***Special examination of the cardiovascular system***

**Physical examination**

In the examination of animals with suspected heart disease especial attention should be give to:

The rate, rhythm and intensity of the individual heart sounds. And the rate, rhythm and amplitude of the arterial pulse.

Venous pulsation at the jugular inlet and the occurrence and timing of murmur.

**Heart sounds**

In the horse it is not uncommon to hear four heart sounds on auscultation.

The first heart sound: signals the onset of ventricular systole, is synchronous with the apex beat and temporally associated with closure of the mitral and tricuspid valves.

The area for maximal audibility of the mitral valve in the horse is on the left 5th intercostals space, at the level midway between horizontal line dawn through the point of the shoulder and one drawn at the sternum at the caudal edge of the triceps muscle.

With cattle, sheep, goats and swine, the sounds is located at a similar level but at the 4th intercostals space.

The area for maximal audibility of the tricuspid valve is on the right side of the chest slightly ventral to the equivalent level for the mitral valve and at the 4th intercostals space in the horse, and at the level of the costochondral junction at the 3rd intercostals space for the other species.

The second heart sound: is associated with aortic and pulmonic valve closure and is synchronous with the end of the systole and the beginning of cardiac diastole.

The aortic component is most audible just ventral to a horizontal line drawn through the point of the shoulder and in the left 4th intercostals space in the horse, and the left 3rd in the other species.

The pulmonic component is most audible ventral and anterior to the aortic valve area in the left 3rd intercostals space in horses and the left 2nd or 3rd intercostals space close to the costochondral junction in the other species.

Splitting of the second sound in the horse can be detected on phonocardiographic examination but is not usually detectable on auscultation.

The third heart sound: is associated with rapid filling of the ventricle in early diastole and is heart as a dull thudding sound occurring immediately after the second sound.

It is usually most audible on the left side just posterior to the area of maximal audibility of the first heart sound. However, it is frequently heard over the base and also over the area of cardiac auscultation on the right side, phonocardiographically there are two components to this sound but these are not usually detectable on clinical auscultation.

The fourth heart sound: is associated with atrial contraction .it is also called the ' a' sound .it occurs immediately before the first heart sound and is a soft sound most audible over the base of the heart on the left and right hand side.

The sequence of occurrence of he sounds is thus 4, 1, 2, and 3.

The intensity of the third and fourth sounds is less than that of the first and second and the complex can be described as du lubb dup boo.

The third and / or fourth sound may be in audible so that 1-2, 4-1-2, and 1-2-3 variations occur.

The name Gallop rhythm is frequently applied when these extra sounds occur. Gallop rhythm also occurs in cattle and may be due to the occurrence of a fourth or third sound or to true splitting the components of the first heart sound.

In sheep, goats and pigs only two heart sounds are normally heard.

The occurrence of third or fourth heart sounds in horses and cattle is not an indication of cardiovascular abnormality as is in other species.

**Variation in heart sound intensity:**

A decrease: a decrease in the intensity of heart sounds generation occurs in disease where there is poor venous return, Decreased strength of cardiac contractibility such as in terminal heart failure, in hypocalcemia in cattle, or peripheral vascular failure in all species.

Increase in heart sounds intensity with cardiac hypertrophy and metabolic diseases such as hypomagnesemia.

The intensity of the first heart sounds is increased by exercise, fear and excitement.

Muffling: muffling of the heart sounds suggests an increase in tissue and tissues interfaces between the heart and the stethoscope. This can be due to a shift in the heart due to displacement by a mass changes in the pericardium (increased fluid or fibrous tissues), changes in the pleural space or increased subcutaneous fat.

**Heart rate**

The relative temporal occurrence, and the intensity of the third and fourth heart sound, changes with heart rates. At moderately elevated heart rates the third heart sounds becomes more audible. At faster heart rates the third sound may merge and sum with fourth sound or the fourth sound may merge with the first sound if the P-R interval decreased.

Variations in the intensity of the individual heart sounds or complete absence of some of them can occur in conductions disturbances and arrhythmic heart disease and can provide valuable clinical information.

**Examination of the arterial pulse**

In arrhythmic heart disease the arterial pulse should be examined in more detail than that applied during routine clinical examination.

The pulse rate should be examined over a period to determine if there is any sudden change in rate such as can occur with a shift in pacemaker to an irritable myocardial focus.

At some stage during the examination of animals with tachyarrhythmia the heart rate and pulse rate should be taken synchronously to determine the presence of a pulse deficit. A convenient artery for this purpose is located on the posterior medial aspect of the carpus in the horse and cow.

Pulse rhythm is carefully examined. In conditions such as atrial fibrillation where there is no regular pacemaker it is not possible to establish any basic rhythm.

Amplitude of the pulse should also be carefully examined. Variation in pulse amplitude is associated with those arrhythmias that produce a variation in diastolic filling period within the heart. The extreme of this is a pulse deficit.

**Examination of the Jugular vein:**

In the normal adult horse and cow, the jugular vein will be distended with blood some 5-8 cm above the level of the base of the heart when the animal is standing with its head in a normal position.

There is a rapid but minor fall in the level of jugular distension associated with he fall of blood into the ventricle during the period of rapid filling during ventricular diastole followed by a slower rise un the level of jugular filling to its original point.

Superimposed on this and immediately preceding the fall , is a small wave or retrograde distension associated with a trial contraction ('a' wave) and a second smaller retrograde wave ('c' wave) associated with bulging of the atrial ventricular valves into the atrium during ventricular systole.

These pulsations can be observed in most horses and cattle by careful observation of the jugular vein at its entrance into the thorax and can be timed in conjunction with auscultation of the heart.

Observation of the presence or absence of the atrial 'a' wave is an aid in the clinical differentiation of first and second degree heart block.

Cannon atrial waves occur periodically incomplete heart block when atrial contraction occurs against a closed atrioventricular valve.

An accentuated 'c' wave occurs with tricuspid valve insufficiency.

***Electrocardiography***

The electrocardiography (ECG) provides a record and measure of the varying potential; difference that occur over the surface of the body as the results of electrical activity within the heart. This is associated with depolarization and repolarization of the myocardium.

At any one instant during depolarization and repolarization there are generally several fronts of electrical activity within the heart. At the body surface the potential difference is generally the sum of the activity and at any one instant the electrical activity in his heart registers as single dipole vector that has polarity, magnitude and direction.

The polarity is determined by the charge on the surface of the cells while the magnitude and direction ids determined by the mass of muscle being depolarized or repolarized and the sum of the instaneous vectors. Thus a wave of depolarization or repolarization over a muscle mass such as the atria or the ventricles is presented at the body surface as a sequence of instantaneous vectors with changing magnitude and direction.

The electrocardiograph can be considered as a voltmeter consisting of two input terminals, an amplifier to allow the recording of low input signals and a galvanometer with an attached recording device, such as a heated stylus on heat sensitive paper or an ink pen or ink squirter.

