Animal welfare in the newborn piglet: a review

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ABSTRACT: The objective of this review is to integrate clinical findings and laboratory analyses in such a way to improve the welfare of newborn piglets and achieve better prognoses of neonatal viability. Deaths during the intrapartum period account for a significant proportion of pre-weaning mortality in farms worldwide. Piglets which die during parturition generally have normal size and typically lack gross lesions at post-mortem examination. However, circulatory abnormalities in the umbilical cord help in assessing piglet viability. Cord lesions can be classified as normal (adhered), oedematous, congested or haemorrhagic and should always be evaluated in perinatal deaths. The likelihood of neonatal survival decreases rapidly as the severity of umbilical cord lesions increase. The physiometabolic blood profile which includes acid-base balance, degree of dehydration, mineral balance, metabolic expenditure and gas exchange are also useful clinical elements for properly assessing neonatal viability. Neonatal survival is notably reduced when the blood pH falls below 7.0, lactate rises above 90 mg/dl, bicarbonate drops below 10 mmol/l, or the pCO_2 increases above 110 mm/Hg. Blood calcium is also an excellent indicator of neonatal stress when used in combination with the other parameters mentioned above. Trembling due to an imbalance in the movement of calcium in muscle is also a factor involved in neonatal mortality. Neurological function in the newborn piglet could also be evaluated by adapting the Apgar score widely used in human perinatology. Neonates with scores lower than 6 in a 10 point scale have generally lower survival rates. The two most important indicators for this vitality score are breathing latency and bradycardia. If the neonate has apnoea for more than 5 min and the cardiac frequency does not increase to more than 110 beats per minute the prognosis for survival is rather poor.

Keywords: umbilical cord; neuronal damage; asphyxia; physiological profile; piglet viability

Contents

1. Introduction

Approximately 14% of all live born baby pigs have low postnatal viability due to decreased foetal blood flow and oxygen during birth (Mota-Rojas et al. 2005b, 2012) Low viability significantly increases the time required for a neonatal pig to find for the first time a teat in the dam and begin suckling colostrum. This problem may be related to neurological abnormalities caused by *in utero* asphyxia and decreased cerebral blood flow in neonatal pigs (Trujillo-Ortega et al. 2007; Orozco-Gregorio et al. 2008; Mota-Rojas et al. 2012).

Neonatal mortality is a serious problem for the pork industry worldwide where 2% to 9% of stillborns result from perinatal asphyxia (Vallet et al. 2010). The proportion of live born and stillborn piglets often impact on the economic outcome of the farm (Houska et al. 2010). Numerous predisposing factors are incriminated in this situation; some are directly related to sows while others are attributed to the piglets themselves (van Dijk et al. 2005, 2006; Alonso-Spilsbury et al. 2007). The most important maternal factors involved in neonatal viability are number of parturitions, mothering skills, diameter of the pelvic canal, abnormal hormonal regulation and uterine physiology, prolonged expulsion periods secondary uterine inertia (uterine atony), maternal hypoxia resulting from temperatures above 35 °C in the delivery room, among many others (Alonso-Spilsbury et al. 2005, 2007; Odehnalova et al. 2008; Olmos-Hernandez et al. 2008).

The most common piglet factors involved in neonatal mortality and reduced viability are the birth weights in which larger neonates are more likely to die; the birth order in which the last ones to be born are more likely to die; umbilical cord lesions which depending on severity can cause foetal hypoxia; and the degree of meconium staining of the skin and meconium aspiration which reflect intrauterine foetal distress (Mota-Rojas et al. 2002, 2007; Trujillo-Ortega et al. 2007). Piglet sex is another risk factor since male piglets are more likely to die than females (Trujillo-Ortega et al. 2011).

It is of vital importance to assess the performance of newborn piglets, a task for which veterinarians should be well prepared to do or to supervise. For example, the degree of meconium staining is a key element, but one that must go hand-in-hand with other clinical findings such as asphyxia, hypoxaemia and acidosis (Castro-Najera et al. 2006; Mota-

Rojas et al. 2006). The morphology and vascular alterations in the umbilical cord are also key predictors for neonatal survival. Piglets born with severe umbilical cord lesions are less likely to survive or have normal postnatal performance (Mota-Rojas et al. 2005c). Therefore, it is imperative to evaluate umbilical cords and to pay particular attention to evidence of ruptured, torn or haemorrhagic cords since all these lesions are clearly associated with a piglet's performance at birth (Mota-Rojas et al. 2002, 2008).

