Circulatory Failure

Dr. Khaled M. Al-Qudah
The primary function of the CVS 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 the tissue.
Failure of the circulation in any degree interferes with these exchanges, leading to circulatory failure.

There are two forms of circulatory failure:

1). Heart failure — defect in the heart
2). Peripheral failure — deficiency in the vascular system.
Heart Failure: can be subdivided into two types:

- Congestive heart failure
- Acute heart failure
Congestive Heart Failure
C.H.F
Blood returns to heart from body

Blood goes out to the body

Blood goes to the lungs

Blood returns to heart from lungs

Blood returns to heart from body
The heart due to some intrinsic defect is unable to maintain circulatory equilibrium at rest. Circulatory equilibrium is *deranged* when ventricular output is less than the venous return. If this persists for a significant period, then blood accumulates in the veins and signs of congestive heart failure develop.
These signs include:

1). congestion of the venous circuit,

2). dilatation of the vessels,

3). edema of the lungs or periphery,

4). enlargement of the heart

5). ↑ in the heart rate.
Etiology:

- Myocardial diseases
- Endocardial diseases
- Pericardial diseases
- Pulmonary hypertension
- Systemic hypertension
cardiac defect $\rightarrow$ myocardium contraction reduced $\downarrow$

Compensatory mechanisms by using the cardiac reserve

$\uparrow$ heart rate
$\uparrow$ ventricular filling
$\uparrow$ redistribution of blood flow $\downarrow$

hyperfunction $\downarrow$

dilatation and hypertrophy of the myocardium
Hypertrophic Cardiomyopathy

Normal Heart  Hypertrophied Heart
this will make the animal not able to cope with circulatory emergencies as well as normal animals:

*stage of waning cardiac reserve*  
*(OR)*  
*poor exercise tolerance*

The animal look normal at rest, but incapable of performing exercise.
Finally the compensatory mechanisms reach their physiological limits. When the cardiac reserve is completely lost, **decompensation** occurs with inability to maintain circulatory equilibrium. 

\[ \text{C.H.F} \]
Congestive heart failure may occur in either the right or the left ventricles or in both together:
Right side C.H.F:

Venous congestion is manifested only in the greater circulation.

The systemic veins and capillaries distend as a consequence of right heart being unable to move the venous return forward.

RHF is more common in pericarditis, brisket disease, cor pulmonale, and myocardopathies.
Right side C.H.F. causes involvement of the liver and kidneys.
Kidney

The ↑ of hydrostatic pressure is offset by the reduced flow of blood through the kidney

↓

anoxia

damage of the glomeruli causes increased permeability and escape of plasma protein into the urine

↓

protein urea (albumin urea)
Liver

venous congestion of the portal system is an unavoidable sequel of hepatic congestion, and is accompanied by impaired digestion and absorption and eventually by transudation into the intestinal lumen and diarrhea.
Left side C.H.F.:

venous congestion and engorgement and edema are restricted to the lesser pulmonary circulation.

Pulmonary Oedema
Clinical findings:

Early stages = when the cardiac reserve is reduced, but compensation has not yet occurred:

↑ Respiratory rate
↑ Heart rate

The time required for return to the normal resp.

and pulse rate is prolonged, with cardiac enlargement and the resting heart rate is moderately increased.
Clinical findings...

Left side C.H.F:

↑ the rate and depth of resp. at rest.

Pulmonary edema

**Auscultation** → moist crackles at the base of the lung.

**Percussion** → ↑ dullness on the ventral borders of the lung.
Terminally their is severe dyspnea and cyanosis.

Cough.

The heart rate is increased and their may be murmur referable to the left aterioventricular or aortic semilunar valves.
Clinical findings...

Right side C.H.F.:

↑ heart rate

edema, usually limited to the ventral surface of the body, neck and jaw, manifested as:

anasarca
hydrothorax
hydropericardium
ascites
Clinical findings… Right side C.H.F

1). urine flow is reduced and containing small amount of **albumin** urea.

2). profuse **diarrhea** in late stage

3). body weight may increase because of **edema** but the appetite is poor and the condition is lost rapidly.
Clinical findings... Right side C.H.F

4). the superficial **veins are dilated** particularly the jugular vein.

5). In severe cases their is enlargement of the **liver**, protruding beyond the right costal arch
Prognosis in large animals:

“unfavorable”
Diagnosis of C.H.F:

1. History
2. Clinical findings: edema, engorgement of the J.V.
3. X-ray
4. Albumin urea
5. Aspiration of the fluid from the swelling areas → Transudate

In this case mostly the fluid contain some proteins, because of the leakage of the plasma protein through the dilated capillaries.
Differential Diagnosis:

1. Chronic peritonitis $\rightarrow$ exudate $\rightarrow$ pus

2. Rupture of the urinary bladder
   Accumulation of the urine in the abdominal cavity
   $\rightarrow$ Abdominosenthenesis
3. **Hepatic Fibrosis**

Obstruction of the portal circulation

→ Liver enzymes

→ Liver biopsy

Accumulation of Transudate but it is accompanied by jaundice.
4. **Bottle Jaw** - resulted from hypoproteinemia caused by haemonchus or liver fluke +v fecal analysis for parasites.
Treatment...
1. Give rest to the animal.
2. Salt intake should be reduced to as low as possible.
3. Diuretics: Furosemide
4. Drainage of serious cavities by paracentesis
   Don’t do it very fast.
   Don’t remove more than 2/3 of the accumulated fluids at the one time. *dehydration, shock and death.*
5. You must improve the cardiac contractility\(\rightarrow\) **digitalization**. Digoxin is the extract of digitalis.

