CHANGES IN PLASMA OXYTOCIN, PROSTAGLANDIN E₁, AND
13,14-DIHYDRO-15-KETO-PROSTAGLANDIN F₂α DURING
LABOR INDUCED BY PROSTAGLANDIN E₂
OR F₂α AND SPONTANEOUS LABOR

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Synopsis
To observe the changes in endogenous oxytocics during spontaneous and induced labor, the plasma concentrations of oxytocin, prostaglandin E₁ (PGE₁) and 13,14-dihydro-15-keto-prostaglandin F₂α (PGFM) were measured during labor in 9 cases of spontaneous labor (group 1), 10 of PGE₁-induced labor (group 2), and 7 of PGF₂α-induced labor (group 3). Unextracted samples were used for radioimmunoassay of oxytocin. PGE and PGF were extracted and separated for radioimmunoassays of PGE, and PGFM. Although oxytocin levels in groups 1 and 3 did not change during labor or slightly increased toward delivery, those in group 2 decreased as labor progressed. The mean oxytocin in group 2 was significantly lower at the times of established labor (15.3±3.2μU/ml, mean±SE) and crowning of the fetal head (10.8±3.0μU/ml) than before labor (52.7±14.8μU/ml). Plasma PGE levels in groups 1 and 3 were low and did not change during labor. Plasma PGFM levels in groups 1 and 2 gradually rose toward delivery. These results suggest that exogenous PGE suppresses oxytocin secretion during labor and stimulates endogenous PGF₂α production, that endogenous PGE may not play an important role in the progress of spontaneous and PGF₂α-induced labor, and that endogenous PGF₂α may participate in the promotion of all kinds of labor.

Key words: Oxytocin, Prostaglandin E₁, Prostaglandin E₂, Prostaglandin F₂α, Induction of labor

Introduction
Endogenous hormones which potently contract myometrium are oxytocin and prostaglandins E₁, E₂ and F₂α. Therefore, it may further our understanding on the mechanism of labor to observe changes in plasma concentrations of these hormones during labor. Although several investigators have evaluated plasma levels of these hormones, there are few reports in which the interaction between endogenous and exogenous oxytocic hormones is investigated during the course of labor. We have previously found two types in human labor, oxytocin-dominant and oxytocin-subordinate types. Most spontaneous labors belonged to the former type, whereas most labors induced by amniotomy, prostaglandin F₂α, and prostaglandin E₂ belonged to the latter type. It was supposed that in the oxytocin-subordinate type of labor, prostaglandins might initiate labor process. In order to confirm this contention, we have attempted to evaluate changes in the endogenous oxytocin, prostaglandin E₁ and prostaglandin F₂α release during the courses of labor induced by the administration of prostaglandin E₂ or F₂α. Here we report the alterations of plasma oxytocin, prostaglandin E₁ and metabolite of the prostaglandin F₂α (13,14-dihydro-15-keto-prostaglandin F₂α) during prostaglandin E₂-induced labor, prostaglandin F₂α-induced labor, and spontaneous labor.

Materials and Methods
Twenty six uncomplicated pregnant women who underwent delivery between 38 and 40 weeks of gestation were the subjects of this study.

Group 1 consisted of 9 subjects whose blood samples had been taken weekly after 38 weeks of pregnancy and whose labors and deliveries were occurred spontaneously.

Seventeen subjects were admitted to our hospital at term but before onset of labor for the purpose of planned induction of labor, and randomly divided into two groups. Group 2 consisted of 10 subjects whose labors were induced by prostaglandin E₂ (PGE₂) vaginal suppository. Group 3 consisted of 7 subjects whose labors were induced by prostaglan-
Table 1. Age, parity, duration of pregnancy, length of uterine fundus, and Bishop's pelvic score at the time of hospitalization in a total of 26 subjects

<table>
<thead>
<tr>
<th>Labor group*</th>
<th>No. of subjects</th>
<th>Age (yr)</th>
<th>Parity</th>
<th>Duration of pregnancy</th>
<th>Length of uterine fundus</th>
<th>Bishop score</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>Range</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>26.5</td>
<td>22–35</td>
<td>4</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>26.9</td>
<td>25–30</td>
<td>3</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>28.6</td>
<td>25–32</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

*Group 1, spontaneous labors.
Group 2, labors induced by prostaglandin E₂ vaginal suppository.
Group 3, labors induced by prostaglandin F₂α drip infusion.