The physiometabolic profile has come to be used by some researchers to properly evaluate neonatal survival. However, this profile should be established only in an integrated fashion using clinical and laboratory findings together as routinely done in human perinatology. The physiometabolic profile includes metabolic, mineral, acid-base and gas exchange levels in blood which relate well to asphyxia (Nodwell et al. 2005; Villanueva-Garcia and Mota-Rojas 2008, 2009; Gonzalez-Lozano et al. 2010, 2012; Orozco-Gregorio et al. 2010; Trujillo-Ortega et al. 2011). Though somewhat outdated and subjective, a vitality scale such as the Apgar score used in humans is another important clinical tool for assessing animal neonates (Zaleski and Hacker 1993; Veronesi et al. 2009).

In the following sections, we review how clinical evaluation and laboratory analyses should be integrated to enable veterinarians or animal scientists to properly assess the welfare of neonatal piglets. This combined approach allows for more accurate prognoses of viability at birth and gives better options for treatment and control.

2. How do we evaluate umbilical cord morphology and uterine dynamics?

Human foetuses and newborn babies are particularly vulnerable to asphyxia during the time of labour or shortly thereafter. When asphyxia begins *in utero*, either before or during labour, blood flow to the placenta and umbilical cord is compromised causing a myriad of physiological abnormalities in the neonate (Balchin et al. 2011). Due to their notoriously low tolerance for anoxia (caused by asphyxia), porcine foetuses commonly suffer irreversible brain damage when the umbilical cord is ruptured 5 min prior to delivery (Alonso-Spilsbury et al. 2005). A ruptured umbilical cord seriously compromises the normal blood flow between the
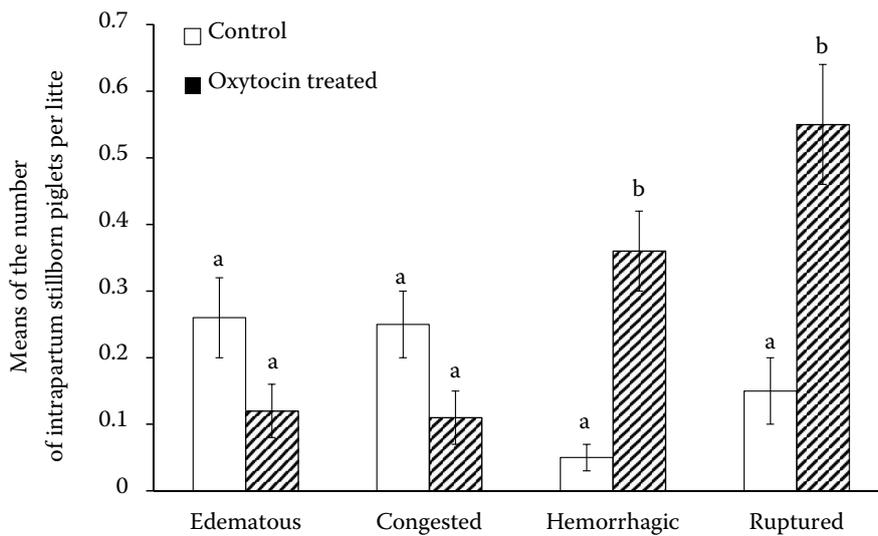
Figure 1. Umbilical cord abnormalities of intrapartum stillborn piglets (control and oxytocin treated sows)

a, b = different letters between bars indicate statistical differences to the Student t-test; taken from Mota-Rojas et al. (2002)

dam and the foetus (Curtis 1974). The relationship between umbilical cord changes and perinatal viability or mortality in piglets has been intensively investigated during the last few years. The results of these investigations have shown that umbilical cord morphology and circulatory abnormalities are excellent predictors of welfare for neonatal piglets born to sows given birth accelerators (Mota-Rojas et al. 2002). It has been reported that even at commercially recommended doses, oxytocin caused a significant increase ($P < 0.05$) of 300% in the number of ruptured cords, and resulted in seven times more haemorrhagic cords ($P < 0.05$), as shown in Figure 1.