When a potential difference exists across the input terminals (electrodes) current flows through the coils of the electromagnet suspended between the poles of the permanent magnet to cause a deflection of the recording pen.

The electrocardiograph can therefore detect the polarity of the cardiac electrical vectors and by calibration of the machine and appropriate placement of electrodes on the body surface it can detect their magnitude and direction.

Calibration of most electrocardiographs is such that an input of 1 mv produces a 1 cm deflection of the recording pen.

Recording speeds are generally 25 or 50 mm/second.

In recording an ECG, certain standard electrode positions are used for recording.

A lead is the recording or circuit between two recording points .depending upon the wiring within the electrocardiograph the same potential difference a cross a lead could result in an upward or downward deflection of the recording pen.

In order to allow standard recording and comparison between recordings the polarity of the electrodes for standard leads has been established by convention and the leads are always recorded at these polarities.

The electrodes of a lead are commonly called positive or negative.

Appositive electrode in a lead is one which when electrically positive relative to other, due to a potential difference between them, yields an upward or positive deflection, of the recording pen.

**Depolarization and repolarization**

In the normal heart, depolarization and repolarization of the myocardium occurs in a definite pattern and sequence and the electrocardiography can be used to measure and time these events. Thus discharge of the sinoatrial node results in a wave of f depolarization over the atria to produce P wave in the ECG, the delay in conduction at the AV node is registered by no electrical activity at the body surface and an isoelectrical P-R interval on the ECG.

Depolarization of the ventricles occurs with several sequential fronts to produce QRS complex which ids followed by another isoelectric period before repolarization represented by T wave.

The order of ventricular activation in horses, cattle, sheep and swine differs from that of man and dogs in that ventricular depolarization is represented by only two fronts of activity.

Depolarization of a large proportion of the myocardium mass in large animals is not recognized by the surface electrocardiogram due to the fact that the purkinge fibers penetrate much more deeply in these species and depolarization occurs over multiple minor fronts that tend to cancel out , rather than a are single front as in dogs . For this reason, the detection of cardiac hypertrophy and myocardial abnormality by vector analysis of the electrocardiogram is, in general, not possible in large animals.

**Lead system**

Traditional lead systems are based on Einthoven's triangle as used in man and the standard bipolar limb leads (I,II,III0 and the augmented unipolar limb leads (aVR,aVL ,aVF).are commonly used un conjunction with an exploring unipolar chest lead,.

Variations in the position of the feet may produce changes in ECG wave forms with this lead system and recordings should be taken with the animal standing square or with the left front foot set slightly in advance of the right front foot.

This lead system is quite satisfactory for the detection of conduction disturbances and arrhythmic heart disease but is subject to movement artifact.

**Vector- Based lead systems**

The standard limb leads are primarily influenced by vectors in the frontal plane (Longitudinal and transverse) where as early and late forces in the myocardium are significantly directed in the vertical direction. Furthermore the heart is not electrically equidistant from the electrodes of each lead and distortion of recorded vector loops can result.

A partial correction of these deficiencies can be made by recording as lead using an exploring electrode at the V 10 position over the dorsal spinous processes in addition to the standard limb leads.

Bipolar and Base Apex lead system

Are most commonly used as they record the major electrical forces in the heart of large animals with best amplitude wave forms?

The bipolar lead system and its placement is determined as one that is least affected by movement of the animal.

The most commonly used unipolar lead placement in horses and cattle consist of two electrodes, one positive and one negative.

The Y lead system attaches the left arm lead (positive electrode) at the xiphoid about 8-10cm behind the girth and the right arm lead (negative electrode) in front of the chest at the thoracic inlet.

Lead one on the standard electrocardiograph is used for recording

An alternate and common lead system is the base – apex monitor lead

The right arm electrode is attached two – third of the way down the neck in the jugular furrow and the left arm electrode is placed over the apex of the heart just behind the left elbow. `

Again lead one is used for recording.

With both systems, the ground electrode is placed remote from the heart, usually over the withers.

With sheep, where wool interferes with placement on the neck the negative electrode can be placed on the midline of the poll.

**Other uses of the electrocardiogram**

Changes in the electrocardiogram occur with some electrolyte imbalance in large animal species.

There is a linear correlation between Q-T interval and plasma calcium in cattle, with elongation of the interval in hypocalcemia and shortening in hypercalcemia states.

Decreased amplitude and flattening of the P wave. Widening of the QRS complex and increase amplitude of the T wave is seen with hyperkalemia.

***Phonocardiography***

Phonocardiography allows the recording and measurements of heart sounds.

A special microphone is placed over the various ausculatory areas of the heart and the heart sounds are recorded graphically on moving paper or on an oscilloscope.

Phonocardiograms are usually recorded in conjunction with an electrocardiogram which allows timing of their occurrence in relationship to the electrical activity within the heart. Phonocardiograms can provide considerable information on heart sounds additional to that acquired by stethoscopic examination. In the horse, up to 11 sound events can be detected in each cardiac cycle.

In cardiovascular disease, phonocardiograms arte primarily used for the characterization and timing of murmurs, especially at fast heart rates where simple stethoscopic examination may not allow this.

In conjunction with an electrocardiogram, the phonocardiogram can be used to measure systolic time intervals which may be altered in congenital and acquired cardiovascular abnormalities.

***Exercise tolerance***

Dyspnea, fatigue and a prolonged elevation in heart rate following exercise are signs suggestive of cardiac insufficiency. Frequently animals with suspect cardiac disease are exercised in an attempt to elicit these signs and to get an estimate of exercise tolerance.

There is obviously a considerable difference in the amo8nt of exercise that a beef bull or trained racehorse can tolerate under normal conditions, and the amount of exercise given to any one animal is determined by the clinician's judgment.

The rate of fall in heart rate following exercise and the time required to reach resting levels depends upon the severity of the exercise even in fit horses.

Heart rate falls rapidly over the first minute and then more slowly over the ensuing 10-15 minute period.

**Cardiac output**

The one almost universally applied in large animal is the indicator dilution technique using thermo dilution or indicator dyes such as Evan's blue or indocyanine green. With dye dilution, an exact amount of dye is injected into the jugular vein or pulmonary artery via a catheter and the serial collection of blood samples is from a suitable proximally located artery.

Cardiac out put can be calculated from a dye dilution curve by determining the mean concentration of the dye and the times taken for one circulation through the heart. Cardiac output is expressed as liters per minute (L/ min.), and is usually corrected to cardiac index on the basis of weight or body surface area. The cardiac index for horses, sheep, and cattle at rest has been determined as 86 +13, 131+39 and 113 + 11 ml / min .per kg respectively.

**Measurement of arterial blood pressure**

Blood pressure may be determined directly by arterial puncture and pressure measurement but this is impractical in clinical cases.

