Resuscitation procedures and life support of the newborn calf

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SUMMARY

In order to reduce calf mortality after calving and during the first 28 days of life, veterinarians generally use appropriate resuscitation procedures and supportive care. The main situations where resuscitation techniques apply, are hypoxia, neonatal diarrhoea and septicemia with or without shock. In cases of hypoxia, resuscitation procedures aim mainly at maintaining oxygenation and ventilation, cardiac output and blood pressure, and at correcting acidosis. In cases of a severe neonatal diarrhoea, the priority for therapy is to restore hydration and, electrolyte and acid-base status. In cases of a septicemia, treatment needs to be aggressively directed against bacteraemia and toxic shock, but also against dehydration, metabolic acidosis, electrolyte disorders and negative energy balance. Moreover, in all cases, supportive care should be used to increase chances of calf recovery. Supportive care requires colostrum feeding, nursing and maintenance in satisfactory ambient conditions. All these resuscitation procedures and supportive care should be attempted each time the newborn calf health is compromised, and pursued until vital state is normalized.

Keywords: Acidosis, calf, diarrhoea, hypoxia, newborn, resuscitation, septicemia, shock, supportive care.

RÉSUMÉ

Procédures de réanimation et soins essentiels à la survie du veau nouveau-né

Afin de réduire la mortalité des veaux nouveau-nés dans leurs 28 premiers jours de vie, le vétérinaire est amené à mettre en œuvre des procédures de réanimation et des soins de soutien. Les principales affections qui nécessitent la mise en œuvre de techniques de réanimation sont l’hypoxie, les diarrhées néonatales et la septicémie néonatale avec ou sans choc. En cas d’hypoxie, les procédures de réanimation ont pour but de maintenir les fonctions pulmonaires et cardiaques tout en corrigeant l’acidose. En cas de diarrhée néonatale sévère, la priorité du traitement est de corriger les troubles acido-basiques et hydro-électrolytiques. En cas de septicémie, le traitement doit être dirigé contre la bactériémie et le choc septique mais aussi contre l’acidose métabolique et les déficits hydro-électrolytiques et énergétiques. De plus, dans tous les cas, des soins de soutien doivent être mis en œuvre pour augmenter les chances de guérison du veau malade. Les soins de soutien concernent l’apport de colostrum, les soins médicaux et le maintien du veau dans les meilleures conditions environnementales possibles. Toutes ces procédures, tant de réanimation que de soutien, devraient être tentées sur tout veau malade et être continuées jusqu’à ce que son état vital se stabilise.

Mots clés : Acidose, choc, diarrhée, hypoxie, nouveau-né, réanimation, septicémie, soutien, veau.

Introduction

Mortality of newborn calves is a major issue of the cattle industry losses. For to 8% of calves die during the perinatal period (during calving or in the first 24 hours of postnatal life) [33, 51], and 2 to 10% of calves during the neonatal period (during the first 28 days) [24, 61]. The major factors that pre-dispose newborn calves to death are: 1) respiratory distress, metabolic acidosis and hypothermia following hypoxemia at birth, 2) metabolic acidosis and dehydration during an episode of diarrhoea, 3) shock and endotoxemia following infection and septicemia. In order to reduce newborn mortality and related financial losses for the owner, veterinarians generally apply specific resuscitation procedures and supportive care. Resuscitation procedures aim at re-establishing normal respiratory and cardiac functions, at restoring blood volume, acid-base and electrolyte status. Supportive care mainly aims at providing adequate energy intake and warmth, and preventing nosocomial infections.

Resuscitation procedures of a hypoxic calf

ABNORMAL ADAPTATION TO EXTRA-UTERINE LIFE IN THE CALF

At birth, a series of structural (cardiac and pulmonary) and physiologic changes are necessary for the calf to survive. This adaptation to extra-uterine life is sometimes difficult, and perinatal mortality is often due to cardiovascular, pulmonary, thermoregulatory or metabolic abnormalities [51]. Dystocia and severe birth hypoxia compromise physiologic transitions, increasing the risk of neonatal mortality.

During the pregnancy, the foetus is normally hypoxic (relatively low oxygen concentration within the arterial blood, with about 80% O₂ saturation and PaO₂ = 21 to 28 mmHg) and hypercapnic (relatively high partial pressure of carbon dioxide within the blood, with a PaCO₂ = 44 to 48 mmHg)
when compared with the newborn calf (about 95% O₂ saturation, PaO₂ = 64 to 80 mmHg and PaCO₂ = 52 to 62 mmHg 12 hours after calving) [5, 56]. In the foetus, the lungs are filled with fluids and are perfused with only a small proportion of the cardiac output. At this time, the foetal lungs are not involved with blood oxygenation and carbon dioxide removal, as they will be following parturition. Moreover, in the foetus, the foramen ovale and the ductus arteriosus are open, and blood crosses from the right to the left side of the circulation as a result of high pulmonary artery pressure and high pulmonary vascular resistance. Pulmonary vascular resistance is high primarily because of local vasomotor responses caused by the relatively hypocoxic condition in the lungs of the foetus. The high pulmonary vascular resistance causes the pulmonary artery blood pressure to be higher than the systemic arterial pressure, and as a result, a right-to-left flow of blood through the ductus arteriosus [7].