Other studies have demonstrated that myometrial activity notably influences the circulatory physiolo-gy and survival of porcine neonates (Mota-Rojas et al. 2007). For instance, intense uterine contractions can cause a significant decrease in placental blood flow and gas exchange leading to foetal hypoxia and other potentially deleterious effects for the new-born piglet (Tucker and Hauth 1990). However, most deaths at birth occur when the umbilical cord carrying oxygenated blood becomes twisted or ruptures causing severe hypoxemia (Provis and Moyniham 1999). It should be noted nonetheless, that this point of view on the pathophysiology of neonatal hypoxaemia has been challenged by some researchers (Herpin et al. 1996) who postulate that prolonged or intermittent asphyxia *in utero* or during the time of delivery does not necessarily lead to intrapartum stillbirths. In a recent study, Mota-
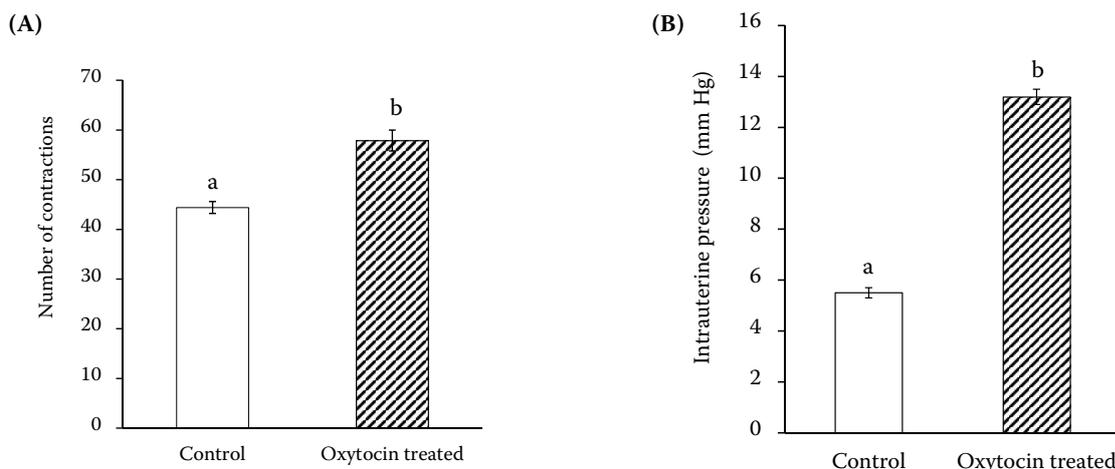

Figure 2. Mean and standard error of the intrauterine monitoring, frequency of myometrial contractions (**A**) and intensity (intrauterine pressure) (**B**) evaluated by means of uterine transducer (control and oxytocin-treated sows)

a, b = different superscripts express significant differences ($P < 0.01$) between groups in the Student t-test; taken from Mota-Rojas et al. (2005a)

Rojas et al. (2005a) showed why birth accelerators administered to sows increase umbilical cord abnormalities at birth. These investigators reported a significant increase ($P < 0.01$) in the number of uterine contractions in farrowing sows treated with oxytocin at a dosage of 1 UI/kg of the live weight of the sow (Figure 2A). In addition, the intensity of uterine contractions (intrauterine pressure) increased significantly ($P < 0.01$), from 5.5 in the control group of sows to 13.2 mm/Hg in the sows treated with oxytocin (Figure 2B). These changes in uterine physiology explain why the incidence of umbilical cord abnormalities rises when sows are treated with birth accelerators to reduce the time of foetal expulsion. Paradoxically, a reduced time of expulsion by oxytocin increases the likelihood of ruptured umbilical cords similar to what occurs in sows with dystocia (Gutierrez et al. 2011).

The relationship between umbilical cord injury, hypoxia and expulsion of meconium has been recognized for many years in human and veterinary perinatology (Miller 1977; Wiswell and Bent 1993). More recently, Mota-Rojas et al. (2005a) studied the effect of oxytocin on umbilical cords and meconium staining in pigs born to sows treated with oxytocin. The results of this work proved a relationship between the degree of meconium staining in the skin and the number of piglets born with ruptured umbilical cords. Live piglets stained strongly with meconium while 43 piglets were born with ruptured umbilical cords to the group of sows treated with oxytocin, compared to just four piglets in the control group.