For adult horse, the cuff is applied snuggly to the base of the tail and pulse sounds are detected in the ventral; coccygeal artery. On deflation of the cuff the pressure at emergence of the first sound is read as the systolic pressure.

Diastolic pressure can be identified by the emergence of a distinct second sound. Systolic and diastolic blood pressures of a large series of trained, thorough breed horses were 112+ 16 mm Hg (14.9+ 2.1 kpa) and 77+ 14 mmHg (10.2+1.9kpa).

Hypertension has been found in association with epistaxis, laminitis in horses and with painful fracture of the distal bones of the limb. Systolic blood pressure is often also elevated in obstruction of the large intestine in horses.

Blood pressure measurements are of values in the assessment of the degree of shock and possibly may prove of value in the differential diagnosis of conditions such as colitis –X, an acute salmonellosis and in assessing the prognosis of colic. Blood pressure readings can be obtained by equivalent techniques from the tails of cattle.

**Arrhythmias / dysrhythmias**

Variations in cardiac rate and rhythm include tachycardia (increase rate), bradycardia (Decrease rate), arrhythmia or dysrhythm (Irregularity in rate and rhythm) and gallop rhythm.

The rate and rhythm of the heart is influenced by

- The integrity of the pacemaker.

- The conducting system and the myocardium

- The influence of the autonomic nervous system.

Variation in the rate and rhythm can occur in normal animals due to strong or varying autonomic influence but can also be a reflection of primary myocardial disease .other factors such as electrolyte imbalance can influence rate and rhythm .the majority of arrhythmias and condition disturbances can be detected on clinical examination.

**Sinus tachycardia / simple tachycardia**

Is used to describe an increase in heart rate caused by detectable influences such as pain , excitement , exercise , hyperthermia a fall in arterial blood pressure or the administration of adrenergic drugs .in resting horses and cattle heart beat are not usually elevated above 48 , and 80 beats / min , respectively and rate above this are usually classified as tachycardia . In the cow and horse it is rare for the causes of sinus tachycardia to elevate the heart rate above 120 beats / min in the resting animal.

**Sinus bradycardia / simple bradycardia**

Is a normal heart action at a decrease rate due to a decrease rate of discharge from the sinoatrial node .it is most commonly associated with highly trained , fit animals .

Bradycardia may also occur in association with an increase in arterial blood pressure, space- occupying lesions of the cranium and increased intracranial pressure, pituitary abscess, hypothermia and hypoglycemia and following administration such as xylazine or detomidine.

***Diseases of the cardiovascular system***

**Introduction**

**Principles of circulatory failure**

The primary function of the cardiovascular system is to maintain the circulation of the blood so that normal exchanges of fluid, electrolytes, oxygen, and other nutrient and excretory substances can be made between the vascular system and tissues.

Failure of the circulation in any degree, interferes with these exchange s and is the basic for circulatory failure,

The two functional units of the system are the heart and the blood vessels and either may fail independently of the other, giving rise to two forms of circulatory failure – heart failure and peripheral failure.

In heart failure the in adequacy is due to involvement of the heart itself, in peripheral circulatory failure the deficiency is in the vascular system which fails to return the blood to the heart.

Circulatory failure can also result where there is a decreased circulatory volume.

**Heart failure**

The failure of the heart as a pump can result from a defect in filling of the heart, an abnormality in the myocardium or conducting system, an exercise work-load, or a combination of any of the three.

The two criteria used in assessing cardiac efficiency are the maintenance of circulatory equilibrium and the maintenance of the nutritional requirements of tissues .the maintenance of oxygen requirements is most important, the nervous system in particular being very susceptible to depolarization of oxygen.

It is usual to subdivided heart failure into two types, acute heart failure and congestive heart failure.

Circulatory equilibrium is not maintained when cardiac output is deficient, if this develops sufficiently slowly, compensatory mechanisms, plus the failure of the heart itself as a pump, result in an increase in venous pressure and congestive heart failure.

If there is an acute reduction of cardiac output, as is caused by sudden cessation of the heart beat, the effect is to deprive tissues of their oxygen supplies and the syndrome of acute heart failure develops.

**Peripheral circulatory failure**

In peripheral circulatory failure the effective blood volume ids decreased because of loss of fluid from the vascular system or by pooling of blood in peripheral vessels.

The failure of venous return results in incomplete filling of the heart and a reduction in its minute volume, although there is no primary defect in cardiac ejection.

The effects are the same as those of congestive heart failure in that the supply of nutrients and oxygen to tissues is reduced.

**Cardiac reserve and compensatory mechanisms in heart failure**

The normal heart has the capacity to increase its output several – fold in response to normal physiological demands created by exercise and to a less extent by pregnancy, by lasctati0n and by digestion .collectively, these responses comprise the cardiac reserve.

Similar response is utilized by the failing heart in an attempt to maintain cardiac output.

The major mechanisms whereby cardiac output can be increased and circulatory efficiency improved are:

Increase in heart rate

Increase in stroke volume

Increased extraction of oxygen from the blood.

Redistribution of blood to vital organs, or organs with particularly high requirements at the time.

**Cardiac reserve and hear rate**

Elevation of heart rate alone is a significant factor in increasing cardiac output in the exercising horse .there is a limitation to heart rate reserve because with increasing heart rates there is a decrease in diastolic filling time, and stroke volume falls at excessive heart rates.

**Cardiac reserve and stroke volume**

Stroke volume is variable and depends upon the amount of shortening that the myocardial fibers can attain when working against arterial pressure. It is determined by interplay of three factors.

* Ventricular distending or filling pressure (Preload).
* Contractility of the myocardium (Inotropic state).
* The tension that the ventricular myocardium must develop during contraction (after load).

An increase in ventricular distending pressure (end- diastolic pressure or volume) will increase ventricular end diastolic fiber length which, by the Frank – starling mechanism, will result in increased stroke work and a large stroke volume.

Ventricular distending pressure is influenced by atrial contraction and is greatly engmented by increased venous return associated with exercise and increased sympathetic activity.

**Cardiac reserve and oxygen tension**

In normal animals, at rest, the oxygen tension of mixed venous blood is above 40 mmHg which represents a considerable reserve. Increased extraction of oxygen from the blood by various tissues, with a subsequent increase in arterial venous oxygen difference, occurs during exercise and also in states of cardiac insufficiency.

In uncompensated heart failure where stroke volume is reduced the arterial venous oxygen difference is large.

There is also a redistribution of blood flow to vital organs,

In the horse the spleenic storage capacity for erythrocytes is large and the spleen may contain one third of the total red cell volume.

**Cardiac reserve in cardiac insufficiency**

In cardiac insufficiency the principal defect is in the contractile state of the myocardium, and ventricular performance at any given end- diastolic volume or pressure is diminished.

