A SUMMARY OF STUDIES CONCERNING THE EFFECTS OF PROSTAGLANDINS SYNTHESIS INHIBITION, PGE2 AND PGF2α, ON THE EXPULSION OF PLACENTA AND MYOMETRIAL ACTIVITY IN THE COW

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It is known that prostaglandins and thromboxanes regulate physiological and pathological processes in several organs by changing microcirculation, softening of connective tissue and muscle activity. Since placentation in the cow is provoked by similar mechanisms, it was decided to investigate the influence of prostanooids upon that process in order to achieve a more rational approach for the explanation of the etiology of placental retention (PR) in the cow.

This influence was assessed in two trials by (1) prostaglandin biosynthesis inhibition and (2) experimental induced imbalance in PGE2 and PGF2α, during the post partum.

Prostaglandin biosynthesis inhibition was carried out in five dairy cows by giving them an aspirin-like drug, the lysin acetilsalicylate (LAS), a known inhibitor of the cyclooxygenase, a key enzyme in prostaglandin synthesis. These cows received 10g. of LAS intravenously starting five minutes after calving and then every six hours afterwards for 24 hours. Five other cows were used as placebo. Placenta was retained longer than 24 hours in all LAS treated cows compared to only one cow in the placebo group (P<0.001). The other four cows released their placenta within the first six hours calving.

The results presented above suggest that prostaglandins play a key role on the expulsion of the afterbirth in the cow. To determine the individual role of PGE2 and PGF2α either on the separation/expulsion process of the placenta and on myometrial activity another set of experiments was performed. In these experiments 23 dairy cows were divided in four groups as follows: I -7 cows were used as placebo; II -5 cows received 20g of LAS Intravenously, III -5 cows received 5mg of PGE2 by intrauterine route together with the 20g of LAS intravenously; IV -6 cows received 25mg of PGF2α intramuscularly together with the 20g of LAS intravenously. All these treatments were administered twice, at five minutes and five and a half hours after calving. Myometrial activity was recorded in all but two cows for one hour beginning at 6 hours after calving and it was assessed by intrauterine pressure (IUP). Placental retention defined as lack of expulsion of the placenta during the first 12 hours after calving, was significantly higher in groups II and III (P<0.01) than on the placebo group. Considering cows having a PR later than 48 hours post - partum, it was shown that group III, in which all cows presented this condition, was significantly different from groups II and IV (P<0.05). Results from IUP records were compared among groups by analysis of variance being the amplitude of the uterine contractions the only parameter showing significant differences (P<0.05). By the least significant difference, amplitude values from groups II and IV were significantly lower than those of placebo (P<0.05).

The results above confirmed our previous observation that ciclooxigenase inhibition early in the post - partum induces PR in cows, which was shown to be accompanied by a significant reduction of the amplitude of the uterine contractions. They have also shown that PGE2 potentiates the action of LAS upon the duration of placental release and my also exert a negative effect on the separation process at the placento membrane level, since
no significant reduction of myometrial activity was evident in this group. Finally PGF$_2\alpha$ seemed to favor placental expulsion, since 50 per cent of the treated cows, which were expected to retain their placentas by the action of LAS, released them within 12 hours *post-partum*. This effect is also suggested to be exerted within placentomes, since FGF$_2\alpha$ couldn't prevent the effect of LAS in reducing the amplitude of the contractions.

Since PR was shown not to be necessarily accompanied by a significant reduction of myometrial activity, other local prostaglandin – dependent mechanisms in placentomes and placentas such as softening of connective tissue and changes in placenta vascular bed, seem to be of great responsibility for the normal detachment process of the afterbirth in cattle.

It is suggested that PR in cows may be originated by a local PGE$_2$ and PGF$_2\alpha$ imbalance within the placentome, favoring PGE$_2$ synthesis and a lack in PGF$_2\alpha$. This is in agreement with results of Leidl et al. (3) who have shown that cows with PR synthesize less PGF$_2\alpha$ from their fetal cotyledons than normal cows, and Williams et al. (4) who have shown from *in vitro* studies that fetal cotyledons seem to possess a great 9-ceto-reductase activity, an enzyme known to be able to convert PGF$_2\alpha$ into PGE$_2$.