Age, parity, duration of pregnancy, length of uterine fundus and Bishop score of each group at the time of admission are listed in Table 1.

Each subject in the induced labor groups (groups 2 and 3) received a sleeping drug at 2100 h on the day of admission, and soap enema at 0600 h next day. PGE₂ or PGF₂α administration was started at 0900 h.

In group 2, PGE₂ (G512, Ono Pharmaceutical Co., Japan) was given hourly by the intravaginal insertion of a 0.5mg PGE₂ suppository until labor was established. In group 3, PGF₂α (Prostalamon F, Ono Pharmaceutical Co., Japan) was administered by means of an infusion pump, starting at a rate of 4μg/min, and the dose was increased at a rate of 1.5μg/min for every 30 to 60 minutes until either regular contraction of the uterus was established or the dose reached 12μg/min. Blood samples were collected at 1500 h on the day of admission, at the onset of labor when irregular uterine contractions began to appear on the cardiotocograph after the start of PG administration, at the time of established labor when regular uterine contraction were shown, at the time of crowning of the fetal head and 2 hours after parturition. In group 1, the blood samples obtained within 7 days preceding the onset of labor was used as the samples “before labor”.

For the assay of plasma oxytocin, 5ml of blood sample was collected by venipuncture into prechilled 10ml evacuated blood collection tube (Vacutainer) that contained ethylenediaminetetraacetic acid (EDTA-2Na) (372μg), orthophenanthroline (5μg), and sodium citrate (250μg). All samples were immediately centrifuged at 3,000rpm for 15 minutes at 4°C. Plasma samples for oxytocin assay were incubated for 30 minutes at 56°C and centrifuged again in the same way as mentioned above. The supernatant was stored at -20°C until assay. For the assay of PGE₁ and metabolite of PGE₂α, another 5ml of blood sample was collected at the same time into the prechilled Vacutainer containing 0.1ml of heparin solution (Nobo Industry AS, 1,000 IU/ml). Plasma samples for PGs assay were stored at -20°C immediately after separation. PGs were assayed within one week of sample collection.

Plasma oxytocin concentration was measured by radioimmunoassay as previously described 11). Plasma PGE₁ concentration was measured by radioimmunoassay after extraction. First, plasma proteins were denatured by addition of 4 to 5 volumes of ethanol 48. After the addition of [5,6-³H(N)]-prostaglandin E₁ (about 1,000cpm, NEN, Boston, 28.1Ci/mmol) for recovery, 1ml of plasma sample was mixed with 4ml of 100% ethanol. After 30 minutes, the precipitate was taken away by centrifugation of 3,000rpm for 10 minutes. The precipitate was washed by 2ml of 80% ethanol and was centrifuged again. The supernatant was collected in a tube, and ethanol was evaporated under a stream of nitrogen. The resulting water “solution” (precipitation of hydrophobic compounds occurred) was diluted with water to 1ml to reduce the concentration of residual ethanol. At this time, the pH was adjusted to pH 8.0 to 8.5 with 3 droplets of 0.1 N NaOH to bring the PGs to the fully dissociated form (i.e., water soluble “soap”), and the nonpolar compounds, such as glycereids, were extracted with 3ml petroleum ether. The water phase was then exposed to 3.0ml of 3:3:1 ethyl acetate: isopropanol : 0.1 N HCl, apparent pH 5.8 and vortexed, and a mixture of 2.0ml of ethyl acetate and 3.0ml of water was added. After

