant degree of cardiovascular compromise we generally premedicate with an opioid such as oxymorphone (0.02-0.05 mg/kg), methadone (0.1-0.5 mg/kg), morphine (0.1-0.5 mg/kg) or meperidine (3-5 mg/kg). These drugs provide some sedation - varying with the drug used - and in cats will often induce a state of euphoria (kneading, purring). Their analgesic properties are useful for placing intravenous catheters and they have minimal affect on the heart at the doses usually used for premedication. In most circumstances I would usually combine this with an anticholinergic. In dogs this would be given to combat the vagotonic effect of the opioid and in cats it would be given mainly to decrease airway secretion. This makes it easier to visualize the larynx during intubation and reduces the likelihood of an increased airway resistance intraoperatively. The anticholinergic could be either atropine or glycopyrrolate at a low dose (0.02 mg/kg atropine, 0.005 mg/kg glycopyrrolate). An anticholinergic may not be given if the animal already has a high heart rate.

For patients with bradycardia that is non-responsive to anticholinergic therapy or that have sick sinus syndrome, it is advisable to place a temporary pacemaker until the permanent one can be implanted. This can usually be achieved by placing an introducer (a large thin walled catheter) into a saphenous vein using local anaesthesia and then advancing the pacing lead up to the heart from this site. We avoid using the jugular vein if possible for placing a temporary transvenous

pacemaker so that both vessels are available for this permanent pacemaker if needed. If the patient is too small for a saphenous approach, the lead can be introduced via the jugular vein.

When choosing an induction technique it is important to consider the pathophysiology of the cardiac lesion. An animal with an aortic stenosis will usually have a thickened left ventricle which means that it won't tolerate low heart rates very well (poor myocardial compliance), nor will it tolerate high heart rates (limited coronary reserve). On the other hand an animal with a dilated cardiomyopathy may benefit from an increase in heart rate just to maintain cardiac output. In cases where we are most concerned about maintaining heart rate and contractility we will commonly use etomidate (1 mg/kg), usually in association with diazepam (0.5 mg/kg) or midazolam (0.25 mg/kg). In dogs (but not in cats) where the heart rate can be allowed to decrease we will use an opioid induction. For us this would typically be fentanyl (10 µg/kg) since this drug is the cheapest of the potent opioids in the USA. In Europe this might be alfentanil or sufentanil. The opioid would again be used with a benzodiazepine (diazepam - 0.5 mg/kg or midazolam - 0.25 mg/kg). We generally use these techniques rather than box/mask inductions because we can achieve a smoother transition from awake to asleep by this approach.

As indicated above it is important to plan the fluid therapy of these cases carefully such that the animal maintains a reasonable preload while not overloading the heart. Dogs and cats are usually maintained with an inhalant and if contractility is poor or it is necessary to maintain contractile force in order to cope with the lesion (e.g. Aortic stenosis) we may add a fentanyl infusion. In dogs this would be at a rate of 0.7-1 µg/kg/min while in cats it would be at 0.3-0.4 µg/kg/min. The addition of fentanyl to the inhalant will allow a decrease of the inhalant concentration and this should improve contractility.

It is essential in these cases to be able to monitor the ECG and arterial pressure. The latter could be done non-invasively (oscillometric or doppler) or invasively (arterial catheter with either a manometer or a transducer). Hypotension must be managed according to the lesion in the animal. An animal with dilated cardiomyopathy will benefit from something which increases contractility (dopamine, dobutamine) whereas an animal with a dynamic aortic stenosis will get worse with such treatment and needs a vasoconstrictor (phenylephrine). Arrhythmias should be managed according to their severity and the treatment should account for the nature of the disease (e.g. Cats with cardiomyopathy have been reported to die after therapy with lidocaine and beta-blockers such as esmolol are more appropriate).

Mitral and/or tricuspid regurgitation
This is most common in older small-breed dogs. The basic lesion involves a volume overloading of the heart since blood is pumped back into the atrium and then refills the heart as well as the normal amount of venous blood. In mild degrees of regurgitation there is very little problem with anaesthesia but as the condition progresses the addition of negative inotropic drugs or excessive fluid loads may push the dog into heart failure. In these cases a decrease in heart rate is thought to increase the degree of stretch on the incompetent valve and lead to a greater degree of regurgitation, so it is usually recommended that heart rate be maintained or slightly increased. Any drug that tends to increase the afterload on the heart will tend to worsen regurgitation so vasoconstrictors should be avoided. As indicated above,

these dogs may be on diuretics (to reduce the volume overload) and/or vasodilators (hydralazine or angiotensin converting enzyme inhibitors – to decrease systemic vascular resistance). With these treatments it is expected that the animal might be slightly dehydrated and, with ACE inhibitors, it is common to have some elevation in urea nitrogen because of the effect of these drugs on renal perfusion. This makes it essential to be able to provide fluids intraoperatively and to be able to know the effect of this therapy. The anaesthetic technique should also aim to decrease afterload – acepromazine or other phenothiazines could be used for premedication because they will tend to reduce afterload. Opioids should be used with an anticholinergic in order to prevent bradycardia. Alpha-2 agonists should not be used as they will increase systemic vascular resistance and decrease heart rate. Induction should be carried out carefully since circulation times in these animals can be prolonged, meaning that it takes more time for the drug to get to the brain and if the depth of anaesthesia is not monitored carefully it is easy to give a relative overdose of the induction drug. Etomidate would be the ideal drug although ketamine/tiletamine are also acceptable (both increase heart rate with minimal effect on systemic vascular resistance). Propofol and thiopental are both negative inotropes and would be less desirable for these cases. Maintenance with a balanced technique would be ideal in order to try to avoid the negative inotropic effect of the inhalant as much as possible. While anaesthetized these patients should be monitored using both blood pressure and CVP. The former gives a guide to the effectiveness of myocardial contraction while the latter is reflective of the ability of the heart to generate forward flow. Monitoring heart rate and oxygenation are also important. If the animal becomes hypotensive (and this cannot be managed by decreasing the inhalant or supplying volume) it would be best to use a positive inotrope such as dobutamine in order to increase contractility, while minimizing any effect on afterload.