In early failure, cardiac output may a still be maintained in the normal range by an increase in filling pressure, and through utilization of the Frank-starling principle, the ventricles can eject a normal stroke volume despite the depression in contractility.

**Measurement of cardiac reserve**

**Clinical estimations:**

A clinical estimation of cardiac reserve based on physical examination is important when a prognosis is to be made on an animal with heart disease.

Some of the important criteria used in making this assessment include the heart rate, the intensity of its sounds, the size of the heart, the characters of the pulse, and the tolerance to exercise.

A resting heart rate above normal indicates loss of cardiac reserve because the ability to raise the minute volume of the heart is thereby reduced.

The absolute intensity of the heart sounds suggests the strength of the ventricular contraction, soft sounds suggesting weak contractions, and sounds which are louder than normal suggesting cardiac dilatation and possibly hypertrophy. Exercise tolerance is a good guide to cardiac reserve.

It is best measure by estimation of the maximum heart rate attained after a standard exercise test, and the speed with which the heart rate returns to normal.

***Manifestations of circulatory failure***

**Congestive heart failure**

**Etiology**

The broad categories of causes of congestive heart failure are as follows:

**Valvular disease**

Endocarditis resulting in either valvular stenosis or valvular insufficiency

Congenital valvular defects – most commonly valvular stenosis.

Rupture of valve or valve chordate

**Myocardial disease:**

* Myocarditis – bacterial , viral , or parasitic
* Myocardial degeneration – toxic or nutritional
* Congenital and hereditary cardio- myopathy
* Chemicals affecting cardiac conduction

**Pericardial disease**

-pericarditis

-pericardial tamponade

Hypertension

-Pulmonary hypertension – high attitude disease, corpulmonale

- Systemic hypertension – rare cause of heart failure in large animals

**Congenital defects producing shunts**

-defects of myocardium, such as septal defects.

- Vascular abnormalities.

**Pressure load**

Pressure loads occur with lesions that produce an obstruction to outflow an as aortic or pulmonary valve stenosis where the heart is required to perform more work to eject an equivalent amount of blood.

Pressure loads are not necessarily associated with lesions in the heart. For example, pulmonary hypertension a as occurs in high altitude disease of cattle due to an increase in pulmonary vascular resistance, may result in cardiac insufficiency.

In general, the left ventricle can tolerate a pressure load to a much greater extent, without overt signs of cardiac insufficiency than the right ventricle.

**Volume load**

Volume load (flow loads) occur commonly with both acquired and congenital heart defects. In both aortic valve insufficiency and mitral valve insufficiency the volume of blood delivered to the body tissues does not differ significantly from normal. However, in order to achieve this, the stroke volume of the ventricle is markedly increased and the heart is much more inefficient for the same amount of effective work.

In a similar manner a patient ductus arteriosus or an interventricular septal defect with a large shunt of blood can place a considerable flow load on the left ventricle.

In general, the right ventricle is more capable of sustaining a flow load than the left ventricle.

Diseases of the endocardium, myocardium and pericardium, that interfere with the flow of blood into or away from the heart or that impair myocardial function, may result in congestive heart failure.

**Failure of the heart as a pump (systolic failure)**

Cardiac insufficiency may occur without any increase in workload if there is a primary weakness in the myocardium or defect in its rhythmic and coordinated contraction.

Myocaditis, cardiopathy, and neoplasm of the heart, especially bovine viral leucosis of the right atrium, are the common cause.

Filling defects (diastolic failure).

Pericardial disease such as pericarditis and cardiac tamponade can result in cardiac insufficiency through interference with diastolic filling.

**Clinical findings**

Generalized venous congestion and edema in right – sided failure. Pulmonary congestion and respiratory distress in left – sided failure.

**Treatment**

Cardiac glycosides and diuretics

Treatment of specific cause.

***Acute heart failure***

**Etiology**

Acuter heart failure can occur when there is a severe defect in filling, when there is failure of the hear as a pump, either due to severe tachycardia or bradycardia, and where there is a sudden increase in workload. The sudden occurrence of tachy arrhythmias in association with excitement and severe enough to cause acute heart failure presumably results from the exacerbating influence of catecholamines.

Acute heart failure can also occur in the absence of primary cardiac disease in the many causes of acute heart failure include

**Disorders of filling**

- Pericardiac tamponade – atrial and ventricular rupture.

- Aortic and pulmonary artery rupture

**Tachy arrhythmia**

- myocarditis e.g., encephalomyocarditis virus, foot and mouth disease.

- nutritional deficiency myopathy e.g. copper or selenium deficiency

- Plant poisoning e.g. phalaris spp.

= electrocution and lightning strike.

**Brady cardia**

- latrogenic e.g. intravenous calcium preparation, xylazine, concentrated solutions of potassium chloride

- plant poisoning e.g. taxus spp.

- Increase in work load

- Rupture of aortic valve

- Acute anaphylaxis

In the horse, arrhythmias and cardiac arrest may occur during the induction of anesthesia with barbiturates and may occur without premonitory signs in horse under halothane anesthesia.

**Pathogenesis**

With excessive tachycardia the diastolic period is so short that filling of the ventricles is impaired and cardiac output is greatly reduced.

In ventricular fibrillation no coordinated contractions occur and no blood is ejected from the heart.

The cardiac output is also seriously reduced when the heart rate slows to beyond a critical point.

In all of this circumstance there is a precipitate fall in cardiac output and a severe degree of tissue anoxia.

In per acute cases the most sensitive organ, the brain, is affected first and the clinical signs are principally neurological.

Pallor is also a prominent sign in this case because of the reduction in arterial blood flow.

In less acute cases respiratory distress is more obvious because of pulmonary edema and although these can be classified as acute heart failure they are more accurately described as acute congestive heart failure.

**Clinical findings**

The acute syndrome may occur while the animal is rest, but commonly occurs during periods of excitement or activity.

The animal usually shows dyspnea, staggering, and falling, and death often follows within seconds or minutes of the first appearance of signs.

There is marked pallor of the mucosae. Although clonic convulsions may occur hey are never severe and consist mainly of sporadic incoordinate movements of the limbs.

Death usually is preceded by deep, asphyxia gasps.

If there is time for physical examination, absence of a palpable pulse and Brady cardia, tachycardia or absence of heart sounds are observed.

Horses with rupture of the aortic valve rupture of mitral valve chordate sudden onset of atrial fibrillation or multiple ventricular extra systoles show a syndrome where sudden onset of respiratory distress is the prominent manifestation.

**Clinical pathology**

In general there is insufficient time available in which to conduct laboratory tests before the animals dies.

**Necropsy findings**

In typical acute cases engorgement of visceral veins may be present if the attack has lasted for few minutes but there may be no gross lesions characteristic of acute heart failure.