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mixing, the two phases were separated by centrifugation (2,000rpm for 5 minutes at 4°C), 3ml of the 3.5ml organic phase were removed by aspiration and dried under a stream of nitrogen at 45°C. The dried materials were vortexed in 0.2ml of solvent 3 (benzene : ethyl acetate : methanol, 60 : 40; 10) and then 0.8ml of solvent 1 (benzene : ethyl acetate, 60 : 40). The combined organic extracts were applied to the columns made of 0.5g silicic acid slurry (Mallinckrodt, Inc. Kentacky, 100 mesh). The fraction developed with 12ml of the solvent 4 (benzene : ethyl acetate : methanol, 60 : 40 : 1) was taken as PGE fraction, dried under a stream of nitrogen at 45°C and dissolved in 1.0ml of gelatin phosphate buffered saline (GPBS), 0.01M phosphate buffer (pH 7.6) containing NaCl (0.15M) and gelatin (1mg/ml), 100μl of the aqueous solution was used for radioimmunoassay. Two hundred μl of the solution was transferred to 10ml vial for counting to permit calculation of recovery. The recovery of the tritiated standard was approximately 72%. Anti PGE₁ serum was donated by Ono Pharmaceutical Co., Japan. 0.1ml of diluted anti PGE₁ serum in GPBS (1:600), 0.1ml of 10,000cpm of ³H-PGE₁ in GPBS, were incubated for 1 hour at 37°C with either unlabeled PGE₁ (15~400pg) in 0.1ml of GPBS or 0.1ml of the unknown sample of PGE fraction dissolved in GPBS. Separation of antibody-bound from unbound ³H-PGE₁ was accomplished by adding 0.1ml of dextran-coated charcoal (Norit SX-3, Wako Pure Chemical Industries LTD, Japan, 25mg/ml: Dextran T40, Pharmacia Fine Chemicals AB, Sweden, 0.25mg/ml in GPBS). After centrifugation for 10 minutes at 4°C, 3,000 rpm, 250μl of the supernatants (containing antibody bound, ³H-PGE₁) were immediately decanted into 5ml of scintillation solution (ATOMLIGHT) and radioactivity was counted. Cross reactivities of the anti PGE₁ serum to PGE₂, PGF₁α, and PGF₂α were 8.1%, 26.8%, and 2.9%. At the dilution used, 1:600, anti PGE₁ serum bound 42% of ³H-PGE₁. The coefficients of variation for intra-assay and interassay of samples that contained 12.5 to 200pg of PGE₁ per assay tube were both less than 15%. The sensitivity of the assay was 25pg.

Plasma concentrations of the PGF₂α metabolite (PGFM) were measured by radioimmunoassay after extraction based on the methods described by Satoh et al. and Mitchel et al.. 0.5ml of plasma sample was acidified to pH 3 with 1N HCl and extracted twice with 5ml of ethyl acetate. The dried extract was dissolved in 1ml of 100mM Tricine buffer (pH 8.0) including 0.025% of gelatin (GTBS), and 0.1ml sample of this solution was assayed in duplicate. Antibody to PGFM (13,14-dihydro-15-keto-PGF₂α) was kindly donated by Dr. Kirtom, Upjohn Ltd., Kalamazoo, U.S.A., and ³H-PGFM (13,14-dihydro-15-keto [5,6,8,9,11,12, 14(N)-³H]prostaglandin F₂α, 80Ci/mmol) was purchased from Amersham International plc Buckinghamshire, England. 0.1ml of dilute antibody of PGFM in GTBS (1:5,000) and 0.1ml of 5,000cpm of ³H-PGFM in GTBS were incubated for 3 hours at 4°C with either unlabeled PGFM (6.25~400pg) in 0.1ml of GTBS or 0.1ml of the sample. Bound and free ³H-PGFM were separated using dextran-coated charcoal as mentioned above. The supernatant (0.25ml) was counted for radioactivity. Extraction efficiency was determined in each assay. The mean recovery was 79.3%. At the dilution used, 1:5,000, anti PGFM antiserum bound 40% of ³H-PGFM. The intra-assay and interassay coefficients of variation for the samples containing 25 to 100pg of PGFM per assay tube were 9.7% and **Table 2. Duration of labor, infant birth weight, and Apgar score in a total of 17 subjects**

<table>
<thead>
<tr>
<th>Labor group*</th>
<th>No. of subjects</th>
<th>Duration of labor (Stages I+II, hours)</th>
<th>Infant birth weight (g)</th>
<th>Apgar score (one minute)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Range</td>
<td>Mean</td>
<td>Range</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>12.0</td>
<td>0.8~27.6</td>
<td>3,109</td>
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<tr>
<td>2</td>
<td>10</td>
<td>6.5</td>
<td>2.7~15.5</td>
<td>3,092</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>5.7</td>
<td>1.9~10.7</td>
<td>3,189</td>
</tr>
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</table>