Dilated cardiomyopathy

Much more common in dogs than cats, this represents a similar spectrum where it is important to maintain preload, keep the heart rate increased and maintain or increase contractility. Afterload should be maintained or reduced. These are very similar goals to those used for mitral or tricuspid regurgitation and so these cases are handled in a similar manner.

Hypertrophic cardiomyopathy

This is seen mainly in cats and may be due to a genetic disorder, hypertension, hyperthyroidism or excessive growth hormone (acromegaly). If possible, it would be best to treat the underlying condition before anaesthetizing the animal. The concerns with these patients are that cardiac output is rather rate dependent since the heart becomes more rigid and cannot expand with increased preload, that coronary perfusion is marginal because of the raised wall tension and increased volume of muscle and that increased contractility may cause a dynamic outflow obstruction from the heart. Consequently the aims of the anaesthetist should be to maintain a fairly normal heart rate. A decreased heart rate will be associated with a decrease in cardiac output while an increased rate will reduce the time for diastolic coronary perfusion and be more likely to lead to ischemia. It is also important not to increase contractility – it may even be beneficial to decrease it to some extent and to ensure an adequate systemic vascular resistance to decrease the likelihood of the dynamic outflow obstruction. To these ends we would typically premedicate with an opioid

and an anticholinergic. Induction with etomidate would be ideal since we cannot use opioid induction techniques in cats. Propofol would be the next best choice but it should be given very carefully since it will reduce afterload and this could exacerbate a dynamic constriction while decreasing coronary perfusion. Thiopental and ketamine/tiletamine should be avoided because they both increase heart rate and thiopental may cause an unacceptable decrease in contractility in a diseased heart. In one study of cats with hypertrophic cardiomyopathy the use of ketamine/tiletamine was identified as a precipitating event in 9 animals that died from congestive heart failure. Maintenance of anaesthesia with an inhalant would be acceptable but a balanced technique would be preferable. Alpha-2 agonists may have a place in these patients in that they increase systemic vascular resistance, decrease the requirement for other anaesthetics and limit the likelihood of tachycardia. In one study a dose of 20 µg/kg medetomidine in awake cats decreased the pressure gradient across the aortic valve from 90 to 5 mm Hg. It is likely that lower doses could be used perioperatively (0.5-1 µg/kg/hour) to provide these benefits without reducing heart rate excessively. As indicated above it would be best to treat hypotension with an alpha-1 agonist (phenylephrine, noradrenaline) rather than a positive inotrope.

Pericardiectomy

This may be undertaken for a pericardial effusion or for a pericardial constriction. Animals with cardiac tamponade, where the intrapericardial pressure approaches the right ventricular diastolic filling pressure, are at immediate risk of circulatory collapse. A pressure of 9 mm Hg caused a 60% reduction in cardiac output while pressures as high as 12-13 mm Hg may be tolerated. Drainage of fluid from the pericardium should be carried out to relieve these symptoms before the animal is anaesthetized. The preferred approach is via sternotomy since this gives the best access to the pericardium but a lateral approach may be used in some cases. Pericardiectomy may also be amenable to endoscopic approaches as well. If an animal with cardiac tamponade must be anaesthetized it should be pretreated with fluids to ensure optimum venous return and a technique used which minimizes reductions in myocardial contractility and heart rate (heart rate is usually elevated in an attempt to maintain cardiac output in the presence of a reduced stroke volume). Etomidate would be the ideal induction drug for this purpose. Once the animal is intubated it is recommended that spontaneous ventilation be maintained to minimize any further reduction in venous return. However, it has also been shown that hypercapnia will further decrease cardiac output so it is better to use IPPV than to allow hypercapnia. If IPPV is used, it is best to use higher breathing frequencies in order to limit peak airway pressures and not to use PEEP. Dobutamine is probably the best drug to use to improve cardiac output since it delayed the onset of tissue hypoxia when compared with norepinephrine, however dopamine also increases cardiac output and improves myocardial perfusion. Once the tamponade has been reduced the anaesthetic management of these patients during the pericardiectomy is relatively straightforward. Ventricular arrhythmias are common during manipulation of the pericardium and so lidocaine should be on hand in case these begin to alter haemodynamic function. Haemorrhage may be marked if a decortication of the epicardium is undertaken and it is necessary to have adequate supplies of typed and cross-matched blood to cope with the blood loss that can occur.