During normal calving, uterine contractions and rupture of the foetal membranes cause disturbances in the foeto-placental circulation, which cause a slight and transitional mixed respiratory and metabolic acidosis considered as physiological (venous pH about 7.2) (Table I). Although metabolic acidosis usually resolves within 2 hours of birth, respiratory acidosis may persist for about 24 to 48 hours [56, 59]. Low acidosis together with cooling and environmental tactile stimuli after birth help to induce the first breath and spontaneous respiration in the newborn calf. As the first breath occurs, lungs are inflated with air and this leads to an increased pulmonary blood flow, higher blood oxygenation and decreased pulmonary resistance. This circulatory changes will result in the closure of the ductus arteriosus and the foramen ovale and the beginning of the newborn’s own circulation [26]. After birth, rectal temperature declines steadily during the first 3 hours of life and is maintained in a narrow range thereafter (Table I) [8, 59].

A partial or complete asphyxiation of the foetus can occur just before or just after calving. Anoxia or hypoxemia is largely responsible for the maintenance of foetal circulation in the newborn by generation of pulmonary hypertension and increased pulmonary vascular resistance. The degree of hypoxia and hypercapnia, as well as the resulting respiratory and metabolic acidosis, depends not only on the duration between complete separation from the dam’s blood circulation and the start of spontaneous respiration, but also on the degree of disturbance of diaplacental gas exchanges during delivery. In the normal newborn, metabolic acidosis usually is resolved within 2 hours of birth but respiratory acidosis may persist for about 24 hours. In cases of severe acidosis, postnatal compensation of metabolic acidosis is slower (up to 6 hours) and respiratory acidosis is present up to 48 hours after delivery [51]. Hypoxic calves have significantly higher plasma lactate concentrations contributing to the progressive primary metabolic acidosis because lactate production, presumably from anaerobic metabolism, exceeds capacity of the calf to utilize lactate as an energy source [17, 55]. In some cases, the severe metabolic acidosis is never corrected and contributes to the calf death.

The degree of hypoxia in a newborn calf is evaluated if available by measuring acid-base parameters in arterial blood gas analysis (pH, pCO₂, pO₂, base excess BE and bicarbonate HCO₃⁻) or can be grossly estimated using clinical signs such as muscle tone, reflexes and cardiac status [50], time from birth head-righting, till sternal recumbency [45], first obvious efforts to stand, standing up or first suckling [53]. Actually, there is a significant correlation between tonicity of the newborn immediately after birth and parameters characterising acid-base imbalance. In paediatric medicine, the Apgar score is used to assess the newborn children immediately after birth. This score evaluates skin colour, heart rate, reflex activity, muscle tone and breathing. In calves, score systems have also been developed to evaluate the acid base status without laboratory tests (Table II).

In the normal newborn, respiration is usually initiated within 30 seconds after calving. Respiration tends not to be regular at first, but soon settles down to a rate of 40 to 60 breaths per minute (bpm). Moreover, the calf has a head-righting reflex almost immediately after birth. Sternal recumbency is usually attained within two to three minutes, followed rapidly by attempts to stand after fifteen to thirty minutes. In a hypoxic calf, onset of the normal behaviour is markedly delayed or absent. Hypoxic newborns may struggle and appear bright initially but have difficulty keeping sternal

<table>
<thead>
<tr>
<th>Parameters</th>
<th>5 min</th>
<th>1 h</th>
<th>6 h</th>
<th>12 h</th>
<th>24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.21 (0.04)</td>
<td>7.25 (0.02)</td>
<td>7.28 (0.01)</td>
<td>7.3 (0.02)</td>
<td>7.34 (0.02)</td>
</tr>
<tr>
<td>pO₂ (mm Hg)</td>
<td>27.9 (2.3)</td>
<td>33.7 (2.8)</td>
<td>35.7 (2.4)</td>
<td>30.1 (2.3)</td>
<td>31.6 (4.1)</td>
</tr>
<tr>
<td>pCO₂ (mm Hg)</td>
<td>75.6 (2.8)</td>
<td>67.5 (3.0)</td>
<td>64.6 (1.6)</td>
<td>64.9 (3.9)</td>
<td>59.8 (3.5)</td>
</tr>
<tr>
<td>BE (mmol/L)</td>
<td>1.65 (0.6)</td>
<td>1.9 (1.0)</td>
<td>2.66 (0.7)</td>
<td>4.22 (0.4)</td>
<td>5.48 (1.4)</td>
</tr>
<tr>
<td>HCO₃⁻ (mmol/L)</td>
<td>28.3 (0.7)</td>
<td>28.2 (0.8)</td>
<td>28.7 (0.5)</td>
<td>30.0 (0.5)</td>
<td>30.4 (1.4)</td>
</tr>
<tr>
<td>Rectal temperature</td>
<td>39.6 (0.03)</td>
<td>39.4 (0.20)</td>
<td>39.0 (0.23)</td>
<td>38.8 (0.16)</td>
<td>39.1 (0.11)</td>
</tr>
</tbody>
</table>