*Group 1, spontaneous labors.
Group 2, labors induced by prostaglandin E₃ vaginal suppository.
Group 3, labors induced by prostaglandin F₃α drip infusion.
Table 3. Plasma concentrations of oxytocin, PGE$_1$ and PGFM during labor in the three groups

<table>
<thead>
<tr>
<th></th>
<th>Before labor</th>
<th>At the time of onset of labor</th>
<th>At the time of established labor</th>
<th>At the time of crowning of the fetal head</th>
<th>2 hours after parturition</th>
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<tbody>
<tr>
<td></td>
<td>Mean±SE</td>
<td>Mean±SE</td>
<td>Mean±SE</td>
<td>Mean±SE</td>
<td>Mean±SE</td>
</tr>
<tr>
<td>Oxytocin (µU/ml)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1 (n=9)</td>
<td>56.6±15.5</td>
<td>61.4±14.1</td>
<td>78.0±20.2</td>
<td>82.8±19.0</td>
<td>74.5±24.9</td>
</tr>
<tr>
<td>Group 2 (n=10)</td>
<td>52.7±14.8</td>
<td>23.5±7.7</td>
<td>15.3±3.2</td>
<td>10.8±2.0</td>
<td>33.4±13.1</td>
</tr>
<tr>
<td>Group 3 (n=7)</td>
<td>46.6±17.1</td>
<td>58.1±14.3</td>
<td>66.7±17.5</td>
<td>72.9±21.8</td>
<td>69.5±32.3</td>
</tr>
<tr>
<td>PGE$_1$ (ng/ml)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1 (n=9)</td>
<td>4.1±1.5</td>
<td>4.5±2.1</td>
<td>5.3±2.5</td>
<td>6.2±1.4</td>
<td>4.9±2.0</td>
</tr>
<tr>
<td>Group 2 (n=10)</td>
<td>4.5±0.9</td>
<td>33.7±6.3‡</td>
<td>47.4±6.7‡</td>
<td>31.4±11.8</td>
<td>17.1±6.4</td>
</tr>
<tr>
<td>Group 3 (n=7)</td>
<td>3.7±1.0</td>
<td>4.8±0.6</td>
<td>7.3±1.9</td>
<td>7.6±1.8</td>
<td>5.0±1.0</td>
</tr>
<tr>
<td>PGFM (pg/ml)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1 (n=9)</td>
<td>352±185</td>
<td>220±70</td>
<td>465±115</td>
<td>990±270</td>
<td>415±55</td>
</tr>
<tr>
<td>Group 2 (n=10)</td>
<td>725±286</td>
<td>872±180</td>
<td>1,337±59</td>
<td>1,945±75**</td>
<td>785±115</td>
</tr>
<tr>
<td>Group 3 (n=7)</td>
<td>913±370</td>
<td>&gt;2,000</td>
<td>&gt;2,000</td>
<td>&gt;2,000</td>
<td>&gt;2,000</td>
</tr>
</tbody>
</table>

*Group 1, spontaneous labors.
Group 2, labors induced by prostaglandin E$_6$ vaginal suppository.
Group 3, labors induced by prostaglandin F$_{1α}$ drip infusion.
**p<0.05 vs. the value before labor.
‡p<0.01 vs. the value before labor.

15%, respectively. In this assay, the cross-reactivity was 20% with 13,14-dihydro-15-keto-PGF$_{2α}$, 1% with C-16 urinary metabolites of PGF$_{2α}$, 0.5% with 13,14-dihydro-PGF$_{2α}$, and 0.1% with PGF$_{2α}$, respectively. The sensitivity of this assay was 12.5pg.

Statistical analysis was performed using Wilcoxon's rank test.

Results

The duration of labor, infant birth weight and one minute Apgar score in the three labor groups are listed in Table 2. There were no significant differences in these outcomes among the three labor groups.

The mean (±SE) of the plasma concentrations of oxytocin, PGE$_1$ and PGFM in the three groups were listed in Table 3.