In another study carried out by Mota-Rojas et al. (2005c) it was shown that the number of piglets with ruptured umbilical cords increases significantly ($P < 0.003$) from 37 to 76 neonates when oxytocin is administered, but decreases significantly to just 9.4 piglets when a uterine contraction modulator called vetrabutine hydrochloride is used (Gonzalez-Lozano et al. 2010, 2012). Other studies conducted by Gonzalez-Lozano et al. (2009a,b) also showed that sows with difficult births give birth to piglets with more umbilical abnormalities as compared to a sow with normal parturition

3. What is meconium staining and meconium aspiration syndrome?

It has been known for many years that intrauterine hypoxia in humans and animals results in meconium staining of the amniotic fluid and neonatal skin (Miller 1977). The basic mechanism starts with *in utero* hypoxia which increases intestinal peristalsis and relaxes the tone of the anal sphincter resulting in the expulsion of meconium into the amniotic fluid. Once in the amniotic fluid, the meconium stains the skin giving it a typical yellow discoloration. If hypoxia persists, foetuses gasp with open glottis and inhale meconium-contaminated amniotic fluid (Randall and Penny 1967; Alonso-Spilsbury et al. 2005). Pigs, like other mammals that suffered lack of oxygen prior to or during birth are often born covered by meconium, some of which could be present in the oropharynx or airways (Lopez et al. 1994; Mota-Rojas et al. 2002, 2012; Lopez 2007).

Meconium is a viscous, greenish sterile substance present in the foetal intestine. It is composed of a mixture of gastrointestinal secretions, bile, pancreatic juice, mucus, cellular detritus, amniotic fluid, vernix caseosa, lanugo and blood (Rapoport and Buchanan 1950; Srinivasan and Vidyasagar 1999). Chemical analyses have shown that mucopolysaccharides constitute approximately 80% of the dry weight of meconium. Interestingly, foetuses with serious hepatic abnormalities which impair bile secretion are born with white meconium. A recent study suggests that the release of meconium in humans is not only related to hypoxia but that also factors such as race and gestational age play a major part (Balchin et al. 2011). Future studies should investigate if gestational age and breed in swine are also implicated in meconium expulsion.

Meconium aspiration syndrome (MAS) is a significant cause of morbidity and mortality in the perinatal period and has been implicated in the pathogenesis of airway dysfunction in foetuses and neonates (Amir et al. 1999). During foetal asphyxia, the redistribution of blood from the intestine into the vital organs such as heart and brain causes an increase in intestinal peristalsis and relaxation of the anal sphincter, and together these two hypoxia-induced responses ultimately lead to the release of meconium into the amniotic fluid. If foetal anoxia persists, increased respiratory movements with an opened glottis result in the aspiration of amniotic fluid contaminated with meconium into the lungs (Martinez-Burnes et al. 2002, 2003; Castro-Najera et al. 2006; Mota-Rojas et al. 2006). For these reasons, the passage of meconium into the amniotic fluid is generally regarded as a good indicator of foetal distress. It is also known that not all infants

with foetal distress and passage of meconium into the amniotic fluid develop MAS. As one study recently pointed out, only severe airway obstruction by meconium results in perinatal death (Castro-Najera et al. 2006).

In human perinatology it is well recognized that those that survive meconium aspiration may have some long-lasting postnatal consequences (Swaminathan et al. 1989). On the other hand, there is paucity of information regarding the outcome and performance of animal neonates surviving meconium aspiration and some of the information fortuitously originate from swine models for human diseases (Aaltonen et al. 2003) Studies in human perinatology report long-term sequelae in some babies surviving intrapartum meconium aspiration. The most notable ones are persistent foetal circulation in which there is a delay closure of foetal heart communication (ductus arteriosus and foramen ovale), and hyper-reactive airways disease in which affected children suffer an asthma-like syndrome (Swaminathan et al. 1989). Much remains to be done in veteri-nary medicine to elucidate the long-term sequelae in animals surviving meconium aspiration. What we know from experimental studies is that meconium or amniotic fluid aspiration causes a mild but long term alveolitis characterised by early alveolitis which is followed by the formation of microscopic granu-lomas (Martinez-Burnes et al. 2002, 2003). It should be emphasised that meconium aspiration does not cause sufficient lung damage to kill an animal but rather the animal dies because of systemic physi-ological abnormalities such as acidosis, hypertension and hypoxaemia (Lopez 2007).

