Veterinary Bioscience: Cardiovascular System

WEEK 1 – THE HEART IN THE THORAX

LECTURER: PROFESSOR LIZ TUDOR

Liz Tudor is a Professor in Veterinary Biosciences in the Faculty of Veterinary and Agricultural Sciences and teaches into multiple Veterinary Bioscience modules in DVM 1 and 2. She is also the Director of the Melbourne Academy of Veterinary and Agricultural Learning and Teaching (MAVALT). Liz is a veterinarian who completed a PhD in the Faculty of Medicine at Monash University, and worked for ten years in private small animal practice in Melbourne. She has an ongoing interest in the development of dog health programs in remote indigenous communities. Her research interests are in bone and muscle biology, and the pharmacology of pain management in animals.

Email: etudor@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Explain how the electrical and mechanical events of the heart are linked to achieve effective pumping.
- In particular you should be able to:
- List the major distinct phases of the cardiac cycle as delineated by valve opening and closure.
- Describe the pressure changes in the atria, ventricles and the aorta during each phase of the cardiac cycle.
- State similarities and differences between mechanical events in the left and right heart pump.
- Describe the function of the heart valves
- Explain the origin of the heart sounds.
- State the relationship between cardiac output, heart rate and stroke volume.
- Identify the major determinants of stroke volume.

KEYWORDS

Diastole, systole, atrioventricular valve, aortic semilunar valve, end diastolic volume, ejection fraction, stroke volume, heart sounds, murmur, stenosis, regurgitation.

LECTURE 6 – THE CARDIAC CYCLE- MEASURING THE ACTIVITY OF THE PUMP

The cardiac cycle describes a sequence of co-ordinated electrical and mechanical events that ensure effective cardiac pump function. For effective function blood must flow in only forward direction (this is achieved by valve function) and the ventricles must repetitively fill and empty.

This lecture will describe the events of the cardiac cycle- how the electrical and contractile activities are linked- and the pressure changes that occur during the cycle. It will be useful for you to refer to the figure (following page) during this lecture.

The two phases of the cardiac cycle are **systole**- contraction, and **diastole**- relaxation.

Towards end of diastole, all heart chambers are relaxed.

- The valves between atria and ventricles are open (atrial pressure is slightly greater than ventricular pressure)
- Pulmonary and aortic valves closed (aortic and pulmonary pressure slightly higher than ventricular)

The cycle begins when SA node initiates the heart beat

- atrial depolarization causes the **P wave** of the ECG

Atrial systole – contraction of atria completes ventricular filling

- Normally blood flows directly from vena cava to atria to ventricles.
- Up to 80% of ventricular filling is contributed by venous pressure and atrial contraction provides only 20% of ventricular filling at rest.
- As heart rate rises, atrial contribution rises, as there is less time in diastole for filling to occur
- an animal can cope at rest with non functional atria (e.g. atrial fibrillation or AV block), but under stress the absence of atrial pumping causes 30-40% loss of cardiac efficiency.
- There are no valves between the atria and the great veins, so some regurgitation of blood occurs during atrial contraction. This can be observed as a pressure wave in the jugular vein that corresponds with atrial contraction.
- The ventricular volume at end of atrial systole (ventricular diastole) is refered to as **end diastolic volume**
- EDV in a 25kg dog is approximately 40 ml
- end diastolic ventricular pressure is low. (but is higher in left than right ventricle due to the thicker ventricular wall)

Ventricular systole involves two phases:

Ventricular systole is preceded by the QRS complex of the ECG, as depolarisation sweeps through the conducting pathways of the ventricles and across the ventricular wall.

The first phase of ventricular systole is an Isovolumetric (isometric) contraction. The onset of ventricular systole causes an increase in ventricular pressure that causes the atrioventricular valves to close, but time is required (.02-.03 sec) for ventricular pressure to build above aortic pressure in order for the semi-lunar (aortic and pulmonary) valves to open.

Increasing pressure during isovolumetric contraction causes AV valves to bulge into the atria, causing an atrial pressure wave (c wave) in the great veins.