**Horse**: give digoxin by oral way or intravenously (I.V.). Initial dose: 1.0 - 1.5 mg/100 kg followed by Maintenance dose: 3.5 mg/100 kg (I.V.)

**Cattle**: you can’t give digoxin orally to the ruminants, Initial dose: 2.2 mg/100 kg followed by 0.34 mg/100 kg every 4 hours.

Don’t give digoxin l.m it cause sever muscular necrosis.
Congestive Heart Failure in Horses

Etiology:

- Primary myocardial diseases:
  - toxic
  - nutritional
  - Viral
  - immune-mediated
Congestive Heart Failure in Horses

- **Secondary myocardial diseases:**
  - toxic
  - drug-induced
  - hypoxia
  - endotoxemia
  - septicemia
  - electrolyte or metabolic disturbances
Clinical Signs

- Jugular distension and pulsation
- Ventral edema
- Fluid-line on auscultation and/or percussion of thorax (pleural effusion)
- Abdominal distension (ascites)
- Diarrhoea
- Atrial fibrillation
Ventral abdominal edema in a horse with heart failure
Ventral abdominal edema in a horse with heart failure
Venous distension (lateral thoracic vessels) in a horse with heart failure
Traumatic Reticuloperitonitis
in Cattle
(Hardware Disease)
a Module for CHF

Dr. Khaled M. Al-Qudah
• Traumatic reticuloperitonitis, or TRP, is a relatively common disease in adult cattle caused by the ingestion and migration of a foreign body in the reticulum.

• Cattle are more likely to ingest foreign bodies than small ruminants since they do not use their lips for prehension and are more likely to eat a chopped feed.
The typical foreign body is a metallic object, such as a piece of wire or a nail, often greater than 2.5 cm in length.

Metal door spring removed from a cow’s reticulum
• The majority of affected cattle (85%) are dairy cattle and 90% are older than 2 years of age.

• Dairy cattle are more commonly affected than beef cattle since they are more likely to be fed a chopped feed, such as silage or haylage.
- A large number of adult dairy cattle have metallic foreign bodies in their reticulum without signs of clinical disease.

- It is likely that a predisposing factor in otherwise normal cows, such as tenesmus or a gravid uterus, causes migration of the foreign body into the reticular wall.
Clinical Signs:
The classic signs associated with TRP are consistent with an:

- Acute, localized peritonitis include:
  - Anorexia,
  - Fever,
  - Tachypnea,
  - **Arched stance with abducted elbows** (indicating cranial abdominal pain)
If the foreign body has penetrated the diaphragm and pericardium, affected cattle also can have:

- Muffled heart sounds
- Jugular pulses
- Brisket edema, secondary to congestive heart failure caused by pericarditis.
Not all cattle develop acute peritonitis

- A significant population of affected cattle develops chronic or subclinical TRP that is not as easily diagnosed as acute TRP.
Clinical signs associated with chronic peritonitis include:

- Anorexia
- Unthriftiness
- Decreased milk production
- Rumen hypomotility
- Change in manure consistency.
A piece of wire has penetrated the reticulum and diaphragm before lodging in the pericardium. Pericardial effusion and fibrin deposition resulted from this traumatic injury.
A nail has penetrated the reticulum, causing traumatic reticuloperitonitis (hardware disease) and the death of this cow
A nail is embedded in the mucosa of the reticulum
High Altitude Disease

Pulmonary Hypertension

Another Module for CHF

Dr. Khaled M. Al-Qudah
The syndrome of right ventricular hypertrophy or right heart failure secondary to pulmonary hypertension.
Etiology:

- Exposure to high altitude:
  At high altitude the low density of the atmosphere results in environmental anoxia (CHRONIC HYPOXIA)
Pathogenesis

environmental anoxia

↓

Pulmonary dysfunction

↓

Prolonged hypoxia and persistence pulmonary vasoconstriction can lead to medial muscular hypertrophy of small pulmonary arteries and arterioles

↑ pulmonary vascular resistance
pulmonary hypertension

↑ in right heart workload

Right ventricular hypertrophy

( COR PULMONALE )

CHF
Treatment:

- Treatment of is aimed at reversing hypoxia. Moving the animal to a lower altitude, if that is not possible, some cows will improve if placed in a warm barn.
- Pneumonia or other pulmonary disease is present it should be treated.

- Supportive therapy:
  - Administration of $O_2$ or $O_2$-enriched air
  - Vasodilators are not effective in treatment of pulmonary hypertension.