While the morbidity and mortality caused by meconium aspiration has been recognised for many years in human foetuses, only recently has this problem been investigated in domestic and labora-tory animals (Martinez-Burnes et al. 2001, 2002). Meconium-stained amniotic fluid is an indicator of prenatal asphyxia and postnatal viability in pigs (Castro-Najera et al. 2006). Earlier reports indicate the same problem occurs in calves, lambs and foals and it is postulated that animals born with meconi-um-stained skin are generally weaker and more sus-
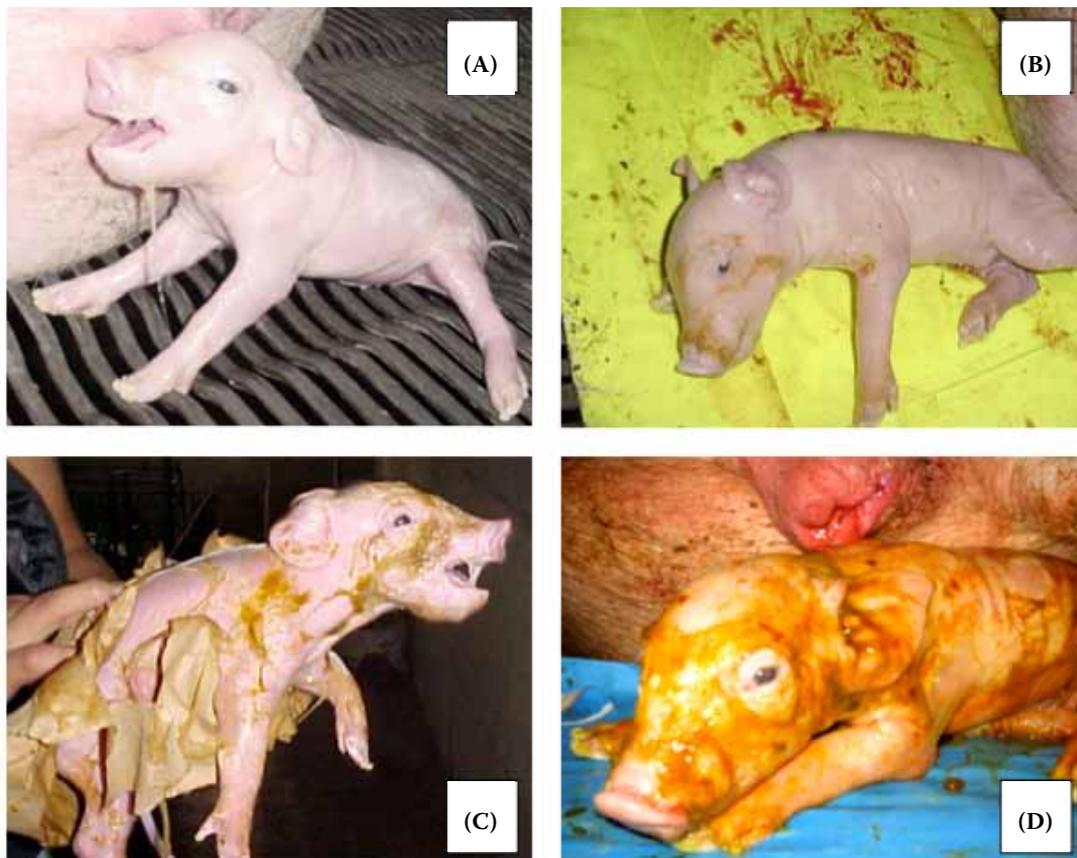

Figure 3. Degree of meconium staining of skin in newborn piglets; normal (without staining) (**A**), light (**B**), moderate (**C**), severe (**D**)

ceptible to perinatal death. The strong relationship between acidosis, meconium aspiration and failure of passive transfer of colostrum has been postulated in calves but many questions regarding the pathogenesis of this syndrome remain unanswered (Lopez and Bildfell 1992). Future research should perhaps investigate the relationship between acidosis, meconium aspiration and failure of passive transfer of colostrum and postnatal susceptibility of infection in pigs.

Recent studies conducted in puppies and foals showed a high incidence of neonates born stained with meconium and with meconium aspiration in the lungs. It has been suggested that the presence of meconium could be used as an indicator of foetal hypoxia (Mendoza et al. 2008; Santiago et al. 2008).

A severe degree of meconium staining of the skin has been associated with low or failing scores on the neonatal vitality scale and this occurs regularly when the umbilical cord has been ruptured (Mota-Rojas et al. 2002, 2005a,b; Trujillo-Ortega et al. 2011). Meconium staining during farrowing correlates well with the rupture of umbilical cord in piglets born live from sows treated with oxytocin. In one study there was a positive relationship ($r = 0.58$) between the number of live born piglets with ruptured umbilical cords and the magnitude of meconium staining; thus it is suggested that the damage to the umbilical cord damage induced by oxytocin causes a sufficiently serious asphyxia that reflects in a higher degree of meconium staining in the neonatal skin (Mota-Rojas et al. 2005a). Intrapartum asphyxia is also corroborated by the greater frequency of cyanosis and pale skin in piglets born to sows treated with oxytocin. Figure 3 illustrates the different degrees of meconium staining on the skin of pigs at birth.