Patent Ductus Arteriosus

A PDA may be corrected by using coils deployed into the vessel from a catheter which is usually put in via the femoral artery. This procedure is relatively non-invasive and has a high success rate. Coils that pass through the ductus and into the lung rarely seem to cause problems whereas coils that get pushed back down the aorta can end up in vessels that are critical (e.g. mesenteric artery). A surgical approach is usually only indicated when the ductal vessel is very large with a high flow passing through it or when the animal is too small to safely use a coil system. Most animals anaesthetized for correction of a PDA are young and are usually in good health, apart from the changes caused by the PDA. In the early stages there are very few myocardial changes associated with a PDA. However, if the ductus is large and/or the lesion has not been recognized early there can be significant enlargement of the left ventricle with cardiac failure, due to volume overload, occurring terminally. If the animal is operated on before significant changes have occurred there are few concerns for the anaesthetist. It is expected that the diastolic pressure will be very low in these patients before ligation of the ductus because of the connection of the systemic circulation to the low resistance pulmonary system. Because of this, phenothiazines and butyrophenones should be avoided for premedication because the alpha-blockade will tend to decrease systemic vascular resistance, decreasing diastolic pressure still further. The maintenance of diastolic pressure is important in the effectiveness of coronary perfusion. Systolic pressures are usually in the normal to high range but, because of the low diastolic pressure, the mean pressure is also normal or reduced and is often in the 50-60 mm Hg range during the approach to the ductus. Positive inotropes may increase systolic pressure and also increase mean pressure but should only be used if systolic pressures fall below 90 mm Hg. Peripheral vasoconstrictors should not be used since an increased systemic vascular resistance will tend to increase the shunt through the ductus and may lead to pulmonary oedema. If there are problems with the dissection of the ductus it is possible to lose large quantities of blood very quickly. A second IV catheter should be placed so that fluids can be given rapidly if needed although the outcome is rarely favorable if the ductus is ruptured. Before the ductus is ligated it is wise to give an anticholinergic because the sudden change in blood pressure associated with ligation can elicit a strong baroreceptor reflex which can even cause cardiac arrest. The anticholinergic blocks the vagal part of this reflex. We typically use the anticholinergic at the time of premedication but it could be given closer to the time of ligation. Monitoring of direct arterial blood pressure in these cases is helpful in allowing the assessment of moment-to-moment changes and is also of use in ensuring that the ductus has been ligated or sufficiently blocked with coils. Typically there is a sudden increase in diastolic pressure as soon as the ductus is tied off or blocked with the coils. In surgical cases a method has been described to induce deliberate hypotension using sodium nitroprusside if bleeding occurs. The mean blood pressure can be quickly decreased to 50-60 mm Hg until the bleeding is under control. The advantage of nitroprusside is that it can be titrated to effect and its effect wears off very rapidly.

Patients with signs of failure or pulmonary oedema should be treated with diuretics and positive inotropes for 1-2 days prior to surgery. The anaesthetic technique should attempt to maintain myocardial function as much as possible and not to cause further decreases in peripheral vascular resistance. Premedication with an opioid/anticholinergic combination is useful and induction with an opioid/benzodiazepine technique or etomidate ± benzodiazepine is preferred. It could be argued that halothane would be a better anaesthetic than isoflurane for these cases because it has less effect on systemic vascular resistance, but the

increased negative inotropic effect and the risk of catecholamine arrhythmias countermand this argument. Maintenance of anaesthesia in these patients should use a balanced technique combining an opioid and/or a muscle relaxant with the inhalant in order to reduce the negative effects of the inhalant. Patients with a right to left shunt are rarely amenable to surgical correction since closure of the duct often results in right heart failure due to the pulmonary hypertension. If such an animal needs to be anaesthetized for part of the diagnostic work up or for other reasons, the regime described for the PDA with some degree of heart failure should be used. It is important to maintain or increase systemic vascular resistance while aiming to reduce (or at least not increase) pulmonary vascular resistance.

Pulmonic stenosis

Management of dogs with pulmonic stenosis is normally by balloon valvuloplasty although a surgical dilation or patch graft technique may also be used. These dogs usually present with right ventricular hypertrophy and it is very rare for them to be in right heart failure. Animals with pressure gradients of <40 mm Hg across the stenosis are at low risk for anaesthetic complications and may be handled in a relatively routine fashion. Animals with gradients >40 mm Hg may have significant hypertrophy and should be regarded as having an increased anaesthetic risk, although it is uncommon to see problems with these cases until the gradient exceeds 80 mm Hg. The right ventricular hypertrophy makes these animals more prone to reduced myocardial perfusion and ventricular arrhythmias. Some cardiologists advocate the use of beta-blockers for a couple of weeks prior to anaesthesia in these cases. The anaesthetic technique chosen for these animals should aim to minimize the possibility of bradycardia. Since the right ventricle is thick and non-compliant, cardiac output is more dependent on heart rate than in a normal animal. At the same time a tachycardia will tend to increase myocardial oxygen demand while shortening diastole and reducing coronary perfusion. These two changes may lead to myocardial ischemia and precipitate severe arrhythmias. Premedication should therefore aim to minimize anxiety and prevent tachycardia. Opioids combined with low doses of anticholinergics will usually achieve this aim. Induction of anaesthesia can be carried out using etomidate ± a benzodiazepine as the technique of choice since there is minimal change in heart rate or contractility. An opioid/benzodiazepine technique can be used as long as an anticholinergic has been given previously and the heart rate is monitored carefully during induction. Maintenance of anaesthesia can be with an inhalant such as isoflurane or a balanced technique can be used with the addition of an opioid or nitrous oxide. Lidocaine should be readily available as a first line treatment for ventricular arrhythmias which may occur during therapy. Animals with a dynamic component to their stenosis should receive phenylephrine for hypotension as described below (aortic stenosis). It is very important to establish the location of the coronary vessels prior to carrying out a balloon valvuloplasty. Some dogs have an aberrant left main coronary vessel that goes around the base of the pulmonary artery and this can be transected with a standard surgical approach or ruptured when a balloon valvuloplasty is carried out.

If a surgical approach is used for this condition the anaesthetist should add a second venous access and have blood products available for rapid transfusion.

Aortic stenosis

Aortic stenosis is also associated with ventricular hypertrophy as it is also caused by a pressure overload. Balloon valvuloplasty for this condition has been very disappointing and the best results with surgical correction have been achieved using

cardiopulmonary bypass. Closed aortic valvotomy has also been described. This requires a thoracotomy and there is a significant risk of haemorrhage and cardiac arrhythmias. If an animal with aortic stenosis must be anaesthetized for diagnostic procedures or non-cardiac surgery then most of the comments above (dealing with right ventricular hypertrophy) apply. However, there is more concern with reduction in systemic vascular resistance since coronary perfusion may be reduced if this occurs and so the phenothiazines and butyrophenones are usually avoided. Management of intraoperative hypotension is also different. Many cases of aortic stenosis have a dynamic component which is exacerbated if a positive inotrope is given. For these animals the author typically uses phenylephrine (2- 5 µg/kg slow bolus and infusion of 0.1-1 µg/kg/min) to treat hypotension since it can be titrated to give an alpha-1 mediated vasoconstriction with minimal positive inotropic effect. For closed aortic valvotomy the same techniques are used but the animal should be cross-matched prior to surgery and have at least two patent intravenous accesses. Lidocaine may be started prior to the ventriculotomy.