TABLE I: Changes in neonatal venous blood pH, acid base variables and rectal temperature during the early postpartum period in healthy newborn calves (mean value +/- SE) [59]. Peripheral venous samples are less satisfactory than arterial samples to evaluate the calf’s quality of ventilation and respiratory function. However, in practice, there are often used it’s easier to take them.
recumbency, have depressed or absent sucking reflex, are slow to stand or remain recumbent and develop a depressed mentation (reduced or absent response to stimuli, somnolence, lethargy) within hours. Hyperventilation (> 60 bpm) initially occurs, followed within a few minutes by a period of progressively lesser breathing efforts [21, 26]. If the calf exhibits superficial abdominal breathing, has poor reflexes, or takes more than 15 minutes to achieve sternal recumbency, the vital prognosis is poor [45, 56]. The vital prognosis is also reserved if the newborn a primary apnea (absence of spontaneous breathing for 1 to 5 minutes after delivery). Abnormal neonatal behaviour in the immediate post-natal period is commonly secondary to perinatal hypoxia. In utero infections and congenital neurologic abnormalities should also be considered as possible causes of abnormal neonatal behaviour. Compromised newborn calves that survive to delivery are also prone to hypothermia and infectious diseases due to lack adequate colostrum intake [3, 8, 54]. It is usual to observe a mild combined respiratory and metabolic acidosis in a newborn calf just after parturition. Immediately after birth, venous blood pH could be about 7.2 and base deficit of +1 to +3 mmol/L (Table I). In cases of dystocia and hypoxia, pH may fall to 7.0 or even less and base deficit is about -10 to -15 mmol/L [17, 23, 45].

### SPECIFIC RESUSCITATION PROCEDURES TO CORRECT HYPOXIA AND ITS CONSEQUENCES

A hypoxic newborn calf requires resuscitation procedures in order to maintain oxygenation and ventilation, cardiac output and blood pressure and, to correct acidosis. These procedures must be applied immediately after birth if necessary, according to the “ABC” order: “A” for “airway patency”, “B” for “breathing stimulation” and “C” for “circulation support”. However, before the birth (i.e. during the calving), if the calf begins to exhibit signs of reduced vigor (capital or lingual edema, buccal or lingual cyanosis, scleral hemorrhages, reduced responsiveness to stimulation), a forced extraction of the calf or a caesarean section should be fast performed to reduce the foetal distress and to initiate resuscitation procedures as quickly as possible. Resuscitation can begin while the calf is still in the birth canal by airway clearance (with rubbering of nasal cavity using a suction pump) and breathing stimulation (with rubbing of nasal cavity using straw or a finger) [12].

Just after birth, the initiation and maintenance of respiration is of the greatest importance since, without it, neurologic damage rapidly occurs through hypoxic tissue damage. Thus, newborn’s respiratory tract must be cleared immediately after delivery in order to reduce respiratory resistance and to improve alveolar ventilation. Fluid are cleared using a suction pump or by suspending the calf by the hind limbs [21, 34, 57]. Although only a small volume of fluid (< 10 mL) is generally removed, the suctioning procedure significantly benefits pulmonary gas exchanges and acid-base balance [57]. Moreover, suspending the newborn by the hind legs during less than 90 seconds just after delivery could be useful for clearing the airways and improving respiratory and metabolic adaptation to extra-uterine life [57, 58]. After that, if the newborn breathes spontaneously, it is placed in sternal recumbency because that position has a favourable impact on the efficiency of pulmonary gas exchanges and on correction of the mixed acidosis [58].

If the calf does not breathe or move spontaneously within a few seconds after birth, tactile stimulations (inserting a finger or a length of straw in the nasal cavity, rubbing the calf’s body with straw or towel, pouring cold water in the ear or over the head or whole body of the calf) is necessary to initiate a gasping reflex and help aerate the lungs [19, 32, 33, 34]. Hypothermal stimulation with cold water has a beneficial effect on pulmonary gas exchange and acid-base balance in calves [57]. In dyspneic neonatal calves, the respiratory centre can be stimulated with respiratory stimulants such as doxapram (1 mg/kg intravenously), cropropamide-crothetamide (1 mg/kg intranasally) or naloxon (10 μg/kg intravenously) (only the two first molecules existing in France for use in calves) [33, 38, 40, 56], with acupuncture by inserting and rotating a needle in the nasal philtrum [44] or with pinching of the nasal septum [33]. However, the use of respiratory stimulants is controversial because these drugs could be ineffective in cases of secondary apnea (absence of spontaneous breaths 5 minutes or more after calving) if the hypoxic newborn neonate suffers profound central nervous system depression secondary to hypercapnia. It is then necessary to realise a manual ventilation without losing of time [34, 56]. Cerebral oxygenation could be improved with vasodilators such as xamthine derivatives such as aminophylline (2.2 to 4.4 mg/kg intramuscularly) or tolazoline (1 mg/kg intravenously, licensed in France for use in calves) even if their real efficacy is called into question. Xanthine derivatives such as aminophylline (2.2 to 4.4 mg/kg intramuscularly) can be given to stimulate bronchodilatation and diaphragmatic contractility [51]. Alpha-two-antagonists could be used to decrease the pulmonary vascular resistance but their use is not allowed in cattle in France. Tolazoline is used slowly by intravenous route at the dose of 0.5 to 2.0 or even 4.0 mg/kg and atipamezole by intravenous route at the dose of 20 to 60 μg/kg [40, 56].

If these procedures do not result in spontaneous breathing within 2 min after birth, more intensive resuscitation efforts

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TABLE II: Evaluation of the degree of vitality in the newborn calf from muscle tone, attitude and cardiac status [50].