The second phase is a period of ejection. Initially ejection is very rapid- ventricular pressure rises above diastolic arterial pressure, (80mm in left ventricle, 8 mm right ventricle), the semilunar valves open and blood flows into the arteries. Flow into the arteries is initially very rapid. (50% of emptying occurs in first quarter of systole.) In the later stages of the ejection phase, almost no blood flows from the ventricle to arteries, yet the ventricles remain contracted. The aortic pressure begins to fall because blood is leaving the aorta and large vessels faster than it is entering from the left ventricle.

Eventually flow briefly reverses causing the outlet valves (aortic semilunar and pulmonary valves) to close. The abrupt closure of the aortic valve causes a transient dip in pressure, which is seen on the pressure trace as the **dichrotic notch**, as a small volume of blood flows back to fill the leaves of the valve.

Active contraction then ceases and ventricular muscle repolarizes, associated with **T wave** of ECG. At the end of ventricular systole, build up of blood (from veins) in atria, causes another atrial pressure wave described as the atrial **v wave.**

VENTRICULAR DIASTOLE- RELAXATION AND FILLING

Appropriate relaxation of the ventricles is essential for effective filling and cardiac pumping. At rest diastole is twice the length of systole, but it decreases with exercise and increase in heart rate.

Following closure of the outflow valves, the rapid relaxation of the ventricles occurs, but ventricular pressure still exceeds atrial pressure so the AV valves remain closed. As ventricular pressure falls below atrial pressure, the AV valves open, and atrial pressure falls rapidly as ventricles fill. This is assisted by elastic recoil of ventricular walls, effectively sucking blood in. The ventricles then relax fully, and filling slows.

These events can be described as pressure or volume changes against time, or as pressure volume loops. The pressure volume loop shows coincidental pressures and volumes in sequence during one cardiac cycle.

THE RIGHT PUMP

The heart has a single electrical system**,** so similar mechanical events occur simultaneously in both sides of heart. Two sides are arranged in series so flows must be equal and stroke volumes must be equal. The major difference is in the magnitude of systolic pressures. The lungs have much lower resistance to flow, so less arterial pressure required.

As there are no valves between the atria and the great veins, atrial pressure waves are transmitted in retrograde fashion to the great veins (e.g. jugular v), and these can be useful in clinical assessment.

MEASURING THE PERFORMANCE OF THE PUMP

A number of parameters can be measured to assess the efficacy of the cardiac pump. Below are some important terms that relate to performance of the heart as a pump.

ESV: end systolic volume: blood volume in ventricle at end of systole EDV: amount of blood in the ventricle at the completion of filling

Stroke volume (SV)- volume of blood ejected from the heart with each contraction SV= EDV-ESV Ejection fraction: the proportion of EDV ejected with each contraction

EF=SV/EDV for example if SV = 70ml and EDV = 120 ml, Ejection fraction is 70/120 or 58% Cardiac output (CO)=heart rate (HR) X stroke volume (SV)

CARDIAC VALVES AND THE ORIGIN OF THE HEART SOUNDS

The movements of valve leaflets are essentially passive. Orientation of the valves is responsible for unidirectional flow. Valves are set in distinct outpocketings of the vessel walls. Movement of blood past the valves creates eddy currents in these sinuses, which keep valves floating in current (mid way between the closed position and vessel wall), and prevent obstruction of the openings to the coronary arteries. Valves open when pressure in proximal chamber exceeds pressure in the distal chamber and close when pressure in the distal chamber exceeds pressure in the proximal chamber. The papillary muscles do not help close valvular orifice, but prevent excessive bulging of valves into the atrial chambers during ventricular contraction.

The first and second heart sounds mark the beginning and end of ventricular systole.

The **first heart sound** is associated with closure of AV valves. It is caused by oscillation of blood in the ventricular chambers and vibration of chamber walls, following closure of the valves. The vibrations are transmitted to the chest wall, and are best heard on the left side of thorax, 4^{th} and 5^{th} intercostal space, at the apex beat as a low frequency "lubb" sound.

The **second heart sound** is caused by sudden closure of aortic and pulmonary semilunar valves. The vibrations travel through fluid until they reach chest wall (sounding board). Hence the site of maximal intensity of sound on chest wall may not correspond to position of valves. It is shorter, sharper and higher pitched sound than the first sound - "dup".

The third and fourth heart sounds are less audible in most species.

