The acute heart failure is a cardio-circulatory clinical disease that takes place when a sudden fall of the cardiac expense takes place, without having time to establish the necessary precautions of compensation induced by the mechanisms of regulation of the cardiac expense, generating a situation of tissular anoxia that generates the clinical detectable consequences.

**CARDIOGENIC SHOCK**

It is of a hemodynamic-metabolic secondary syndrome to the acute, serious and widespread alteration of the tissular perfusion and to it immediate consequences on the cellular metabolism, due to a sudden and severe decrease of the cardiac expense.

**ETIOLOGY:**

1. Electric or arrhythmic causes:
   
   Asystole, sinusal node disease (SSS), bradyarrhythmias and tachyarrhythmias.

2. Mechanic causes:
   
   Cardiac alterations that generate a low volume of ejection:

   Congestive heart failure, hypertrophic or expanded cardiomyopathies, arteriovenous shunts, aortic stenosis, cardiac plug, pericardial or valvular fibrosis, heartworm and myocardial ischemia.

3. Extra cardiacal causes:
   
   Chronic respiratory insufficiencies, pleuritis, asthmatic crisis, bronchopneumonia…

   Also we must include the metabolic alterations that can be unleashed indirectly from the already said electrical alterations: hypo and hyperkalemia, hypo and hypercalceamias…

**SYMPTOMATOLOGY:**

In the first phase, due to the increase of the levels of circulating catecholamines and the coronary insufficient perfusion, the patient presents tachycardia, whereas the respiration is fast and superficial. The typical hypotension of the states of shock is demonstrated by a weak and filiform pulse and a long capillary filling time.
The mucous membrane coloration is modified as the state of shock advances: in a beginning they have brilliant red tone due to the tachycardia, later the coloration is a more extinguished red because of the congestion, later the mucous membranes turn into a pale colour due to the vasoconstriction and, finally, a blue-purplish tone is demonstrated because the cyanosis.

Due to the widespread hypoperfusion, it takes place a descend of the corporal temperature that is progressive as the process advances.

The physiopathologic consequences from the shock at a renal level takes place in a quick way, to a descend of the pressure of glomerular filtration with decrease of the rate of this one, with this, initially there is a generation of oliguria and later it turns into an anuria situation with the restoration of a renal insufficiency of pre-renal origin.

When the shock progresses, the clinical picture corresponds to a very pronounced blood hypotension: the pulse is imperceptible, the respiration is slow, bradycardia, cold extremities and a cardiac stop could take place. The patient who in the initial phases is anxious and waved, becomes in the later phase depressed and there could be loss of conscience, coming even to the coma that can be irreversible.

* Poliglobulia with progressive increase of the hematocrit value due to the haemoconcentration.

* Hyperglycemia: as consequence of the sympathetic adrenal response.

* Increased blood urea and creatinine, parallel to the evolution of the renal failure.

* The levels of blood bicarbonate are diminished by the acidosis, the levels of serum lactate are increased.

**DIAGNOSTIC:**

As this is an urgency, the diagnosis must be immediate. It has to be done based on the clinical status and they are necessary, so much for this one as to be able to establish a prognosis, the electrocardiograph, radiographic and laboratory information.

It is necessary to differentiate the type of shock that is being evaluated: important blood losses, traumatism, anaphylactic reactions or endotoxemia.

**PROGNOSIS:**

It is always a serious condition, it will be more if the state of shock is more advanced. The levels of blood bicarbonate, lactate and pH allow to establish a more exact prognosis according to these values, while lower are the levels of bicarbonate and pH and higher those of lactate, more serious is the prognosis.

**TREATMENT:**

It is a question of maximum medical urgency situation and must be establish immediately:

**RESPIRATION MAINTENANCE:**

The respiratory route must be always kept permeable and if there is loss of conscience or state of coma, we have to intubate the animal.

If natural respiration exists, simply we must monitor that is kept and if it is possible to initiate an oxygen therapy across a oxygen mask or any of the different devices commercialised for this end in company animals.

If there is no spontaneous respiration it is necessary to begin a ventilation assisted by a ambu bag, the respiration bag of an anaesthesia equipment or of an automatic fan.

**ATTACK THE ACIDOSIS:**

The acidosis must be controlled as soon as possible, since the prognosis of the shock is narrowly related to
the values of lactate in blood.

