THE PREMATURE NEWBORN CALF: HOW TO USE STEROIDS TO IMPROVE SURVIVAL?

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In all farm animals, premature birth may occur spontaneously or is required in certain states of acute disease for the pregnant mother or its fetus. Under such circumstances, it is crucial to be able to maximize the chances of survival for the neonate. Prenatal organ maturation is known to be stimulated by glucocorticoids and maternal dexamethasone treatment is used for both humans and cattle prior to premature delivery, albeit with variable success. We tested the hypothesis that an ACTH-stimulated increase in fetal glucocorticoid secretion is more effective in stimulating fetal organ maturation than treatment of the pregnant mother with synthetic glucocorticoids. At 248 d gestation (term = 275 d), intra-vascular catheters were implanted into 13 calf fetuses. After 4-5 days, ACTH-treated calves (250 mg $ACTH_{1-24}$ 4 times daily, n=6) and control calves (n=7) were exposed to 20 h of maternal dexamethasone treatment (DEX, 25 mg, i.m.) after which all calves were delivered by caesarean section. 24 h after birth the two groups of premature calves (ACTH+DEX and DEX alone) were killed and compared with a group of 24 h-old control calves delivered at full term (TERM).

	DEX	ACTH+DEX	TERM
Blood acidity (pH)	7.22±0.02	7.32±0.02*	7.32±0.01
Blood oxygen (vol%)	3.1±0.8	5.6±0.5*	7.2±1.2
Blood glucose (mM)	1.7±0.3	4.9±0.8*	4.2±0.4
Intestinal lactase (U/g)	23±2	31±2*	33±4
Intestinal ApA (U/g)	0.7±0.1	1.2±0.1*	0.9±0.1
Intestinal DPP IV (U/g)	4.3±0.3	5.6±0.5*	5.4±0.4
Rectal temperature (°C)	37.7±0.1	38.2±0.1*	38.6±0.2

At 2 h after birth, blood acidity, oxygen and glucose values were all improved in the ACTH+DEX group (Table, * $P<0.05$ relative to the DEX group), and the values were similar to those for TERM calves. Increases also occurred in the activities of lactase and 3 peptidases (ApN, ApA, DPP IV) as measured in the proximal small intestine. Finally, rectal temperature over the first 15 h after birth was higher in the ACTH+DEX than in the DEX group (Table). We conclude that prenatal ACTH treatment enhances maturation of the lungs and gut, and it improves glucose homeostasis and thermoregulation in premature neonates born by DEX-treated mothers. A pulsatile pattern of fetal glucocorticoid secretion over a longer period (in response to ACTH-treatment) is more effective in stimulating prenatal organ development than a short-term exposure to pharmacological levels of glucocorticoids administered via the pregnant mother. This method might prove useful to enhance the survival characteristics of particularly valuable newborns subjected to premature delivery.