Changes in the plasma concentrations of oxytocin during labor in the three groups are shown in Fig. 1. In the spontaneous labors (group 1) and in the labors induced by PGF$_{2α}$ (group 3), there was no significant difference in the mean level of oxytocin among various stages of labor. Oxytocin levels in the labors induced by PGE$_2$ (group 2) progressively decreased as labor advanced. However, they recovered to some extent after 2 hours of parturition. The oxytocin concentrations in group 2 were

Fig. 1. Changes in the plasma concentration of oxytocin in five stages during the course of labor in spontaneous labors (group 1), PGE$_2$-induced labors (group 2), and PGF$_{2α}$-induced labors (group 3). * p<0.05 vs. value before induction of labor.
Fig. 2. Changes in the plasma concentration of PGE₁ in five stages during the course of labor in spontaneous labors (group 1), PGE₁-induced labors (group 2), and PGF₂α-induced labors (group 3). ** p<0.01 vs. value before induction of labor.

Fig. 3. Changes in the plasma concentration of PGFM in five stages during the course of labor in spontaneous labors (group 1), PGE₁-induced labors (group 2), and PGF₂α-induced labors (group 3). p<0.05 vs. value before induction of labor.

significantly lower at the time of established labor and at the time of crowning of the fetal head than before induction of labor (p<0.05). The oxytocin levels were significantly lower at the times of onset of labor (p<0.05), at the time of established labor (p<0.01) and at the time of crowning of the head (p<0.01) in group 2 than in groups 1 and 3.

Changes in the plasma concentrations of PGE₁ during labor are shown in Fig. 2. PGE₁ levels in the spontaneous labors (group 1) and in the labors induced by PGF₂α (group 3) were low throughout labor process. PGE₁ levels in the labors induced by PGE₂ (group 2) abruptly increased from the stage before induction of labor to the stage of onset of labor and reached a maximum at the time of established labor and decreased thereafter.

Changes in the plasma concentrations of PGFM during labor are shown in Fig. 3. PGFM levels in the spontaneous labors (group 1) tended to increase toward the time of crowning of the head, although there was no significant difference in the mean level among various stages of labor. The pattern of PGFM in the labors induced by PGE₂ (group 2) was similar to that in the spontaneous labors, although PGFM levels were significantly higher in each stage after the onset of labor than those in the spontaneous labors. The mean PGFM level in group 2 was significantly higher at the time of crowning of the head than before labor (p<0.05). PGFM values decreased to the initial level after 2 hours of parturition in groups 1 and 2. PGFM levels in the labors induced by PGF₂α (group 3) were markedly high at the onset of labor in all subjects and remained there at least until 2 hours after parturition.

Discussion

Several investigators have studied changes in plasma concentrations of oxytocin, PGs and their
metabolites during labors. To the authors’ knowledge, however, there is no study except one \(^3\) in which plasma concentrations of oxytocin and the metabolite of PGF\(_{2\alpha}\) (PGFM) have been measured simultaneously in the course of labor. This is the first attempt to measure oxytocin, PGE\(_2\), and PGFM (13,14-dihydro-15-keto-prostaglandin F\(_{2\alpha}\)) simultaneously in the course of labors induced by prostaglandins, although we measured plasma concentration of PGE\(_2\), circulating levels of PGE metabolite may reflect PGE dynamics more accurately.

It was an unexpected finding that plasma concentrations of oxytocin in the PGE\(_2\)-induced labors decreased as labor progressed. In our previous study \(^1\), oxytocin levels in the PGE\(_2\)-induced labors were low compared with those in the spontaneous labors, and remained there throughout labor process, showing no significant change. However, oxytocin levels in the induced labors were as high as those of the spontaneous labors just before labor induction in the present study. The difference in the pretreatment value between this and previous studies may be due to the difference in the time of labor induction. As we showed in the previous study \(^1\), the plasma level of oxytocin increased as pregnancy advanced. We induced labor this time generally one or two weeks later compared to the previous study. Therefore, the different oxytocin responses to exogenous PGE\(_2\) between the previous and present studies may be accounted for by the different pretreatment oxytocin levels. The administration of PGE\(_2\) may suppress oxytocin secretion during labor when the pretreatment value of oxytocin is high.