<table>
<thead>
<tr>
<th>Vitality score</th>
<th>Clinical signs</th>
<th>Base deficit (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Stillborn (toneless, no head-righting reflex, cardiac activity absent).</td>
<td>25</td>
</tr>
<tr>
<td>1</td>
<td>Toneless, no head-righting reflex but cardiac activity present.</td>
<td>25</td>
</tr>
<tr>
<td>II</td>
<td>Low toxicity, lateral recumbency with head requiring support, reduced response to stimuli.</td>
<td>9</td>
</tr>
<tr>
<td>III</td>
<td>Normal toxicity, head erect, normal response to stimuli.</td>
<td>1</td>
</tr>
</tbody>
</table>
(positive pressure ventilation) must be performed. Mechanical ventilation could be made with an endotracheal tube and an Ambu-bag provided with pressure valves to avoid the risk of overpressure and alveolar damage. An endotracheal tube with a 7.5- to 11-mm internal diameter is usually used in the newborn calf. Intubation can be accomplished with the aid of a laryngoscope with a long blade. Intubation can also be made blindly by external palpation of artenoids with one hand, while guiding the endotracheal tube into the lumen of the trachea with the other hand [21, 23]. The lungs are inflated manually with room air by squeezing the Ambu-bag once every 2 seconds while taking care not to overinflate them (less than 40 cm H₂O, pressure controlled by the pressure valve and manometer attached to the bag). Higher pressure (40 to 80 cm H₂O) is required only for the first few breaths to open the airways and fill the lung with air [26, 27]. During ventilation, it is important that the calf is held in sternal recumbency so that both left and right lung fields are ventilated. If endotracheal tube and Ambu-bag are not available, respiratory ventilation can be improved with a face mask or just by a mouth-to-nose ventilation and the oesophagus should be closed by pinching to avoid that air filling the abomasum [40]. However, it is difficult to establish a normal respiratory pattern by these methods and moreover, with the mouth-to-nose ventilation there is a risk of contracting zoonotic diseases [34].

Immediately after birth, a tachycardia (150 to 180 bpm) is normal while a bradycardia (< 60 beats/min) or an arrhythmia is distress sign of the newborn calf [56]. Chest compression must be initiated if bradycardia persists despite ventilation and a nonperfusing rhythm (ineffective heart rhythm to sustain the perfusion of vital organs) is present. The calf is placed in lateral recumbency while the operator kneels are at the calf’s back. Compression is performed with the palm of the hand on the chest. The compressions are rhythmic at a rate of 60 to 70 per minute. The aim is to maintain mean pressure in the circulation rather than to increase peak pressures. The calf is ventilated every three cardiac compressions [21]. Low dose epinephrine (0.02 mg/kg intravenously or, if vascular access is not available or cardiac activity ineffective, intratracheally – less effective - or intracardiac) could be administered to increase cardiac output. This produces a rapid tachycardia [34, 40]. If there is no response within 3 min, low additional doses could be repeated every 3 to 5 minutes. Rapid intravenous infusion of isotonic fluid (20 to 40 ml/kg) may help to increase the vascular volume. If, despite chest compression, the calf remains severely bradycardic or asystolic, vasoactive substances as dopamine, dobutamine or ephedrine may be administered. Cases of sinus bradycardia respond well to atropine. Atropine is administrated intravenously to the dose of 0.01 to 0.03 mg/kg [21] and could be repeated every 3 to 5 minutes. However, the administration of drugs must be considered secondary to chest compression and ventilation which are the most critical procedures to initiate in the resuscitation procedure.

Furthermore, buffer therapy (with sodium bicarbonate NaHCO₃ or carbicarb) and glucose infusion are necessary to compensate severe respiratory and metabolic acidosis, and hypoglycemia. However, acidosis correction with NaHCO₃ requires that the calf is breathing normally to eliminate produced CO₂. Contrary to NaHCO₃, carbicarb (an equimolar mixture of sodium bicarbonate NaHCO₃ and sodium carbonate Na₂CO₃) does not increase the concentration of CO₂ because of its composition and mechanism of action. However, BLEUL et al. [4] have found that there are no life threatening changes in blood gas variables (pH, pCO₂) after treatment with either NaHCO₃ or carbicarb, even in severely acidotic calves. In comparison with NaHCO₃, carbicarb seems to have only some advantages in animals being ventilated or with extremely ventilatory ability [10].

**Therapeutic procedures of the diarrhoeic calf**

**PATHOGENESIS OF NEONATAL DIARRHOEAS IN THE CALF**

Regardless of the etiologic agent, the metabolic changes resulting from diarrhoea include dehydration, acidosis, electrolyte imbalance due to intestinal losses and negative energy balance or hypoglycaemia, or both. The major cause of dehydration in these calves is fecal fluid loss, which can be as much as 13% of body weight in 24 hours. Acidosis results from intestinal bicarbonate and strong cation losses in the faeces, lactic acid accumulation in tissues (formation of L-lactate from anaerobic glycolysis following tissue hypoperfusion), decreased renal excretion of acid and increased production of organic acid secondary to bacterial fermentation in the intestine [37]. Elevated levels of D-lactate are common in the blood of diarrhoeic calves which cause alterations in posture, reflexes and behaviour by action on the nervous system [29, 30]. Along with water and bicarbonate, sodium, chloride and potassium are lost in the faeces, which results in a total body deficit of these ions. Many dehydrated, acidic calves are hyperkaliemic, yet they have a total body potassium deficit. This paradox is the result of a shift of potassium from the intracellular compartment into the extracellular compartment during acidemia.