4. How do we determine the physiometabolic profile?

The proper identification of acid-base alterations through the interpretation of blood gas data facilitates the clinical approach to the patient, helps in making a more accurate diagnosis and provides a better choice for appropriate treatment (Nodwell et al. 2005). Recently, the assessment of animal welfare has been made easier by the application of blood analysis and particularly through the use of novel technologies such as Arterial Blood Gas (ABG) equipment (Sanchez-Aparicio et al. 2008, 2009; Villanueva-Garcia and Mota-Rojas 2008; Becerril-Herrera et al. 2009, 2010; Trujillo-Ortega et al. 2011). The ABG equipment assesses the transport of blood gases and gives an index of gas exchange between the alveolar air and blood plasma. This is done in two stages: first, the incorporation of oxygen and the excretion of carbonic anhydride; and second, the acid-base balance which analyses the animal´s metabolic status (Villanueva-Garcia et al. 2008; Villanueva-Garcia and Mota-Rojas 2008, 2011).

For human and animal neonates, blood analyses provide important information by estimating the degree of oxygenation, biophysical profiles and the acid-base balance. In addition, it allows the detection of alterations in pO_2 concentrations, O_2 saturation (SaO_2), pCO_2, lactate, bicarbonate, hematocrit and pH (Low et al. 1994; da Silva et al. 2000).

The analysis of blood ABG has also been used in other animal species. For instances, ABG was used in one study to examine the effect of birth order in puppies delivered by caesarean section (Crissiuma et al. 2010); in equine neonates ABG was used to study the relationship between blood lactate and neonatal diseases such as sepsis, septic and haemorrhagic shock, haematomas, trauma and prematurity or immaturity (Castagnetti et al. 2010).

In pigs blood ABG has also been used to establish the physiometabolic profile of piglets born with and without intrapartum asphyxia in various conditions such as controlled climates and feeding (Orozco-Gregorio et al. 2008) as well as in pigs raised in a natural farm environment or in laboratory facilities (Trujillo-Ortega et al. 2007; Sanchez-Aparicio et al. 2008; Sanchez-Aparicio et al. 2009; Gonzalez-Lozano et al. 2009a,b). ABG has also been used to validate an experimental pig model that simulates *in utero* asphyxia (van Dijk et al. 2006, 2008). In this study, asphyxia was simulated by experimentally occluding the umbilical cords of neonates born by caesarean section. A study conducted by Gonzalez-Lozano et al. (2009a) showed that glucose, calcium and lactate levels rise significantly ($P < 0.01$) in piglets born to sows with dystocia (Figure 4).

Other researchers have used ABG analysis to assess therapies geared to re-establishing the respiratory process providing inhaled oxygen (Zaleski and Hacker 1993; Herpin et al. 2001; Tollofsrud et al. 2001). Vallet et al. (2010) assessed the expulsion intervals and concentrations of steroids in newborn piglets and their relation to stillborns and concluded that (1) birth intervals greater than 1 h are associated with an increased number of
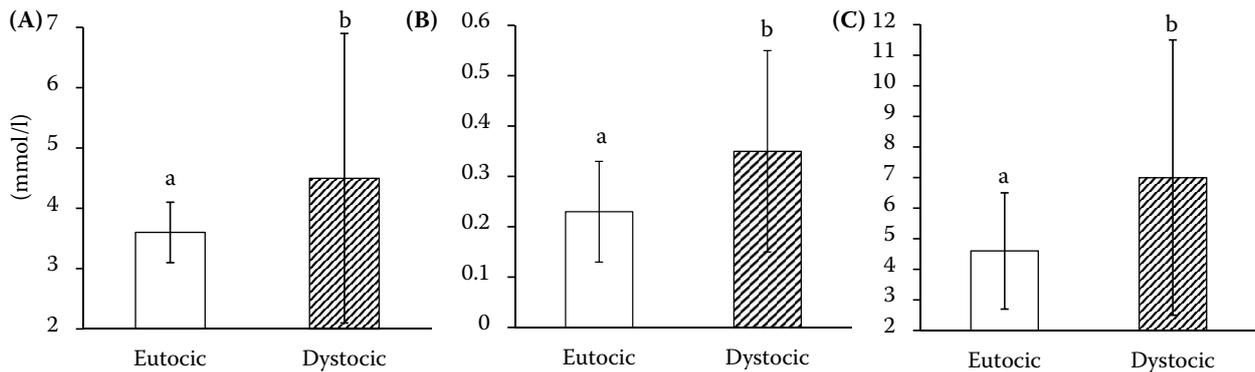
Figure 4. Glucose (**A**), calcium (**B**), and lactate (**C**) levels in live-born piglets of dystocic and eutocic sows; data represent mean ± standard deviation