Plasma concentrations of oxytocin tended to increase during the course of spontaneous labors and labors induced by PGF\(_{2\alpha}\) although they did not achieve statistical significance. This suggests that exogenous PGF\(_{2\alpha}\) slightly enhance the secretion of oxytocin. Oxytocin and PGF\(_{2\alpha}\) may work synergistically for initiation and promotion of labor in the PGF\(_{2\alpha}\)-induced labors.

PGE\(_2\) levels were low throughout labor process in the spontaneous labors and the PGF\(_{2\alpha}\)-induced labors when compared with those in the PGE\(_2\)-induced labors. Consequently, endogenous PGE\(_2\) may not be involved in the spontaneous labors or PGF\(_{2\alpha}\)-induced labors, though it cannot be ruled out that PGE\(_1\) levels in peripheral plasma does not reflect endogenous PGE\(_1\) production. The significant rise of PGE\(_1\) in the early stage of PGE\(_2\)-induced labors suggest that PGE\(_1\) as well as PGE\(_2\) may participate in the initiation of labor in this group, because PGE\(_1\) measured in the PGE\(_2\)-induced labors may reflect not only endogenous PGE\(_1\) but also in part PGE\(_2\) given exogenously, as anti PGE\(_1\) serum used had 8.1% of crossreactivity to PGE\(_2\). Plasma PGE\(_1\) level has scarcely been reported, whereas plasma PGE\(_2\) has been measured by some authors. Cornely et al. \(^3\) reported a significant rise of PGE\(_2\) at the time of delivery and after delivery in spontaneous labor. Plasma PGE\(_2\) could not be fully measured by our assay system for PGE\(_1\) although anti PGE\(_1\) serum showed 26.8% of crossreactivity to PGF\(_{2\alpha}\), the two PGs could be separated by column chromatography.

PGFM levels gradually increased toward delivery during labor in the PGE\(_2\)-induced group. This pattern in PGFM was similar to that in the spontaneous labors, although PGFM levels were almost two times higher at any stage of labor than those of the spontaneous labors. Lackritz et al. \(^9\) also reported a significant rise of PGFM in peripheral plasma in labors induced by PGE\(_2\) vaginal suppository. PGFM levels in the spontaneous labors were almost the same as those reported by Satoh et al. \(^1\) and Fuchs et al. \(^2\). These changes in PGFM suggest that endogenous PGF\(_{2\alpha}\) may take part in the progress of labor in PGE\(_2\)-induced labors as well as spontaneous labors.

Fuchs et al. \(^2\) proposed that oxytocin might be initiator and PGF\(_{2\alpha}\) might be promoter in spontaneous labor. However, changes of plasma oxytocin, PGE\(_1\), and PGFM observed in this study suggest that PGE may be initiator in the PGE\(_2\)-induced labors, although PGF\(_{2\alpha}\) may take part in the progress of labor. As plasma PGFM is thought to be a good index of prostaglandin F\(_{2\alpha}\) production \(^9\), exogenous PGE\(_2\) is considered to stimulate production of PGF\(_{2\alpha}\) in the utero-placental tissues in order to progress labor. Husslein et al. \(^7\) observed similar phenomenon in oxytocin-induced labors. They estimated plasma PGFM during oxytocin-induced labors and suggested that exogenous oxytocin stimulated PGF production in the pregnant uterus.

Since PGFM concentrations had already reached markedly high level at the time of onset of labor in
OXYTOCIN AND PROSTAGLANDINS DURING LABOR

the PGF$_{2\alpha}$-induced labors, it is supposed that exogenous PGF$_{2\alpha}$ can act as initiator as well as promoter of parturition in the PGF$_{2\alpha}$-induced labors.

In conclusion, human labors may be controlled by various endogenous oxytocic hormones. Endogenous oxytocin may be concerned with initiation and/or promotion of parturition in PGF$_{2\alpha}$-induced labors as well as spontaneous labors. Endogenous PGE may play a role in the initiation of labor in the PGE$_2$-induced labors. Endogenous PGF$_{2\alpha}$ may participate in the promotion of all kinds of labor.

References


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