**SPECIFIC THERAPEUTIC PROCEDURES TO CORRECT CONSEQUENCES OF DIARRHOEA**

The priority of therapy for calves with acute, severe infection of the digestive tract is to restore hydration and electrolyte status, and to correct acidosis and hypoglycaemia. The therapy is completed by administration of antimicrobials with a predominantly Gram-negative spectrum of activity to prevent septicaemia and bacteraemia, and by administration of non-steroidal anti-inflammatory agents to decrease inflammation in the gastrointestinal tract, to reduce intestinal pain and to prevent the effects of possible endotoxemia [11, 40].

Before application of any therapy, hydration, volemia and acidosis must be checked. The degree of dehydration can be evaluated from clinical signs as sunken eyeballs (enophthalmos) and decreased skin turgor (skin tent duration over the thorax) (Table III). The degree of dehydration (in percent of body weight) can be estimated by multiplying the enophthalmos (in mm) by 1.71 [13]. The effective circulating blood volume and cardiac output can be evaluated from heart rate, pulse...
However, laboratory determination of acid-base parameters with using venous blood gas analysis is not easy to perform in the veterinary practice. Acidity can be evaluated from clinical signs even though it can be difficult to accurately assess its intensity upon clinical examination. The clinical signs to consider are: 1) warmth of oral cavity, by fingers in contact with the hard or soft palate, 2) warmth of extremities, in clapping the hand around the fetlock, 3) intensity of sucking reflex, by the index finger over the tongue, 4) menace reflex with rapid hand movement toward eye, 5) tactile reflex, by pinching skin over lumbar area and 6) ability to stand. The stronger the metabolic acidosis, the more the reflexes (such as suckling reflex) are decreased, cool extremities, calf depressed, able to stand but with difficulty. The determination of haematocrit or blood total protein can be used to evaluate dehydration intensity. The more the dehydration is, the more the haematocrit and total protein are increased. After evaluation of dehydration, the degree of metabolic acidosis must be evaluated in the diarrhoeic calf. The acidosis degree is normally determined by measurement of acid-base parameters with using venous blood gas analysis (pH, HCO₃⁻, BE). However, laboratory determination of acid base status is not easy to perform in the veterinary practice. Acidosis can be evaluated from clinical signs even though it can be difficult to accurately assess its intensity upon clinical examination. The clinical signs to consider are: 1) warmth of oral cavity, by fingers in contact with the hard or soft palate, 2) warmth of extremities, in clapping the hand around the fetlock, 3) intensity of sucking reflex, by the index finger over the tongue, 4) menace reflex with rapid hand movement toward eye, 5) tactile reflex, in pinching skin over lumbar area and 6) ability to stand. The stronger the metabolic acidosis, the more the reflexes (such as sucking reflex) are decreased, cool extremities, calf depressed, able to stand but with difficulty.

### TABLE III: Guide to assessing the degree of dehydration and the base deficit of calves with neonatal diarrhoea [25, 40, 48].

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>Percentage of dehydration (%)</th>
<th>Approximate base deficit (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of diarrhoea with minimal clinical signs</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Eyes beginning to sink (2 to 4 mm), skin turgor decreased</td>
<td>About 7</td>
<td>10</td>
</tr>
<tr>
<td>(skin tent duration of 1 to 2 seconds), sucking reflex present</td>
<td>(mild dehydration)</td>
<td></td>
</tr>
<tr>
<td>Eyes sunken (4 to 6 mm), skin turgor more decreased</td>
<td>About 9</td>
<td>15</td>
</tr>
<tr>
<td>(skin tent duration of 2 to 5 seconds), mucous membranes tacky, sucking reflex decreased, cool extremities, calf depressed, able to stand but with difficulty</td>
<td>(moderate dehydration)</td>
<td></td>
</tr>
<tr>
<td>Eyes deeply sunken (6 to 8 mm), skin tent and does not return or very slowly (5 to 10 seconds), dry mucous membranes, sucking reflex very weak or absent, cold mouth, cold extremities, calf depressed, unable to stand and possibly comatose</td>
<td>About 11</td>
<td>20</td>
</tr>
<tr>
<td>Eyes very deeply sunken (8 to 12 mm), calf comatose or dead</td>
<td>&gt; 12</td>
<td>&gt; 20</td>
</tr>
<tr>
<td></td>
<td>(fatale dehydration)</td>
<td></td>
</tr>
</tbody>
</table>

Depending on calf’s clinical state (degree of dehydration and intensity of sucking reflex), oral or intravenous fluids are administered. When the calf is mildly to moderate estimated dehydration (Table III), sucking reflex is present and gastrointestinal functions are not too seriously compromised (no ileus or abdominal distension), fluid requirements are provided by the enteral route, using commercial dextrose and electrolytes mixtures. However, if the gut alteration is severe (ileus and abdominal distension) or moderate to severe dehydration is present (Table III) or sucking reflex is weak or absent, intravenous route is a preferred method of fluid administration. Volumes of fluid to be administered depend on existing fluid deficit and ongoing fluid losses associated with the diarrhoea (Table IV).