To attack the acidosis it is necessary to administer bicarbonate solutions to 1/6 M, the needs of the same one can be decided of direct form since:

\[ \text{Bicarbonate needing} = 0.5 \times (\text{weight in kg}) \times (\text{blood bicarbonate defect}) \]

If the determination of bicarbonate is not possible, this one can be used in dose of 1-2 mEq/kg of a gradual and slow form by IV route. An excess in the administration of bicarbonate produces hypotension, depression, vomiting, cerebral edema and hypercapnia.

The correction of the acidosis must be done gradually since a very rapid administration of the same one can produce ventricular arrhythmias and can aggravate the already existing hypotension.

The clinical state of the patient must determine the rapidity in the treatment with bicarbonate, in extremely serious cases we can administer 3-4 mEq/kg in 1-2 hours, in moderate serious cases we can use 2-3 mEq/kg in 2-3 hours, the least serious cases can be treated mEq/kg in 2-3 hours.

The cats are very sensitive to the changes of serum bicarbonate, they will only receive this product in cases of extreme need and under analytical control since it is frequent to find nervous alterations of dramatic consequences when the therapeutic necessary levels exceed.

**CORTICOTHERAPY:**

Though it is in constant controversy, the corticotherapy in high doses and with medicines of immediate and short action, is beneficial in practically all the shock cases, but its efficiency is excellent in the septic, traumatic and in the hypovolemic shock. The principal effects of the corticoids in the cardiogenic shock probably are:

* To improve the cardiac expense and the functionality of the myocardium, there are authors who affirm that the corticotherapy diminishes the production of the depressor factor of the myocardium.

* To normalize the vasomotor tone, for which they diminish the peripheral resistance and improve the blood perfusion, increasing the cerebral and coronary blood irrigation. Due to this effect, they protect the brain from the metabolic alterations that free themselves at this level.

* Increase the metabolism of the lactic acid, to improve the efficacy of the glycolitic enzymes and to stabilise the lysosomic enzymes.

Though its efficacy in the cardiogenic shock is not so high as in other types of shock mentioned earlier, it is possible to use: dexamethasone in a dose of 2-4 mg/kg-IV or metil-prednisolone in a dose of 10-20 mg/kg-IV.

The dexamethasone has an effect duration of approximately 72 hours and the metil-prednisolone of approximately 12-24-36 hours, this duration must be kept in mind to repeat the later administrations.

**INCREASE THE CARDIAC CONTRACTION:**

This increase is indicated in certain clinical cases, fundamentally in the congestive cardiomyopathies. On the contrary, it is not indicated in the hypertrofic cardiomyopathies, cardiac plugs or constrictive pericarditis or in other types of shock in which the cardiac contraction should be normal.

In case of serious cardiac arrhythmias, the need of usage must be valued since they are factors that unchain arrhythmias, some of them, potentially lethal.

The medicines with positive inotropic action that we can use are:

* **SYNTHETIC CATECHOLAMINES:**

  **DOPAMINE:**
5-10 mcg/kg/minute

**DOBUTAMINE:**

5-10 mcg/kg/minute

In cats: 2.5-5 mcg/kg/minute.

**ISOPROTERENOL:**

0.05 mcg/kg/minute

In all cases, by IV route diluted in infusion solutions.

* PHOSPHODIESTERASE INHIBITORS

**AMRINONE:**

5-10 mcg/kg/minute

**REDUCTION OF THE CARDIAC POSCHARGE:**

The use of sodium Nitroprussiate to a dose of 1-5 mcg/kg/minute, is classic in human medicine, but it represents a major risk of producing hypotension, that is why only it should be used if we can monitor the blood pressure.

**OTHER THERAPEUTIC RESOURCES:**

The temperature must be controlled and prevent it from descending below the normal limits.

The diuresis must be controlled, for which, it will be placed an urinary permanent probe aseptically, supporting a closed system of collection. If it is observed that the diuresis is not adequate, it is necessary to initiate the diuresis with a suitable therapy. The diuretic of election is the Furosemide to dose of 2-4 mg/kg every 4-6-8 hours by IV route. If in 15 minutes following the administration it is not observed an adequate response in the production of urine, it is necessary to administer a new additional dose, even repeating a third time and if the absence of response is kept it is possible to double the dose in the fourth attempt.

To control the intravascular disseminated coagulation, anticoagulants must be administered, as the heparin. It is necessary to initiate the therapy with a dose of 80-100 UI/kg-SC, fitting the following doses and the intervals of dose according to the tests of coagulation. The heparin is ineffective if the acidosis is still present in the peripheral capillaries.

The patient who has overcome a shock will not be completely restored and with his normal constants up to ten or twelve weeks after the process and it must be controlled suitably.

Bibliography