Ventricular septal defect with left-to-right shunting
The amount of shunting is going to be largely dependent on the size of the defect. Small defects have a significant resistance to flow so the animal may have a murmur but with very little hemodynamic consequence. With a moderate hole there is a greater flow into the right ventricle during both systole and diastole. The recirculation of blood to the left ventricle leads to a volume overload on this side and the increased flow into the right side may show up as a pressure overload with increased pulmonary circulation. With larger defects the overcirculation leads to failure of the left ventricle and subsequent pulmonary oedema. The aim of medical therapy is to reduce the oedema (diuretics) and decrease afterload so that more flow goes out through the aorta than through the defect (hydralazine or ACE inhibitors). The anaesthetist should also try to do the same thing so an alpha-1 antagonist such as acepromazine would be beneficial whereas an alpha-2 agonist such as medetomidine would be contraindicated. Opioids have little effect on systemic vascular resistance but at high doses may have some effect that increases pulmonary vascular resistance. It is unclear whether this is a significant benefit at clinical doses. Induction of anaesthesia with propofol would be a reasonable choice since it decreases systemic vascular resistance, an opioid technique would also be reasonable. Etomidate has little effect on systemic vascular resistance so it is not beneficial but could be used. Thiopental should not be used and the dissociative drugs, while not changing systemic vascular resistance a great deal, may increase heart rate with a subsequent increase in myocardial oxygen demand. Maintenance with an inhalant would be adequate but a balanced technique may provide a little more control over hemodynamic changes. The only surgical approach to these cases without cardiopulmonary bypass is a pulmonary banding technique so the usual issues arise for a thoracotomy. Many of these animals will be anaesthetized for other reasons (e.g. diagnostics, spay or castration) than to treat the condition. If hypotension occurs it can be treated with dobutamine. This would be preferable to dopamine since it is less likely to increase systemic vascular resistance.

Ventricular septal defect with right-to-left shunting (Eisenmenger's complex)
With a large septal defect the increased flow through the pulmonary circulation can lead to thickening and fibrosis in the pulmonary vessels with an increase in pulmonary vascular resistance. When this exceeds the systemic vascular resistance the shunt changes to a right-to-left shunt and the animal will have low PaO_2s

(cyanosis). There is no definitive treatment for this condition and it carries a poor prognosis. If the animal has to be anaesthetized the main aim is to try to limit the right-to-left shunt in order to improve systemic oxygenation. This can be achieved to some extent by increasing systemic vascular resistance so medetomidine may be useful in these patients. Care must be taken not to overdo this because the increased load on the right heart might put it into failure. Monitoring central venous pressure is helpful in these cases. Induction with etomidate would be ideal. An opioid technique would be acceptable from a cardiovascular viewpoint but leads to significant respiratory depression that would necessitate IPPV. Increasing intrathoracic pressure may further increase pulmonary resistance and exacerbate the shunt. Thiopental could be used but in practice I would be concerned about the negative effects on a compromised myocardium and the dissociative drugs would certainly increase oxygen demand which would be a negative effect in an hypoxemic animal. Hypotension should be treated with an alpha-1 agonist such as phenylephrine or noradrenaline.

Tetralogy of Fallot

This consists of a large ventricular septal defect, pulmonic stenosis, dextroposition of the aorta and right ventricular hypertrophy. The pulmonic stenosis and septal defect results in venous blood being shunted into the systemic circulation with subsequent systemic hypoxemia. The latter stimulates the production of erythropoietin and results in a significant polycythaemia and it is not uncommon for these animals to have haematocrits that exceed 0.7 L/L and this creates a substantial increase in blood viscosity. If an animal with this defect needs to be anaesthetized the first therapy is to reduce the haematocrit to around 0.6-0.65 L/L by removing blood and replacing it with 2-3 times the volume of crystalloid using the formula:

$$\text{Vol blood to be removed (mL)} =$$

$$(\text{BWkg} \times 0.08) \times 1000 \times \frac{\text{Actual Hct} - \text{Target Hct (L/L)}}{\text{Actual Hct}}$$

Care should be taken not to decrease the Hct excessively since this will result in decreased oxygen delivery to the tissues. These dogs may be on beta blockers to reduce the dynamic component of their pulmonic stenosis. The aim of the anaesthetist is to ensure adequate perfusion and to prevent excessive decreases in systemic vascular resistance. Hence acepromazine would be contraindicated while medetomidine may be useful for these cases. Induction with propofol (vasodilation) and dissociatives (increased oxygen demand) would be less desirable than the use of etomidate, thiopental or an opioid technique. Hypotension should be treated with an alpha-1 agonist such as phenylephrine and even in the absence of hypotension this may be helpful to reduce the right-to-left shunting and decrease systemic hypoxia.