**Differential diagnosis**

Acute heart failure should be a major consideration as a cause of sudden death in large animals, especially when death is associated with exertion or excitement

Acute heart failure may be mistaken for primary disease of the nervous system but is characterize by excessive Brady cardia or tachycardia, pallor of mucosae, absence of the pulse, and the mildness of the convulsions.

Epilepsy and narcolepsy are usually transient and repetitive and have a characteristic pattern of development.

**Treatment**

Is not practicable in large animals because of the short course of the disease.

Deaths due to sudden cardiac arrest or ventricular fibrillation while under anesthesia can be avoided to a limited extent in animal by direct cardiac massage or electrical stimulation.

***Peripheral circulatory failure and shock***

**Etiology**

Peripheral circulatory failure occurs when the cardiac output is reduced because of the failure of venous return to the heart.

**Hypovolemic shock**

Occurs when there is a reduction in circulating blood volume due to blood or fluid loss.

**Vasogenic failure**

Occurs when peripheral vasodilatation and pooling of blood in the vessel, and leakage into tissues, reduces the effective circulating volume.

**Septic or toxic shock**

Is a form of vasogenic failure with loss of vascular integrity and increased vascular permeability resulting from toxic and septic influences on blood vessels?

Regardless of the initiating cause, hypoperfusion is the outcome which leads to impairment of oxygen uptake and anaerobic metabolism in tissues. The result is a syndrome called sh0ck and irreversible shock develops as cellular dysfunction becomes so wide spread that the animal will die despite therapy that temporarily restores blood pressure and circulation.

Common precipitating causes of peripheral circulatory failure in large animals are as follows:

**Hypovolemic failure**

* Hemorrhage with loss of 35% or more of total blood volume.
* Fluid loss and dehydration, such as in colitis –X, neonatal calf diarrhea, or stress – induced dehydration, especially if fluid loss in severe and over a short period.
* Fluid loss into the intestine with acute intestinal obstruction, toxic shock is also a contributor.

**Distributive failure**

This form includes the vascular abnormalities that are also referred to as vasogenic shock.

**Examples of causes of distributive shock are:**

* Severe burn injury
* Following extensive surgery
* After prolapsed of uterus
* Too sudden reduction of pressure in a body cavity , e.g. by rapid with drawl of ascetic fluid
* Severe pain as in colic in horses
* Trauma with local sequestration of blood and fluid.

**Non toxic general vasogenic failure**

The most common cause is hypocalcemia, such as occurs in parturient paresis in dairy cattle and hypocalcemia in sheep. There is also a cardiogenic influence with hypocalcemia.

**Toxic and septic shock**

Toxic or septic shock can occur when endotoxin, other toxins, and microorganism, gain access to the circulation and act on the vascular system directly or via mediators released by their interaction with the host cells.

**Some examples of syndromes leading to toxic / septic shock are**

* Septicemic disease: septic shock from endotoxemia is responsible for the death of animals with Gram- negative bacteria sepsis.
* Septic and toxic infections – acute diffuse peritonitis, acute gangrenous mastitis, acute metritis, per acute coliform mastitis.
* Absorption of toxin from the intestine grain engorgement in horses and cattle, infarction of a large segment of intestinal wall in horses, acute intestinal accidents.

**Pathogenesis**

**Hypovolemic and vasogenic shock**

There are a number of interacting responses when cardiac output falls, the carotid and aortic baroreceptors stimulate the sympathetic nerves and adrenal medulla to release catecholamines resulting in vasoconstriction in vessels with alpha – adrenergic receptors.

The decreased effective blood volume leads to decreased renal perfusion which activates the rennin – angiotensin – aldosterone system inducing retention of sodium and water.

**Hypovolemia**

Also stimulates the release of antidiuretic hormone (vasopressin).

There is contraction of the spleen and venous capacitance vessels, an increased peripheral vascular resistance and an increase in heart rate, all in an attempts to maintain cardiac out put and blood perfusion through the coronary and cerebral blood vessels

**Water shifts**

From the interstitium to the vascular space. In the initial stages of hypovolemic failure the primary signs are those of interstitial fluid depletion and dehydration, with pink but dry mucous membranes, sunken eyes and decreased skin turgor.

Peripheral vasoconstriction in the face of continued hypovolemia and falling cardiac output results in a fall in blood pressure , in the opening or arteriovenous shunts and in decreased perfusion of organosystems , with resultant damage from hypoxia and tissues acidosis and the development of clinical signs of peripheral vascular failure and shock.

**Septic shock**

In normal animals the healthy intestinal mucosa is an effective barrier to the absorption of endotoxin that is present in the gut and the small amounts of endotoxin that are absorbed into the portal blood are cleared by the liver and do not reach the systemic circulation.

When the integrity of the intestine is compromised by factors such as ischemia, trauma or inflammation, sufficient endotoxin can be absorbed to overwhelm the clearance mechanism of the liver and endotoxin may also lack to the peritoneal cavity and so gain access to the systemic circulation.

This is important in the genesis of septic shock associated with acute intestinal accidents and some inflammatory gut disease. It also allows the contribution of endotoxin in the final stagers of most shock syndromes as intestinal mucosal integrity is lost due to tissue hypoxia.

Endotoxin and other toxins can also be absorbed from sites of local infection, as with acute – diffuse peritonitis and coliform mastitis, or can be released from bacteria in the blood system. Endotoxin and other bacterial toxins cause direct endothelial damage .they also activate macrophage and neutrophils provoking the release of a multitude of inflammatory mediators , including interleukins tumor necrosis factor , and platelet activating factor which lead to endothelial damage , leaky vessels , hypotension and vasculitis and eventually decreased intravascular volume .

**Inadequate tissue perfusion**

The end result of inadequate tissue perfusion is the development of multiple organ failure. Metabolic acidosis and lactic acidemia, the hypodynamic stage of shock.

Hypovolemia and poor tissue perfusion results in cold extremities, elevated heart rate, a weak thready pulse, decreased capillary refill times and altered mental status.

Cardiac arrhythmias may occur because of myocardial; ischemia and electrolyte and acid – base disturbance. There is anorexia and gastrointestinal stasis. Signs of renal failure include anuria or oligouruia and azotemia.

**Clinical findings**

Depression, weakness, and listlessness are accompanied by a fall in temperature to below normal. The skin is cold and skin turgor is decreased. The mucoase are pale gray and dry but severe blanching is not present unless hemorrhage is the initiating cause of peripheral failure. Capillary refill time is extended beyond 3-54 seconds. There is an increase in heart rate (120-140) beats per minute in both horses and cattle.

With abnormalities of the pulse including small amplitude, weak pressure, and an increased vessel tone, although this latter is increased in the terminal stages. The absolute intensity of the heart sounds is reduced and there may be cardiac arrhythmias. Venous blood pressure ids greatly reduced and the veins are difficult to rise.

The respiratory rate is increased and respirations are usually shallow. Anorexia is usual but thirst may be evident and there is anuria or oligouria.