### TABLE IV: Determination of isotonic fluid volume and bicarbonate requirements in a diarrheic calf.

<table>
<thead>
<tr>
<th>Isotonic fluid volume need during the first 24 hours in a diarrheic calf</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Fluid volume needed to correct dehydration (liter) = weight (kg) x dehydration degree (%)</td>
</tr>
<tr>
<td>2) Fluid volume needed to correct fluid losses associated to diarrhoea (liter) = 1 to 4 liters depending on the severity degree of diarrhoea</td>
</tr>
<tr>
<td>3) Fluid volume needed to maintenance (liter) = 0.05 liter x weight (kg)</td>
</tr>
<tr>
<td>Total isotonic fluid volume (liter) = 1 + 2 + 3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bicarbonate need to correct acidosis in a diarrheic calf</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bicarbonate requirements (mmol) = base deficit or bicarbonate deficit (mmol/L) x calf weight (kg) x 0.6</td>
</tr>
</tbody>
</table>

*Half of bicarbonate requirements is given first then the degree of acidosis is re-evaluated.*
diarrhoea contains at least 50 mmol/L of an alkalinizing agent (preferably acetate or propionate), have a SID of at least 60 to 80 with a sufficient supply in sodium (90 to 140 mmol/L) and provide energy. A hypertonic oral solution (500 to 600 mOsm/L) is recommended in calves which have been separated from the dam and/or deprived of milk. Isotonic solutions may be used for calves which are still suckling [40, 43, 48]. Milk feeding for calves with diarrhoea must be continued (and not discontinued) because milk is an excellent source of energy (more dense than even hypertonic oral rehydration solutions) which minimizes the weight loss associated with diarrhoea [11]. However, an oral solution containing bicarbonate (more than 25 mmol/L) and citrate (more than 12 mmol/L) must not be fed together with milk or milk replacer because it inhibits clot formation in abomasum [11]; it is then recommended to separate the feeding of milk and electrolytes by 4 hours. For example, if the calf is normally being fed twice a day (morning and evening), the oral electrolytes can be fed in the middle on the day. As a supplement to the milk (3 liters twice), 4 liters of oral electrolytes solution can be given to a 50-kg calf once to twice a day during 1 to 4 days (Table V) [40].

If the calf is severely dehydrated or acidic, depressed, unable to stand, do not suckle for more 24 hours, intravenous fluid therapy is necessary. The optimum type of intravenous fluid to be administered depends on the acid-base, glucose and electrolyte status of the ill calf, which can be determined in practice with portable analyzers (blood gas analyzer, pH meter, Harleco apparatus, glucometer, biochemical analyzer). Specific correction of acidosis with intravenous fluids is not necessary if the blood pH is greater than 7.25. In most circumstances, and in the absence of adapted laboratory services, balanced electrolyte solutions such as lactated Ringer’s, ace-tated Ringer’s or saline-based fluids are satisfactory to replace fluid deficits and to correct moderate acidosis. Lactated Ringer’s solution is routinely used in veterinary medicine but it is theoretically inferior to acetated Ringer’s solution since diarrhoeic calves may have increased blood lactate concentrations. Moreover, the rate of lactate metabolism to glucose is decreased in severely dehydrated calves, resulting in delayed alkalinisation. Acetate in the acetated Ringer’s solution is metabolized more rapidly and therefore increases blood pH at a faster rate than the lactated solution [9, 10].

### Table V: Example of table to calculate the dairy oral (electrolytes solution, milk and/or water) fluid requirement (in liters) and the recommended frequency of administration (assuming 70% of fluid absorbed) [31].

<table>
<thead>
<tr>
<th>Estimated loss associated to diarrhoea (L)</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight of calf (kg)</td>
<td></td>
<td>50</td>
<td></td>
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</tr>
<tr>
<td>40</td>
<td>5.6</td>
<td>7.0</td>
<td>9.9</td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>6.6</td>
<td>8.0</td>
<td>10.9</td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>7.6</td>
<td>9.1</td>
<td>11.9</td>
<td></td>
</tr>
</tbody>
</table>

* If isotonic intravenous fluids are used to correct the initial fluid deficit, calculation of ongoing maintenance and losses should assume 0% dehydration.

- Twice daily
- Three times daily
- Four times daily

The rate of intravenous administration of isotonic solutions to a dehydrated calf can be as rapid as 80 ml/kg/hour [10, 25]. At this maximum rate, the extracellular fluid losses are replaced within one hour in a 40-kg calf with 8%-dehydration. However, the fluids must be administered at a slower rate than this because of the risk of hypertension and possible pulmonary edema. A more reasonable rate of administration is 30 to 40 ml/kg/hour. Using this slower flow rate, rehydration and correction of acidosis is completed in 2 to 3 hours for a 40-kg calf [25].

However, administration of large volumes of isotonic fluids is sometimes difficult to perform in practice because it requires catheterization of a jugular vein, appropriate restraint and periodic monitoring by the farmer. Small intravenous volumes (4 to 5 mL/kg) of hypertonic saline solution (7.2% NaCl) or hypertonic saline solution (7.2%) and dextran 70 (6%) can be given in combination with intravenous isotonic electrolyte solution [60] or oral electrolyte solution [47] for respiratory disease (inability to effectively exhale the excess CO2 generated in buffer reactions) [1].

the treatment of dehydrated diarrhoeic calves. Hypertonic saline can be administered at a rate of 60 ml/kg/h [10].