Bradyarrhythmias and Pacemaker implantation

Pacemakers are used to treat third degree atrioventricular block, sick sinus syndrome with bradycardia or malignant vagal syndromes that are unresponsive to medical management. Some of these cases are atropine responsive when awake but may not respond when anaesthetized. We have anaesthetized many patients with a stable 3rd degree AV block without encountering any major difficulties. We use drugs that are not likely to further decrease heart rate and have a positive chronotrope on

hand (e.g. dopamine) to increase heart rate if it becomes too low. However, for the other bradyarrhythmias and now for most of the 3rd degree AV block patients we place a temporary transvenous pacemaker in the awake patient as described above. Once the pacemaker is in place there is little concern about the cardiovascular effects of the induction method unless the arrhythmia is also accompanied by other myocardial pathology. Some centers have used external pacing rather than a transvenous technique. We have used this as a backup but have generally found this to be more difficult to manage because of the thoracic and diaphragmatic contractions induced by the external pacemaker. Animals do not tolerate this very well even with good sedation. Some animals present with bradyarrhythmias and need to be anaesthetized but are not going to have a permanent pacemaker implanted. Here again we use a temporary pacemaker and put it in sensing mode so that it only fires if the heart rate decreases below a predetermined value. We typically turn the pacemaker off once the animal has recovered from anaesthesia but leave the lead in place for several hours so that it can be used if the bradyarrhythmia leads to syncope.

Perioperative analgesia

Since cardiac surgeries involve a thoracotomy it is important to provide good analgesia to limit the sympathetic stimulation that may occur with pain. In general in the dog and cat a median sternotomy is a lot more painful than a lateral approach to the chest. We use systemic opioids extensively for these cases and they are often given by infusion during and after the procedure (e.g. fentanyl at 0.7 µg/kg/min intraoperatively followed by 0.05-0.1 µg/kg/min postoperatively or morphine at 0.1-0.5 mg/kg/hour). Epidural opioids are also used either as a single injection or through an epidural catheter. For a median sternotomy we will usually advance an epidural catheter up to the mid thoracic level and give morphine at 0.1 mg/kg (0.1 mL/kg) every 6 hours. Local anesthetics may be used to provide intrapleural analgesia. At the end of surgery we place the animal with its incision on the operating table and then give 1 mg/kg of bupivacaine diluted in 0.5-1 mL/kg through the chest drain and leave the animal in that position for ten minutes before taking them to recovery. For lateral throacotomies I usually ask the surgeon to block 3-5 intercostal nerves at the end of surgery. I give them a syringe containing 2 mg/kg bupivacaine and get them to inject this as close to the spinal column as possible, where the intercostal nerve exits from the cord. This technique will block the dorsal branches of the intercostal nerve which are not normally affected by the standard external approach to the intercostal nerves. Dogs and cats that have undergone a median sternotomy and have poor pain control will not lie down and this adds to their suffering since they cannot sleep. This is a clear sign that further analgesic therapy is warranted.

Cardiovascular dysfunction during anaesthesia in large animals.

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Depression of cardiovascular function during general anaesthesia in horses is thought to be one of the main contributing factors to the high perioperative mortality in this species. Consequently there have been many studies on the effects of anaesthetic agents on cardiac function. However, how is cardiac function measured and what are the limitations of these measurements?

One of the first measures of cardiovascular function in horses was in 1726 by the English clergyman Stephen Hayles who measured arterial pressure from the carotid artery of a conscious mare. Whilst we no longer use a 9'6" glass tube, brass pipe and goose trachea for this measurement, the recording of arterial pressure (AP) remains the commonest method of monitoring cardiovascular function during equine anaesthesia (Hubbel 1991). However arterial pressure gives very limited information on cardiac function, being a product of systemic vascular resistance (SVR) and cardiac output (Qt). Recent studies in horses maintained under general anaesthesia with inhalant anaesthetics and undergoing surgery have supported experimental studies showing an inverse relationship between Qt and mean AP (MAP). A mean increase in MAP of 10 mmHg is significantly associated with a mean drop in Qt of 1.8 L minute-1 (95% CI: -2.7 to –0.9) (Raisis *et al.* 2005). Measurement of Qt, arterial pressure and calculation of SVR allows more accurate assessment of a horse's cardiovascular status and response to therapy.

Measurement of cardiac output – Fick technique

Stephen Hayles was one of the first to measure cardiac output. Following exsanguination of a 14.3HH (1.5 metres) mare he filled the left ventricle with melted beeswax and measured the volume of the solidified cast. He erroneously assumed that the ventricle emptied completely during systole and calculated Qt as the measured stroke volume (SV) x heart rate (HR). The volume of the cast was 160 ml, HR was 36 beats per minute and therefore Qt was calculated as 6 L min^{-1}. It was not until 1898 that Qt was measured in conscious horses (Zuntz and Hagermann 1898) using the method first proposed in 1870 by Adolf Fick. Zuntz and Hagermann recorded a Qt of 75 mL kg^{-1} min^{-1}, using these values the Qt of Hayle's mare would have been 36 L min^{-1}. The Fick principle of measuring Qt ($Qt = (Ca – Cv)/VO_2$) has several limitations. Cardiac catheterisation is required to withdraw mixed venous blood samples. The requirement for simultaneous mixed blood and arterial samples has been suggested to be a limitation in horses (Muir, Skarda and Milne 1976). Measurement of VO_2 is slow, as steady state conditions are necessary (Stowe and Good 1961), thus preventing beat-to-beat changes in Qt. A facemask is also required which may result in inaccurate measurement of VO_2 (Fisher and Dalton 1961). Stowe and Good (1961) reported a measurement error of up to 10% in the methods used to measure oxygen consumption and a variation of 15% and greater when measurements were made in quick succession, despite attempts to measure output during steady state conditions.