Nervous signs include depression, listlessness and obtrusion, and coma in the terminal stages. Clonic convulsions may occur.

**Treatment**

**Identification of cause**

It is important in cases where circulatory failure is initiated by conditions that are amenable to surgical correction.

Fluid therapy: isotonic crystalloid solutions

**Colloids**

Intravenous administration of colloid solutions (dextran, gelatin polymers, hexastarch) induces ore sustained increase in plasma volume and smaller volume is required for therapy.

**Corticostereoids**

There is considerable controversy over the use of corticosteroids in shock.

Antibiotic therapy

***Diseases of the heart myocardial disease and cardiomyopathy***

**Etiology**

A number of diseases are accompanied by inflammation, necrosis, or degeneration of the myocardium.

These include several bacterial, viral, or parasitic infections and some nutritional deficiencies.

Disturbance of myocardial function is the primary action of some toxic agents in large animals.

In most cases, the involvement of myocardium is only part of the total spectrum of the disease, although the cardiac manifestation may be clinically pre- eminent.

The term cardiomyopathy is generally restricted to those diseases where myocardial damage is the prime manifestation.

**Causes of myocardial dysfunction include the following**

**Bacterial myocarditis**

* Following bacteria, as in strangle or from navel – ill.
* Tuberculosis – especially horses.
* Tick pyemia in lambs
* Clostridium chawvoei
* Haemophilus somnus
* Extension from pericarditis or endocarditis

**Viral myocarditis**

* African horse sickness
* Equine viral arterities
* Equine infectious anemia
* Equine herpes virus -1 in fetus
* Swine vesicular disease (SVD) .
* Parvovirus in piglets
* Encephalomyocarditis virus infection in pigs.
* PRRS virus in piglets.
* Blue tongue in sheep.

**Parasitic myocarditis**

* Strongylus spp. (migrating larvae). Cysticercosis, sarcocystis and neosporum casninum (in the neonatal calf).

**Nutritional deficiency**

* Vitamin E / selenium deficiency in all large animal species.
* Some forms of chronic copper deficiency in cattle ( falling disease )
* Iron deficiency in piglets and veal calves
* Copper / cobalt deficiency in lambs.

**Poisoning**

* Inorganic poison – selenium, arsenic, mercury, phosphorus, thalium.
* Gossypol from cotton seed cake
* Fuoroacetate (1080) and poisoning by A cacia Georgina, Gastrobolium, and oxyloium spp., Dicha petalum cymosum.
* Plants and weeds including members of Ixiolena , pachystigma , pavette , Asclepias ,Geriocarpa , cryptostigisa , Albizia , Cassia ,Digitalis , Urechites , Pimelea , Atragalus , Fadogia , Cicuta , Colchicum , Karwinskia , Vicia , Cicuta , Trigonella , Brtophyllum , Palicourea , lupines , Lantana , Kalanchoe , Homeria , Hymenoxys , Eupatorium spp.
* Trees including gidgee, yew, oleander, avocado.
* Grasses including phalaris tuberose, corynetoxins in lolium rigidum infested with nematodes and corynebacterium spp.
* Drugs including succinyl choline, catecholamnes, xylazine (ruminant). moneusin – especially in horses , but also cattle , sheep , and pigs – lasalocid and salinomycin in horses , pigs , cattle and sheep , maduramicoon in cattle and sheep fed poultry litter , and adriamycin .
* Overdosing with doxycycline in veal calves.
* Vitamin D and myocardial and endocardial calcification following ingestion of cestrum diumum, Solanm malacoxylon, Trisetum flavescens.
* Calcification also occurs with hypomagnesemia in milk fed calves.

Venoms

* Rattlesnake (Crotalus spp) venom in horses.
* Vipera palaestinae.

**Embolic infarction**

* Emboli from vegetative endocarditis or other embolic disease such as bracken fern poisoning in cattle.
* Tumor
* Viral leukosis of cattle.

**Inherited**

* Unknown or uncertain etiology.
* Myocardial necrosis and hemorrhage secondary to acute lesions in the central nervous system.
* Exertional rhabdomyolysis of horses capture myopathy of wild ruminants, restraint stress in swine.
* Sudden death in young calves associated with acute heart failure and myocardial necrosis and precipitated by periods of intense excitement such as that at feeding time.
* Myocardial lipofuscinosis (brown atrophy) in aged or cachectic cattle, especially Ayrshires.
* Myocardial disease following mild upper respiratory disease in horses.

**Pathogenesis**

The primary effect of any myocardial lesion is to reduce cardiac reserve and limit compensation in circulatory emergencies.

Most commonly, myocardial disease results in conduction disturbances and arrhythmias from primary involvement of the conduction system or establishment of excitatory foci within the myocardium.

While the animal is at rest there may be minimal evidence of cardiac disease but catastrophic disturbances in cardiac conduction mat occur under the adrenergic influences if exercise or excitement.

Endogenous or synthetic, catecholamines can produce multifocal myocardial necrosis, especially in left ventricles.

Myocardial disease may also result in congestive heart failure through its primary effects on the myocardium and the function of the heart as a pump.

**Clinical findings**

In early cases, or cases with mild or moderate myocardial damage, a decreased exercise tolerance, is the usual initial presenting sign.

This is usually accompanied by an increase in heart rate and heart size .dysrhythmias particularly tachyarrhythmias associated with multiple ventricular extrasystols.

In the late stages, or in cases with more severe myocardial damage, there may be sudden death or attacks of cardiac syncope due to acute heart failure, or severe dyspnea or general edema due to congestive heart failure.

**Clinical pathology**

Electrocardiography sand echocardiographies are used in special examination.

Hematological examination, blood culture and serology may be of value in determining the cause of myocardial disease.

Full biochemical profile in multi systemic problems.

Myocardial infarction and necrosis may be associated with the release of cell enzymes into the blood stream during the acute phase and the determination of the serum concentration of lactate dehydrogenase, creatinine kinase, and aspartate aminotransaminase are of value.

The cardio specific isoenzymes of lactate dehydrogenase and creatinine kinase should be of particular value in the biochemical examination of myocardial disease.

**Differential diagnosis**

* Congestive heart failure.
* Acute heart failure.
* Other causes of decreased exercise tolerance.

**Treatment**

* The primary cause must be treated.

***Valvular diseases and murmurs***

**Etiology**

**Acquired**

* Endocarditis
* Rupture of the chordate tendinae, either spontaneous or secondary to endocarditis.
* Laceration, detachment of aortic valve, leaflets, either spontaneous or secondary to endocarditis.
* Dilatation of the right atrioventricular valve annulus, such as occur in brisket disease and secondary to some myocardial disease, may result in functional insufficiency of the valves.

**Congenital**

* Pulmonary valve stenosis
* Fenestration of the aortic and pulmonary valves in horses
* Blood cysts are common on the atrio- ventricular valves of cattle.