Administration of potassium to a hypokalaemic calf seems inadequate at first, but the objective is to replace total body potassium deficit that exists despite hypokalaemia. Administration of potassium to hypokalaemic calves can be accomplished safely at the rate of 0.5 mEq of K+/kg/h if bicarbonate and dextrose are administered concurrently [10, 40].

The addition of glucose (2.2 to 4.4 ml/kg/h of a 5% solution) to rehydration fluids has favourable effects (available energy, increase of the movement of potassium from the extracellular fluid compartment to the intracellular fluid compartment) and usually does not result in significant glucosuria or osmotic diuresis.

**Therapeutic procedures of a septicemic or shocked calf**

**PATHOGENESIS OF SEPTICAEMIA AND SHOCK IN THE NEWBORN CALF**

Neonatal septicaemia is mainly associated with young calves deprived of colostrum. *E. coli* has been traditionally incriminated as the most important agent responsible of septicaemia in the newborn calf [2, 19, 20]. In Europe, the O78 and O117 serotypes are often involved. In order to cause septicaemia, the bacteria must invade the host, survive the non specific as well as the specific immune response mechanisms and finally cause some harm via an endotoxin or exotoxin. The intestine and the umbilicus remain important sites of entry for bacteria. Inadequate passive immunity transfer and dystocia are often associated with septicaemia [19].

Calf septicaemia is accompanied by decreased milk intake, hypoglycaemia and, if diarrhoea is present, increased loss of electrolytes (Na+, Cl-) and water in the faeces. That leads to dehydration, metabolic acidosis, electrolyte imbalance (deficit in Na+, Cl- and HCO3- and excess of K+ in blood), negative energy balance and bacteraemia with toxic shock [9, 22].

Shock is a condition with tissue hypoperfusion, cellular hypoxia and ultimately cell death. In calves, the most commonly encountered type of shock is endotoxic or septic shock caused by septicaemia with *E. coli*. Endotoxic shock is characterized by dyspnea, depression, congested mucous membranes, recumbency and death. Cardiovascular effects include decreased mean arterial blood pressure and cardiac output, and increased pulmonary arterial pressure. Decreased plasma volume and extracellular fluid volume lead to decreased cardiac output, peripheral perfusion and oxygen delivery, ultimately producing a hypovolemic shock. Metabolic acidosis is due to lactic acid production in tissues secondary to tissue hypoxia, decreased acid secretion by poorly perfused kidneys, intestinal bicarbonate loss if diarrhoea is present, and forestomach/intestinal fermentation of lactose and glucose to volatile fatty acids and lactate. The metabolic acidosis results to progressive central depression, decreased suckling reflex, ataxia, recumbency, coma and then death [15].

**SPECIFIC THERAPEUTIC PROCEDURES TO CONTROL SEPTICAEMIA AND SHOCK**

Septicaemia treatment needs to be aggressively directed against bacteremia and toxic shock but also against dehydration, metabolic acidosis, electrolyte abnormalities and negative energy balance. The treatment is based on broad-spectrum antibiotic therapy (administered intravenously when possible) associated with fluid therapy to control toxic shock and acidosis.

The traditional treatment for shock is rapid intravenous injection of crystalloid solutions (NaCl, Ringer’s solution) to increase the central venous pressure and maintain adequate tissue perfusion. Rate of administration can be high (25 to 75 ml/kg/h), especially during the first 30 to 60 minutes. Hypertonic saline solution (7.2% NaCl, 4 to 5 ml/kg over 4 to 5 minutes) can be used first and this therapy must be followed by slower injection of an alkalinizing poly-ionic electrolyte solution and oral electrolyte solution [9, 12, 49]. Pressor agents such as dopamine or dobutamine can be administered in addition to fluid therapy if there is severe hypotension [9].

In cases of metabolic acidosis, calves with a venous blood pH < 7.2 must be treated intravenously with sodium bicarbonate (about 500 ml to 2 liters of isotonic or hypertonic solutions, depending on the base deficit, the need in bicarbonate being determined as in a calf with diarrhoea: see Table IV and the previous part about specific therapeutic procedures to correct consequences of diarrhoea). Acetated Ringer’s solution rather than Lactated Ringer’s solution can be administered to septicemic calves with a blood pH > 7.2, the volume to administrate being function of dehydration degree, severity of diarrhoea and maintenance need (Table IV).

Whole-blood transfusion (10 to 30 ml/kg) or plasma transfusion (10 to 15 ml/kg) may be of benefit septicemic colostrum-deprived calves [10]. Because blood is easier and cheaper to obtain than plasma, whole-blood transfusion is usually used in neonatal calves.

The treatment of septicaemia or endotoxic shock is completed by intravenous administration of nonsteroidal anti-inflammatory drugs (flunixin meglumine at the dose of 2.2 mg/kg then 1.1 mg/kg three times a day, or ketoprofen at 3 mg/kg once a day) because of their analgesic, anti-inflammatory and anti-endotoxemic properties [46]. A single dose of short-acting soluble corticosteroids (dexamethasone at the dose of 2.2 mg/kg, or prednisolone sodium succinate at 1.1 mg/kg) could also be used intravenously in shock treatment when thought to be absolutely essential (i.e. signs of severe hypovolemic shock as a weak pulse, a reduced venous blood pressure, a increased capillary refill time and recumbency or coma) because corticosteroids improve survival rate while improving tissue perfusion, decreasing neutrophil aggregation, leucocytes activation and degranulation, and preventing disseminated intravascular coagulation, in particular.