Measurement of cardiac output – Indicator dilution techniques

Measurement of Qt by the indicator dilution method involves the injection of a known mass of indicator into the venous system. The indicator is diluted in the venous blood and passes through the lungs into the systemic circulation. The concentration of indicator in the arterial blood allows volume to be calculated.

$$\text{Concentration (C)} = \frac{\text{injected mass (m)}}{\text{Plasma volume (v)}}$$

Stewart (1897) first proposed an indicator dilution method to measure Qt. Using saline as the indicator, concentration was determined from the change in electrical conductance of blood in the femoral artery. A modification of this technique used an indicator dye, Qt being determined by colorimetry of consecutive arterial blood samples. In vitro experiments showed the method predicted flow with an average error of 3.2% (Kinsman, Moore and Hamilton 1929). However when used in vivo, following the initial decay of the concentration curve there was a secondary increase in dye concentration due to recirculation of the indicator. As Qt is calculated from the concentration curve of dye from the first circulation, it is important to use some form of extrapolation to determine the area under the primary curve. Fisher and Dalton (1959) used the dye dilution technique to record Qt in horses. The mean Qt recorded in this study (74 mL kg^{-1} min^{-1}) was similar to that measured in horses by Zuntz and Hagermann (1898) using the Fick principle *(75* mL kg^{-1} min^{-1}). Dye dilution techniques have also been used to study changes in Qt in horses under general anaesthesia (Eberley *et al.* 1968; Hall, Gillespie and Tyler 1968; Gillespie, Tyler and Hall 1969; Hillidge and Lees *1975.)* A technique is now commercially available (LiDCO), which uses Lithium Chloride as the indicator and a flow through cell housing a lithium selective electrode to measure the arterial plasma concentration time curve (Linton *et al* 2000).

Indicator dilution methods have the advantage over the Fick technique that facemasks and ventricular catheterisation are not required and time resolution is improved. However recirculation of dye is a problem, which causes error from extrapolation of dye concentration decay curves, especially in diseased hearts. Additionally, some indicators have strict dosage limitations, and others may be lost during passage through the lungs.

Measurement of cardiac output – Thermodilution technique

Fegler (1954) used a thermal indicator to measure Qt in dogs. In this thermodilution method the indicator was a known volume of cold fluid, the dilution curve being obtained from changes in blood temperature measured by a thermistor. The main criticisms of the thermodilution technique were associated with potential losses of cold indicator to the tissues. The thermal indicator must be injected directly into the right atrium rather than a central vein, to prevent overestimation of Qt at low flows due to increased heat loss to the tissues between the site of injection and the thermistor. The Swan-Ganz catheter (Ganz *et al* 1971) has a thermistor located at its tip, which is positioned in the pulmonary artery, and a proximal port, which ends at a side hole, which is located in the right atrium. To avoid error, the thermal indicator should be injected as rapidly as possible into the atrium. The time taken to inject large volumes of saline through the proximal port is a limitation to the use of Swan Ganz catheters in large animals and a second, wider bore catheter is commonly placed in the right atrium. The use of a pressure injector is recommended if large volumes of indicator are used to reduce the injection time. The technique has been reported in horses (Muir, Skarda and Milne 1976, Dunlop *et al* 1992). A further variation of the thermodilution technique uses heat produced by a heating coil, wrapped around part of the catheter, as the thermal indicator (Yelderman 1992).

Recirculation of indicator, a problem in the dye dilution technique is not a major problem in the thermodilution technique because the heat is lost in the periphery. Incomplete mixing of the cold indicator with blood causes error in estimations of Qt by thermodilution however this is the case with all indicator techniques. Inadequate mixing will be evident from the bizarre shape of the dilution curves therefore it is important that any equipment used displays the dilution curve as well as the Qt. The shape of dilution curves also changes if the thermistor is lying against the vessel wall.

A further problem associated with the use of a thermal indicator is the heat gained by the indicator, from the catheter before injection: the lower the temperature of the injectate, the more pronounced the problem. It is essential that the exact temperature of the injectate be known, to ensure accurate Qt estimations. Solutions placed in an ice bath for cooling prior to injection require 45 to 60 minutes to reach a steady-state temperature. Temperature probes inserted directly into the ice will underestimate the temperature of the injectate even after this time (Levett and Repogle 1979). A more accurate determination of injectate temperature is possible when an injectate at room temperature is used however a larger volume is then required to achieve the required calorific difference for the equipment used (Calorific difference = (Blood temp – Injectate temp) x Injectate volume). The pulmonary artery temperature has been shown to fluctuate in phase with respiration causing an unstable base line against which changes in temperature are recorded. Although errors can be minimized by measuring Qt at the same time in the respiratory cycle, as the variation in temperature is caused by changes in the venous return, this would further bias the Qt estimation. Increasing the amount of indicator will increase the signal to noise ratio and reduce errors; this is achieved either by increasing the volume of fluid injected, or by increasing the temperature difference between the injectate and the blood. The disadvantage of increasing the volume of the injectate is the associated risk of altering the Qt and increasing the injection time. A disadvantage of the use of iced indicators is a possible decrease in body temperature if a large number of determinations are made; piloerection and shivering has been observed in a horse after only four thermodilution estimations. Potential errors due to increased loss of indicator prior to injection and inaccurate measurement of injectate temperature are greater when an iced indicator is used (Evonuk *et al.* 1960). However greater variability has been reported when room temperature indicators are used in horses (Muir, Skarda and Milne 1976) however, the temperature of the room was high (27^{o}C).

Cardiac output computers from different manufacturers differ in the methods used to estimate the area under the curve, especially the method used to define the tail of the curve. The shorter the time base of this calculation, the greater the sampling error, especially with pulsatile flow. This is a problem in the use of the thermodilution technique to measure Qt in horses with second degree atrioventricular block. If Qt is measured during the conducted beats, flow will be overestimated, whereas if Qt is measured during the prolonged diastole, flow will be underestimated.

The thermodilution technique has the advantage of avoiding the build up of foreign substances in the body, repeated estimations can be made quickly and if the thermistor has a rapid response time, beat-to-beat changes in stroke volume can be measured. In contrast to other indicator dilution techniques, blood samples are not required for calibration or measurements. Estimations of Qt by thermodilution have correlated closely with dye dilution techniques in horses (Muir, Skarda and Milne 1976; Dunlop *et al*, 1992). However correlation only gives a measure of the linear relationship between the two techniques (Altman 1980). It would be expected that two techniques designed to record the same variable would show a close correlation (Bland and Altman 1986). A close correlation would be expected between thermodilution and dye dilution, as both are based on the same principle, and therefore both are subject to similar errors.