**Pathogenesis**

The important clinical indications of valvular disease are audible murmurs and palpable pericardial thrills.

Murmurs mat occur at any phase of the cardiac cycle and are caused by the vibrations of turbulent flow of blood transmitted to the surface of chest.

**Generation of murmur**

Blood flow is normally laminar and without turbulence.

Turbulence in flow may be produced by a sudden change in the diameter of the vessel through which the blood is flowing.

Its occurrence is directly related to the velocity of flow and inversely related to blood viscosity.

**Valve lesions**

With murmurs associated with valvular lesions the valve lesion produces a sufficient change in stream bed diameter to result in turbulent flow.

The turbulence may occur when the valves do not close properly (regurgitation or insufficiency) and blood is force through atrioventricular orifices during ventricular systole or through semilunar orifices during ventricular diastole.

Turbulence may also occur when the valves do not open completely (stenosis) and blood is force through a stenotic semilunar orifice during ventricular systole or enters the ventricle through a narrow atrioventricular orifice during ventricular diastole.

**Murmurs without valvular disease:**

A change in vessel diameter such as occurs with dilatation of the aorta or pulmonary artery can produce turbulence and a murmur.

A reduces in blood viscosity contributes to the frequency of murmurs occurring in anemic and hypoproteinemic states and hemic murmurs are common in anemic cattle.

**Functional murmurs**

The cause of functional or ejection murmurs that occur commonly in horses during the rapid ejection phase even at rest and especially following exercise.

**Effects of valvular disease**

Stenosis of the outflow valves results in an increased pressure load on the heart and compensatory hypertrophy.

Insufficiency of the semilunar valves or of the aortic or pulmonic valve produces a volume load on the heart and is followed by compensatory dilatation, and hypertrophy.

If the valves on the left side of the heart are affected, especially the aortic valve, the changes in ejection of the blood from the ventricle produces changes in the character of the peripheral pulse.

Involvement of the tricuspid valve will produce changes in the jugular pulse.

**Cardiac reserve**

The presence of valvular lesions and murmurs lead to some degree of cardiac reserve is lost.

**Clinical findings**

When a murmur is detected it should be categorized according to its: Timing; Duration; Intensity; Location; and character.

**Timing: systolic and diastolic, and continuous murmurs.**

* Murmur should be timed with reference to the arterial pulse which occurs in early to mid systole.
* A less satisfactory alternative is timing with the occurrence of the apex beat.
* Timing by relation to the heart sounds.
* Systolic murmurs are associated with stenosis of the outflow valves or insufficiency of the atrioventricular valves.
* Diastolic murmurs are associated with insufficiency of the outflow valves or stenosis of the atriventricular valves.

**Continuous murmurs**

Or one that occurs during both systole and diastole may be associated with both stenosis and insufficiency of the same valve or with multiple valvular lesions but more commonly results from the turbulence flow of blood from a high pressure to a low pressure system with no intervening valve such as occurs with a patent ductus arteriosus.

**Duration**

**During systole or diastole**

Systolic murmurs are classified as early, late, holo- or pansystolic according to their occurrence and duration in the period between the first and second heart sound.

Diastolic murmurs as early (occurring between S2 and S3), holodiastolic, or presystolic (occurring between the atrial fourth heart sound and S1).

Pansystolic and diastolic murmurs occurring throughout the systole or diastole.

**Intensity or loaders**

Grade I, II, III, IV, and V.

**Character**: defined as crescendo, crescendo- decrescendo, decrescendo or plateau.

Or according to their frequency characteristics. By terms such as blowing, harsh, and musical sighing.

***Hypovolemic, hemorrhagic, maldistributive and obstructive shock***

**Etiology**

The circulatory system consists of a pump (the heart) and a circuit (the vasculature).

Circulatory shock can result from abnormal functioning of the pump or circuit, or both.

It is a clinically very important to differentiate pump failure (cardiogenic shock due to acute or chronic heart failure) from circuit failure, because the diagnosis and treatment of cardiogenic shock is vastly different to that of circuit shock.

Circuit failure occurs whenever the cardiac output is reduced below a critical point because of inadequate venous return to the heart.

There are four main ways that circuit failure occurs:

* Hypovolemic shock occurs when there is a reduction in circulating blood volume due to plasma or free water loss.
* Hemorrhagic shock: occurs when there is a reduction in circulating blood volume due to rapid blood loss.
* Maldistributive shock. occurs when there is a reduction in circulating blood volume due to increased capillary permeability , pooling of blood in capacitance vessels ( such as the veins in the splanchnic circulation ) .or pooling of plasma in a large third space such as the thoracic or abdominal cavities .
* Obstructive shock: occurs when there is an acute reduction in venous return due to a mechanical obstruction, such as pericardial, tamponade in pulmonary artery thrombosis.
* Obstructive shock is extremely rare in large animals.

Regardless of the initiating cause or circuit failure and in adequate venous return, tissue hypoperfusion results, leading to impaired oxygen uptake and anaerobic metabolism.

The end result of inadequate tissue perfusion is the development of multiple organ failure, l. lactate academia and strong ion metabolism). Acidosis manifested as the hypodynamic stage of shock.

**Hypovolemias and poor tissue perfusion results in:**

* Cold extremities.
* Elevated heart rate.
* A weak thread pulsed.
* Decreased capillary refill times
* And altered mental status
* Cardiac arrhythmias may occur because of myocardial ischemia and electrolyte and acid – base disturbance.

There is anorexia and gastrointestinal stasis .signs of renal failure includes anuria or oligouria and azotemias.

Common causes of circuit failure in large animals are:

**Hypovolemic shock**

* Fluid loss and dehydration such as in neonatal calf diarrhea and burn injury especially when fluid loss is severe and rapid.
* Fluid loss into the gastrointestinal tract due to acute intestinal obstruction.

**Hemorrhagic shock**

Acute hemorrhage with loss of 35% or more of total blood volume, equivalent to an acute blood loss of 2.8% of body weight will lead to clinical signs of severe hemorrhagic shock.

Traumatic injury or spontaneous ruptures of large blood vessels are the common reasons for acute hemorrhage.

Some of the common causes of hemorrhagic shock are:

**Cattle and sheep**

* Spontaneous pulmonary hemorrhage associated with caudal vena caval syndrome.
* Abomasal ulcer, sometimes originating from a bovine viral leukosis lesion (cattle).
* Enzootic hematurtia with bleeding from a bladder lesion (Cattle).
* Pyelonephritis with bleeding from a renal lesion (Cattle).
* Intra- abdominal hemorrhage as a result of arterial aneurysm possibly associated with copper deficiency (Cattle).
* Laceration of arteries in the wall of the vagina as a result of dystocia.
* Ruptured middle uterine artery during prolapsed or torsion of uterus.
* Cardiac tamponade due to rupture of coronary artery or ventricular chamber, rupture of aorta.
* Rupture of liver associated with dystocia in lambs, and in older lambs possibly associated with vitamin E deficiency.