**Additional support care in the newborn calf**

Sufficient amounts of colostrum (about 2 to 4 liters for a 40-kg calf depending on the content of colostral immuno-
globulins) should be given to newborn calves within the first 6 hours of life because colostrum provides a concentrated source of energy and immunoglobulins. If feeding of colostrum is delayed from this period, it results a hypo or agammaglobulinemia in neonate calves. However, it is important to continue to feed colostrum for 2 to 3 days after birth because it reduces the incidence of neonatal diarrhoea during the first several weeks of life [39, 40]. Colostrum should be suckled by the calf from its mother or fed via nipple bottle or oesophageal feeder if the newborn is too weak to nurse. If possible, it is preferable to allow calf sucking because compared to hand feeling, sucking is a greater source of absorption of colostral immunoglobulins. Moreover, in cases of use of oesophageal feeder, the absorption of immunoglobulins is lower compared with the administration by bottle because colostrum administrated by intubation enters in the rumen before moving into the abomasum; it takes 2 to 4 hours for the colostrum to leave the rumen what therefore delays the absorption of immunoglobulins. Others factors can affect the absorption of immunoglobulins, as the ambient temperature outside (extreme cold reducing the absorption), the colostrum quality (high quality colostrum with high immunoglobulin content resulting in higher serum immunoglobulin than a low quality colostrum for the same amount of colostral immunoglobulins), the milk intake before first colostrum (ingestion of non-colostrum milk accelerating the closure of the intestinal epithelium) and the clinical state of the neonate calf (hypoxia delaying the absorption of immunoglobulins) [39].

Milk must not be withheld or no more than 24 to 48 hours if the calf is too weak to suckle. Ill calves should be stimulated to suck several times a day. Milk must not be administrated by intubation with oesophageal feeder to avoid his putrefaction in the rumen. Milk or oral solutions should be administrated warm (39-40°C) to help correct or prevent hypothermia.

Nursing care (navel disinfection with iodine, dryness of bedding, reduction of stress) must be carefully performed to avoid secondary infections. Stresses due to climatic or environmental conditions and those due to handling (as during dehorning), house change (calving pen, calf hutch, group pen) or treatments should be minimized as possible. If a great risk of nosocomial infections exists in the farm, prophylactic administration of antibiotics to each newborn neonate should be used.

As neonatal calves are remarkably cold tolerant in a dry, still-air environment, management strategies should focus on providing a dry, draft-free bedding sheltered from wind to calves [8]. Moreover, vulnerability of newborns to contagious and opportunistic pathogens is amplified by adverse environmental conditions. Thus, maintenance of satisfactory ambient conditions is a crucial component to improve chances of survival. Just after the delivery, drying the calf with a blanket and a hair dryer not only helps reduce heat loss but also helps stimulating the calf [21]. If the calf is down and hypothermic (< 34.5°C), efforts must be made to provide exogenous sources of warmth so that energy used for metabolic heat production is minimized. This can be optimised by use of radiant heaters and hot-water bottle, by immersion in a warm-water bath at 38°C [36, 41, 42] or by positioning in a warming device whose air temperature varies from 20 to 25°C and even up to about 40°C (such as “Roy-L-Heat Calf box” equipped of a circulating fan and thermostat [52] or other techniques adapted from to those developed for lambs [18, 42]). Immersion in warm water is a more rapid method (in terms of recovery time) for external rewarming than heat pad and heat lamps methods [36, 42]. Moreover, recovery from hypothermia requires less metabolic heat production with immersion technique than with the other warming techniques [42]. It is also advised that the intravenous or oral solutions are warmed before injection (about 39°C) to correct or prevent hypothermia. In hypothermic lambs, injection of a glucose solution prior to warming is effective to reverse hypothermia [18]. Although similar studies have not been conducted in hypothermic calves, administration of glucose to the calf before or during warming procedures is recommended [6].

Moreover, efforts must be made to protect the newborn which may be injured by the mother if the calf is down or weak. If the mother (particularly a first-calf beef heifer) presents a poor maternal behaviour towards her calf (by butting, kicking or trampling) and the newborn calf is judged to be not very vigorous, the calf should be temporarily removed from his mother and placed in a calf pen next to her then replaced near her to suckle under control of the farmer.

Conclusion

All the resuscitation procedures and supportive care should be attempted each time survival of the newborn calf is compromised, and continued until this vital state is normalized. It is advisable to implement them as soon as possible to decrease the risk of calf’s mortality because success in resuscitation will depend largely on the condition of the calf when veterinary treatment is applied. However, as the resuscitation procedures and supportive care are not always successful, it is also important to educate farmers to the risk factors of severe neonatal disorders requiring resuscitation procedures. In order to avoid hypoxia secondary to dystocia or prolonged calving, farmers should practice good calving supervision and, for any calving which needs assistance, intervene about 2 hours after the appearance of the amnion or foetal hooves at the vulva or contact his veterinarian. Additionally, prevention of neonatal infectious diseases is based on maintaining maternity and calf pen hygiene, ensuring adequate vaccination of pregnant cows and early intake of good quality colostrum by the newborn calf, ensuring navel disinfection, early detecting and treating neonatal diseases.

References