Despite the technical limitations, many authors believe that with due precautions and attention to detail, thermodilution provides an accurate means of measuring Qt and may be considered a reference technique (Stetz *et al.* 1982), other authors have concluded that little reliance can be placed on any absolute measurement of Qt by thermodilution, due to large errors in conditions of pulsatile flow (Mackenzie, Haites and Rawles 1986). The major problems associated with the use of Swann-Ganz catheters are dysrhythmias and damage to the heart, valves and great vessels (Schlipf *et al* 1994). In one study in humans, the mortality of intensive care patients

was 24% higher in those patients that had a Swan – Ganz catheter in place compared to those that did not (Connors *et al* 1996).

Measurement of Cardiac output – Doppler echocardiographic techniques

Light (1969) first reported the potential value of Doppler echocardiography for measuring left ventricular stroke volume noninvasively in humans. Flow through a vessel can be calculated from the product of the mean flow velocity (VTI) and the cross sectional area of the vessel, measured at the same site. Calculation of flow by Doppler echocardiography is based on a number of assumptions. The cross sectional area of the vessel is assumed to be constant during ventricular ejection although the areas of the aorta and pulmonary artery have been shown to vary in phase with the cardiac cycle. However it is not necessary to measure vessel area during maximal distension, as this only occurs transiently during systole and an average of a number of measurements during systole is usually used. The velocity profile at the sampling site is assumed to be flat and the measured velocity is assumed to be accurate. The latter assumption requires the ultrasound beam to be parallel with flow. The main error with the Doppler technique is in the measurement of the diameter of the vessel, which is magnified in calculation of the area. For this reason it has been proposed that VTI can be used to reflect changes in stroke volume without the need to measure vessel diameter, and VTI x HR used to indicate Qt. There have been a number of reports of the use of Doppler echocardiography to measure Qt in horses (Mizuno, *et al* 1994, Blissitt *et al* 1997, Linton, *et al* 2000).

Other methods of measuring Qt include: Radionuclide ventriculography; Echocardiography; Electromagnetic flowmetry. However in practice the method most commonly employed is the subjective palpation of the arterial pulse.

Isovolumic indices of ventricular function

Cardiac output provides a measure of cardiac performance, defined as the pumping ability of the heart. However measurement of Qt does not indicate the mechanism behind changes in cardiac performance. Cardiac output is influenced by factors affecting stroke volume (preload, afterload and contractility) and heart rate. The ideal measure of cardiac contractility is a variable generated by cardiac contraction that is independent of the initial fibre length (heart size) and is not affected by changes in afterload (aortic impedance) (Van den Bos *et al* 1973) that is a measure of what the heart is capable of doing rather than what it actually does (Milnor 1990). A large number of variables have been proposed as suitable indices of cardiac contractility. Most are measured in the isovolumic phase of systole i.e. before the aortic valve opens, and therefore should not be affected by afterload. During this phase, the rate of rise of intraventricular pressure (LVdp/dt) is closely related to the simultaneous rate of change of wall tension (Wallace, Skinner and Mitchell 1963). Although it was initially thought that the maximum LVdp/dt ($LVdp/dt_{max)}$ may be independent of changes in preload, as the increased force caused by increased heart size would be balanced by an increased chamber diameter (Van den Bos *et al* 1973), the increased force caused by increased fibre length and the Frank-Starling mechanism dominated the reduction in wall tension by the Laplace effect (Wallace, Skinner and Mitchell, 1963). It has also been shown that $LVdp/dt_{max}$ is influenced by aortic diastolic pressure, (Wallace Skinner and Mitchell, 1963) and it is sensitive to changes in heart rate. Despite these limitations $LVdp/dt_{max}$ has been used in humans (Braunwald 1992) and horses (Hillidge and Lees 1976, Muir 1992, Brown and Holmes 1979) as it shows sensitivity to agents known to produce acute changes in contractility and is relatively simple to obtain.

Ejection phase indices of ventricular function

Indices of myocardial contractility have also been developed based on the maximum acceleration of aortic blood flow (dv/dt_{max}). This was first proposed by

Rushmer in 1964 who used the term 'Initial ventricular impulse' to describe the dynamic properties of left ventricular ejection. Impulse was defined as force x time, and as force = mass x acceleration, Noble et al (1966) suggested that the force exerted by the LV is proportional to the maximum acceleration of the blood. However the use of $LVdv/dt_{max}$ is only valid if the whole column of blood moves from the ventricle in a single mass and if the force is expended to accelerate column of blood not overcome resistance (Noble 1965). At $LVdv/dt_{max}$ only 5% of the stroke volume has been ejected. However $LVdv/dt_{max}$ is not preload independent unless the increase in force by Frank Starling is offset by the increase in the mass of blood to be ejected. It is also not independent of afterload except within a physiological range of pressures however it has shown increased inotropic sensitivity and less afterload and preload dependence than $LVdp/dt_{max}$ (Lambert, Nichols and Pepine, 1983). The measurement of $LVdv/dt_{max}$ requires implantation of electromagnetic flowmeters although velocity of aortic blood flow can now be recorded non invasively by Doppler echocardiography. However it must be remembered that the maximum acceleration recoded from Doppler velocity spectra is not the same as the true maximum acceleration obtained by differentiation of the signal from a high fidelity flowmeter and therefore is more likely to be load dependent. The maximum acceleration derived from the Doppler velocity spectra is merely a measure obtained of the steepest gradient of the upstroke of the velocity spectrum. The $LVdv/dt_{max}$ obtained from Doppler waveforms has been shown to be inversely related to afterload and directly related to preload. However it is more sensitive to changes in inotropy than in preload and afterload and is as sensitive as conventional invasive indices to myocardial contractility although no better at defining inotropy in absolute terms (Wallmeyer, *et al* 1986). Young and others (1995) have validated transoesophageal echocardiography in anaesthetized horses and have shown $LVdv/dt_{max}$ to be as sensitive as $LVdp/dt_{max}$ to changes in inotropy and no more load dependent than LVdp/dtmax. These authors also assessed other ejection phase indices: the peak velocity (Vmax); pre ejection period (PEP); and ejection time (ET). Ejection time and PEP are heart rate-dependent indices of cardiac function influenced by preload, afterload and myocardial contractility. The pre-ejection period is the duration between the onset of electrical activation of the ventricle and the onset of ejection. It is increased with decreased myocardial contractility, decreased preload and increased left ventricular afterload. In contrast, left ventricular ejection time increases with increased myocardial contractility and preload, and decreases with increased ventricular afterload.