a, b = ($P < 0.05$); taken from Gonzalez-Lozano et al. (2009a)

stillbirths; (2) larger litter size reduces birth intervals; (3) the last piglet in the litter has both a prolonged birth interval and an increased risk of stillbirth; (4) plasma progesterone before farrowing does not influence birth intervals or stillbirths; and, (5) plasma estradiol does not influence birth interval or stillbirths, despite its positive relation to litter size. The authors concluded that a better understanding of the effects of litter size and the proportion of the litter that is farrowed on birth intervals could be used in the future to decrease stillbirths in piglets.

On the other hand, a study by Trujillo-Ortega et al. (2007) on physiometabolic blood profiles showed that intrapartum deaths were associated with blood pH levels of less than 6.90. In addition, blood $PaCO_2$ and lactate are also important predictors for viability in piglets. All these findings further support the current view that the severity of metabolic acidosis secondary to hypoxia is a good indicator of intrapartum death (Jezkova and Smrckova 1990). Furthermore, lactate levels and birth weight are positively associated with latency to first udder contact. In one study, glucose levels were more than two times higher in piglets who died intrapartum than in those with mild-to-no intrapartum asphyxia or even than neonates with severe asphyxia, whereas the difference between the means of the two latter groups was 4 mg/dl (Trujillo-Ortega et al. 2007).

5. How do we establish the vitality score and measure latency to first udder contact?

Newborn piglets are regularly assessed using a score that measures their vitality within one min-

ute after birth based on the following variables: heart rate (beats per min) [less than 120 (bradycardia), between 121 and 160 (normal), and more than 161 (tachycardia)]; latency to breathing (in min) (interval between birth and first breath more than 1 min, between 16 s and 1 min, and less than 15 s); colour of the skin on the snout (pale, pink or cyanotic); latency to standing, measured as the interval between birth and the first time the neonate stands on all four legs (classified as more than 5 min, between 1 and 5 min, and less than 1 min); and skin staining with meconium (severe, mild, or absent) (Zaleski and Hacker 1993; Mota-Rojas et al. 2005a,b,c). Each one of these variables is assigned a score between 0 (least favourable) and two (most favourable), as well as an overall result ranging from one to 10 that was obtained for each neonate piglet. A score below six indicates a failing grade on the vitality scale test. The heart rate is measured with a stethoscope. The first breath at birth is defined as a thoracic movement in the thoracic area observed accompanying the exhalation of air. The standing time is defined as the minutes it takes for a piglet to stand on all four feet and it is typically measured with a stop-watch.

Two additional biological factors affecting neonatal viability in piglets are the birth weight and nutritional status. For this reason, iron supplementation is a key element in piglet performance during the first week of life (Svoboda et al. 2004, 2007). Several studies suggest that low neonate birth weight is negatively associated with perinatal survival (England 1974; Caceres et al. 2001; Herpin et al. 2002; Mota-Rojas et al. 2008). In one of these studies it was found that only 28% of piglets with birth weights below 1100 g reached the age of seven
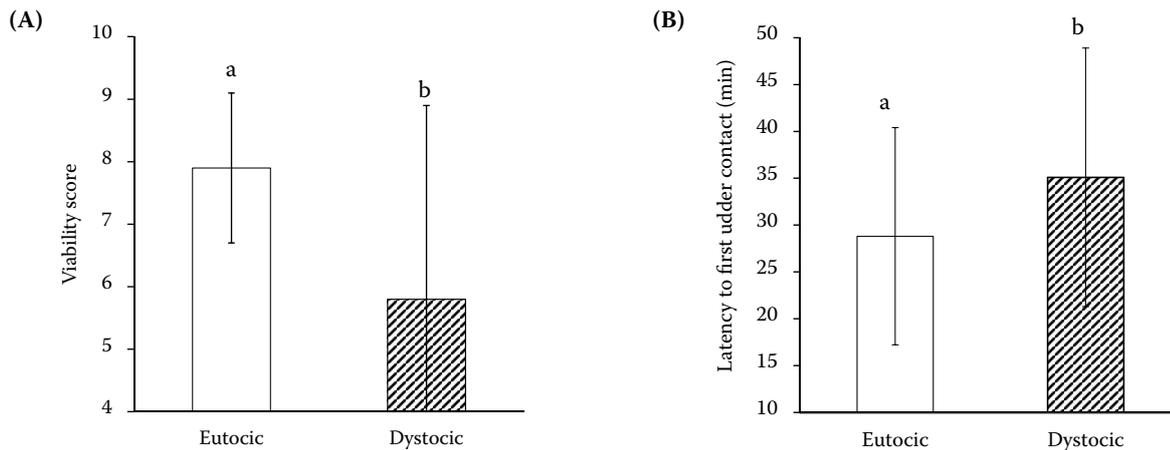
(A)