The third heart sound is weak and not normally heard with stethoscope except in horses. It occurs in mid diastole and is believed to be due to vibrations of ventricular wall produced by deceleration of blood entering the ventricles. It can be an indicator of pathology in other species. The fourth heart sound is a very low frequency sound that occurs when the atria contract. It is caused by inrush of blood to the ventricles, initiating vibrations similar to third heart sound.

ABNORMAL HEART SOUNDS

Murmurs are caused by turbulent blood flow, and may reflect valve defects.

Stenosis : the valve produces a narrowing of the orifice, so that flow through the narrowed orifice is turbulent.

Incompetence: the valve leaks blood while closed, and this regurgitation is heard as a dull roar.

Gallop rhythm: a duplication "splitting" of the first or second heart sound, producing a triple instead of double sound. In humans this is associated with severe disease. A gallop rhythm is normal in dairy cows, particularly if HR is low.

Cardiac cycle in a dog

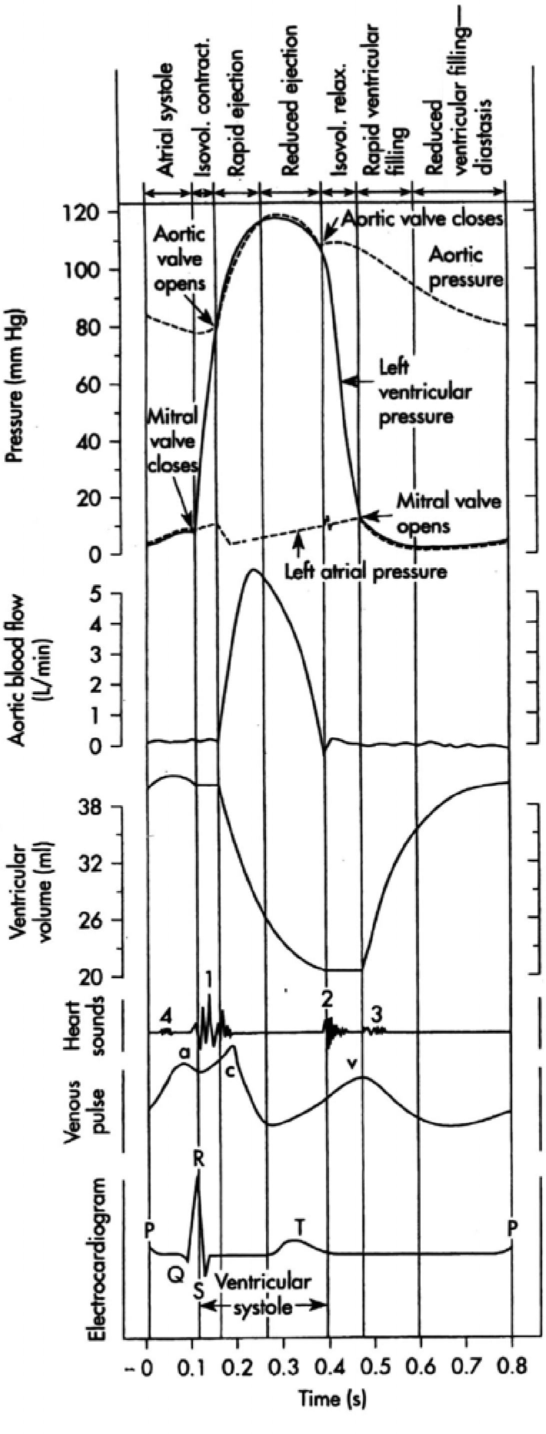

Figure 3-10 ■ Left atrial, aortic, and left ventricular pressure pulses correlated in time with aortic flow, ventricular volume, heart sounds, venous pulse, and the electrocardiogram for a complete cardiac cycle in the dog.

FURTHER READING

Klein BG, *Cunningham's Textbook of Veterinary Physiology,* 6th edition 2020.

Aaronson PI & Ward JPT, The Cardiovascular System at a Glance 4th Edition. 2014. Ch's 9-12 Berne RM & Levy MN, Cardiovascular Physiology, 6th Edition, 2008

Mohrman DE & Heller, Cardiovascular Physiology. McGraw Hill 9th Edition, 2018. eBook available in unimelb library.

Sherwood, L, Human Physiology from Cells to Systems, 8th Edition 2013.

Rhoades RA & Bell DR Medical *Physiology: Principles for Clinical Medicine* 5th Edition 2017