Cardiac function during anaesthesia

In addition to the direct effects of anaesthetic agents on preload afterload, contractility and heart rate, in vivo, the cardiac effects of anaesthetic and other agents on cardiac function is further complicated by simultaneous alterations in autonomic nervous system activity, which differs between species and individuals within species. In humans and dogs dobutamine has very little pressor effect whereas in horses there is a marked increase in arterial blood pressure with often minimal changes in Qt and heart rate. In individual horses Qt may decrease during dobutamine infusion, due to a decrease in heart rate secondary to the large increase in arterial blood pressure.

The inhalant anaesthetics commonly used in equine anaesthesia, produce a dose dependent decrease in cardiac function with halothane producing the greatest depression. Although surgical stimulation was thought to partially offset the depressant effects of halothane, recent studies have shown a temporal decrease in Qt during surgery under halothane anaesthesia. It is not clear whether this temporal decrease in cardiac function, which also occurs with isoflurane, is due to reduced contractility, decreased preload or increased afterload. Qt is better maintained with isoflurane, however depression of respiratory function is more marked. The

increased need for mechanical ventilation further reduces Qt, although Qt still appears to be better maintained than with halothane. However despite the improved cardiac performance with isoflurane, recent studies have shown no difference in mortality between horses anaesthetized with isoflurane when compared to halothane (Johnston 2004).

In the normal animal Qt is regulated more by the loading conditions of the heart than by the contractile function of the myocardium. Horses with higher arterial pressures during anaesthesia have significantly lower Qt. Drugs causing vasoconstriction, used to maintain arterial pressure, also significantly reduce Qt. Conversely treatment of hypertension with acepromazine increases Qt, presumably as a result of decreased afterload. Although recent data has demonstrated changes in ventricular function during surgical anaesthesia, these findings did not assess loading conditions and therefore failed to demonstrate the mechanism underlying these changes (Raisis *et al* 2005). Improvements in non-invasive methods of monitoring diastolic function under surgical conditions are needed to fully understand the mechanism of cardiovascular dysfunction in individual subjects during anaesthesia.

Anaesthesia of horses with cardiovascular disease

Although it is said that horses with cardiac disease are not good candidates for surgery (Robertson 1999), many horses have cardiovascular disease and many horses presenting with obvious cardiac dysfunction are anaesthetized successfully. This is probably due to the huge cardiac reserve in horses compared to other species. The importance of a thorough pre anaesthetic examination cannot be overemphasized in horses with cardiac disease as horses with severe cardiac compromise can be easily overlooked if only a cursory examination is given. In all cases with cardiac disease, it is important to make a diagnosis, assess the severity and understand the pathophysiology of the presenting condition. The commonest causes of cardiac murmurs in horses are those caused by mitral, tricuspid and aortic regurgitation and ventricular septal defects. All of these conditions results in volume overload. Pressure overload only commonly occurs as a result of pulmonary hypertension secondary to left ventricular failure.

The other common presentation in horses with cardiac disease is dysrhythmias. Again it is important to try to understand the pathogenesis of any dysrhythmia. Many dysrhythmias are normal in horses and are associated with high vagal tone. Atrial fibrillation occurs commonly in large horses with no underlying cardiac disease and these horses must be differentiated from those horses with atrial fibrillation that has occurred secondary to atrial dilatation as a result of disease. It is important to appreciate that one of the side effects of drugs (Quinidine Sulphate) used to treat atrial fibrillation is sudden death. If atrial fibrillation is detected as an incidental finding and it is not affecting performance it may be considered unreasonable to put the horse at risk by suggesting treatment of the dysrhythmia prior to anaesthesia. In these cases and in other cases with atrial tachyarrhythmias, ventricular rate is usually controlled by the normal high resting vagal tone. If ventricular rate is not normal further investigations are necessary before anaesthesia to identify further underlying cardiac disease. Care should be taken with these cases when using sympathomimmetic or vagolytic agents that may increase conduction through the atrioventricular node.

Many horses present with isolated premature supraventricular or ventricular beats, or these may develop spontaneously during anaesthesia. If isolated arrhythmias are identified, try to identify and correct the underlying cause (e.g. hypoxaemia hypercapnia if under anaesthesia) treat only when they are affecting cardiac performance or if there is a risk that they are going to develop into something more sinister. Pulseless electrical activity and electro-mechanical dissociation also develop spontaneously in horses. Where pulseless electrical activity occurs as a

result of relative anaesthetic overdose, inotropic support can be life saving, however when electromechanical dissociation occurs as a sporadic event successful treatment options are limited.

Uncontrolled cardiac failure is a contra-indication to anaesthesia, and most large animals with advanced cardiac disease are still euthanased. This may change in the future especially in horses with mitral valve disease, when owners of valuable racehorses may be willing to pay for the repair of ruptured chordae tendineae. This will be a major challenge to the veterinary anaesthetist, requiring cardiac bypass and the maintenance of perfusion in a species prone to the development of post operative myopathies and with a high incidence of perioperative mortality.

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