(B)

Figure 5. Outcome of piglets born to eutocic and dystocic sows, mean viability score (**A**) and latency to first udder contact (**B**). 2-by-k X2 test: followed by a post-hoc 2-by-2 X2 test ($P < 0.05$); data represent mean ± standard deviation; taken from Gonzalez-Lozano et al. (2009a)

days (Marchant et al. 2000). Similarly, Herpin et al. (1996) reported that only 36% of neonates with weights of 1051 g ± 83 g had high viability scores, while piglets weighing 1313 g ± 41, 79 g achieved higher scores on the viability scale ($P < 0.05$). In this study, a low level of viability was associated with the degree of asphyxia suffered during birth, while the percentage of animals that died during the first 10 days postpartum was higher in the group of piglets with lower weights, compared to that seen in the group of heavier neonates (43% vs. 19.4%) ($P < 0.06$). On the other hand, Baxter et al. (2008) pointed out that weight at birth alone is not a reliable indicator for prenatal survival suggesting that body mass should also be taken into account.

In a large study conducted with 230 piglets, Trujillo-Ortega et al. (2007) reported that 8.3% ($n = 19$) of foetuses died intrapartum, 21.7% ($n = 50$) were born with moderate-to-severe intrapartum hypoxia, and 70% ($n = 161$) were born with mild or no evidence of intrapartum distress. Piglets born with few or no signs of intrapartum asphyxia weighed approximately 240 g less than those born with hypoxia or those that were stillborn ($P < 0.0001$). In the same study, viability scores were approximately 3 points lower in the piglets that survived intrapartum hypoxia than in those born with minimal or no evidence of intrapartum asphyxia (control group). Latency to first udder contact was two times greater in the piglets with intrapartum asphyxia than in controls ($P < 0.0001$), while latency to first udder contact was inversely related to viability scores ($r = 0.75$; $P < 0.0001$); i.e., longer

latency times were seen in piglets with lower viability scores.

Dystocia has been known to increase the frequency of weak animals at birth. Studies conducted by Gonzalez-Lozano et al. (2009a), indicate that piglets born to sows with difficult parturition (dystocia) had longer latency to first udder contact and low vitality scores compared with those piglets born in normal (eutocic) delivery (Figure 5).

Neonates need to be able to feed shortly after birth and any factor impeding or delaying this function could result in reduced neonatal viability. Latency to first udder contact is assumed to reflect complex neurological functions in the newborn piglets, since it requires integrating – at least – the olfactory, visual and neuromuscular functions which are required for a well-oriented search for the maternal teat. Similarly, the viability scale includes the latency to standing, which may be altered by neurological impediments. Since these two parameters may not be the best indicators to assess neurological function in neonates, future studies should investigate other neurological functions as possible indicators of intrapartum asphyxia and low neonatal viability (Mota-Rojas et al. 2007; Trujillo-Ortega et al. 2007).

6. CONCLUSION

The degree of animal welfare in newborn piglets is critical in swine production and can be assessed using different pathophysiological ap-

proaches. As mentioned in this review, we believe that the principal diagnostic tools are the vitality scale, morphology of the umbilical cord, latency to first udder contact, the meconium aspiration syndrome, and a recently developed technique which allows the measurement of blood physiometabolic profiles. In conclusion, the extent of biochemical and metabolic alterations may explain the severity of neurological dysfunctions in newborn piglets that survive intrapartum asphyxia and, moreover, diminish animal welfare in the first week of life. Thus, the newborn piglet may provide a naturalistic model for the study of intrapartum asphyxia and the efficacy of neuroprotective strategies.

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Animal welfare in the newborn piglet: a review

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