**Horses**

* Ethemodial hematoma.
* Exercise – induced pulmonary hemorrhage
* Rupture of the middle uterine, uteroovarian (especially – right side) or iliac artery associated with parturition, more commonly in aged mares.
* Nasal bleeding from hemorrhage into the guttural pouch. From carotid or maxillary arteries with guttural pouch mycosis or associated with rupture of the longus capitis muscle following trauma.
* Rupture of mesenteric arteries secondary to strongle larval migration.
* Splenic hematoma or rupture following blunt trauma.
* Rupture of liver with hyperlipemoia.
* Hemangioma, hemengiosarcoma and other neoplasia.
* Persistent bleeding from the valve in association with ulcerated varicose veinsa on the dorsal wall of the vagina.
* Congenital venous aneurysm (rare).

**Maldistributive shock.**

* Endotoxemia in neonatal septicemia, salmonellosis, coliform mastitis in lactating dairy cattle, toxic metritis in cattle.
* Septic shock due to Gram – positive bacterial septicemia.
* Too sudden reduction of pressure in a body cavity, e.g. by rapid with drawl of ascetic fluid.

**Obstructive shock**

* Pericardial tamponade.

**Pathogenesis**

**Hypovolemic shock**

When cardiac output falls as a result of decreased venous return, the carotid and aortic baroreceptors stimulate the sympathetic nerves and adrenal medulla in release catecholamines resulting in vasoconstriction in vessels with alpha adrenergic receptors.

Vasoconstriction leads to decreased renal perfusion, which activates the rennin – angiotensin – aldosterone system, thereby inducing sodium and water retention.

The decreased in renal perfusion can lead (result) in renal ischemia and nephrosis if the ischemia is sufficiently severe and prolonged.

Hypovolemia also stimulates the release of antidiuretic hormone (vasopressin).

There is contraction of the spleen and venous capacitance vessels, an increased peripheral vascular resistance and an increase in heart rate in an attempt to maintain cardiac output and blood perfusion through the coronary and cerebral blood vessels.

**Hemorrhagic shock**

The major effects of hemorrhage are loss of blood volume (hypovolemic shock), loss of plasma protein (decreased plasma oncotic pressure) and loss of erythrocytes (decreased oxygen – carrying capacity).

With acute and severe hemorrhage, the rapid loss of blood volume results in hypovolemic shock and the loss of erythrocytes in anemic anoxia.

The combination of these two factors is tend hemorrhagic shock and is often fatal.

With less severe hemorrhage , the normal compensatory mechanisms including release of blood stored in the spleen and liver and the withdrawal of fluid from the tissue spaces , may maintain a sufficient circulating blood volume , but the anemia is not relieved that the oncotic pressure of the blood is reduced by dilution of residual plasma protein.

The resulting anemia and edema are repaired with time provided the blood loss is halted.

**Maldistribution shock**

In normal animals the healthy intestinal mucosa is an eff3ecvtive barrier to the absorption of endotoxin that is present in the gut and the small amounts of endotoxin that are absorbed into the portal blood are clear by the liver and do not reach the systemic circulation .

When the integrity of the intestine is compromised by factors such as ischemias, trauma, or inflammation, sufficient endotoxin can be absorbed to overwhelm, the clearance mechanisms of the liver, and endotoxin may also leak to the peritoneal cavity and thereby gain access to the systemic circulation.

Endotoxin can also be absorbed from sites of local infection, as with diffuse peritonitis, coliform mastitis, and toxic metritis or released from Gram – negative bacteria in the blood stream.

Intestinal mucosal integrity is lost in the terminal stages of circulatory shock due to tissue hypoxia, and endotoxin translocation from the intestinal tract is markedly increased in the terminal stages of shock.

Endotoxins and other bacterial toxins cause direct endothelial damage.

Endotoxins also activates macrophages and neutrophils provoking the release of multitude of inflammatory mediators , including TNE , interleukin – 1 , interleukin – 6 and platelet activating factor , which lead to endothelial damage , leaky vessels , hypotension and vasculitis and eventually decreased intravascular volume .

**Obstructive shock**

In severe pericardial tamponade, the rapid increase in pericardial fluid volume impedes diastolic filling of the heart and therefore results UN decreased cardiac out.

A similar response occurs in advanced traumatic reticulopericarditis in cattle that have ingested a wire.

**Clinical findings**

Depression, weakness, and listlessness are accompanied by a fall in temperature to below normal.

The skin is cold and skin turgor is decreased.

The mucosae are pale gray to white and dry and capillary refill time is extended beyond 3-4 seconds.

There is an increase in heart rate to 120-140 beats / min. in horses and cattle with abnormalities of the pulse including small and weak pressure amplitudes ( a thready pulse ) ;.

Cardiac arrhythmias are present terminally.

Venous blood pressure is greatly reduced in hypovolemic and hemorrhagic shock and the veins are difficult to rise.

Arterial blood pressure is decreased terminally and fails to produce early indicators of the severity of the circulatory failure.

Anorexia is usual but thirst may be evident and there is anuria or oligiuria.

Nervous signs include depression, listlessness and obtrusion, and comas in the terminal stages.

During the early hyper dynamic stage of maldistributive shock the temperature is normal or elevated; mucous membranes are injected and brick – red in color. There is tachycardia but normal capillary refill time, and the extremities (particularly the ears) are cool to tough. Whereas these signs are not specific for shock they can allow the early instituation of therapy.

**Clinical pathology**

Examinations of the blood for hematocrit and plasma protein concentration are valuable indicating the magnitude of the blood loss in hemorrhagic shock to the progress of the disease.

Abdomencentesis, thoracocentesis and ultrasound are used to identify sites of intestinal bleeding.

**Treatment**

The rapid administration of intravenous fluid in hypovolemic and maldistributive shok.

Isotonic crystalloid solutions balanced electrolyte solutions, such as lactated Ringers solution, are preferable to 0.9% sodium chloride solutions. 50 ml / kg is probably the minimum.

Hypertonic saline solution in fluid therapy of hypovolemic shock.

Small volume ( 4.5 ml /kg ) of hypertonic saline ( 7.2% 2400 mosmol /L) are infused I.V. over 4-5 min.

Colloids

The I.V. administration of colloid solutions (dextran, gelatin polymers, hexastarch) induces a more sustained increase in plasmas volume than crystalloid solutions and small volumes are required for therapy.

Hemorrhagic shock

Whole blood transfusion, hypertonic saline solution is recommended in the initial treatment of hemorrhagic shock.

Ancillary treatment: corticosteroids antibiotic. Vasoconstrictors and vasodilators, immunotherapy.