The Physical and Emotional Effects of Postpartum Hormone Levels

by Ann Behnke

A woman's body is a complex series of changing hormone levels which constantly interact with one another to produce physical and sometimes emotional changes within her body. During pregnancy and postpartum a woman undergoes these alterations to an even greater degree, in addition to the major life adjustments which she is experiencing. There are four major hormones which drive this life-altering time: estrogen, progesterone, prolactin, and oxytocin. These hormones have physical effects, as well as psychological effects, which we are only beginning to recognize. Understanding their effects in pregnancy helps to clarify their effects in the postpartum period.

THE FOUR MAJOR HORMONES

Estrogen is a major female hormone and is a major pregnancy hormone. Estrogen drives the potential delivery of the fetus by controlling cervical ripening and increasing uterine receptors for oxytocin (Weiss 2000). Estrogen also affects the uterine myometrium by increasing uterine excitability (Gay 1978). During pregnancy, the major source of estrogen is the placenta and, to a much smaller extent, the fetus. As these products are delivered at birth, the woman's source of estrogen drops profoundly. Within three hours postpartum, the estrogen level drops to 10% of the prenatal value, and reaches its lowest value by day seven postpartum (Bobak and Jenson 1993). These estrogen levels are so low, they have been found to be equivalent to the estrogen levels of post-menopausal women.

Progesterone is a second major pregnancy hormone. Think of it as pro-gestation. One of its major roles is to quiet the uterus and prevent premature contractions and delivery (Gay 1978; Weiss 2000). These increased levels of progesterone during pregnancy also stop lactation from starting before the delivery of the infant (Riordan and Auerbach 1993). As with estrogen, progesterone levels decrease dramatically after birth and are undetectable by seventy-two hours after delivery. Progesterone levels are reestablished with the first menstrual cycle (Bobak and Jenson 1993).

Oxytocin increases during the expulsion stage of labor. Uterine receptors for oxytocin increase approximately thirty percent from a pre-pregnant uterus to a uterus ready for full-term labor. Oxytocin may also have some affect on cervical ripening at term (Weiss 2000). During postpartum, oxytocin continues to have an affect on contracting the uterus by contracting during the breastfeeding session and for up to twenty minutes after each feeding. It is interesting to note that uterine contractions continue even though oxytocin levels return to baseline within six minutes after nipple stimulation ends (Riordan and Auerbach 1993). Oxytocin also acts on the breast by eliciting the milk ejection response during breastfeeding.

Prolactin is another hormone associated with the breastfeeding process. During pregnancy, breast mass is increased because of the effects of prolactin. However, estrogen and progesterone also play a part in this process (Lawrence 1994). During postpartum, prolactin is the hormone of milk production. The levels of hormone rise and fall in proportion to nipple stimulation. During the first week after birth, prolactin levels fall about fifty percent but remain elevated throughout the course of lactation. For mothers who choose to bottle feed, prolactin levels return to normal by seven days postpartum. Prolactin hormone has been identified in the breast milk. This prolactin, absorbed by the infant, influences fluid, sodium, potassium, and calcium transport (Lawrence 1994). Prolactin has been found to delay ovulation by inhibiting ovarian response to FSH. It has been found to have a relaxing, calming effect; some women report feeling euphoric. Increased prolactin levels may prevent or ameliorate postpartum blues in breastfeeding women (Riordan and Auerbach 1993).

POSTPARTUM PHYSICAL RECOVERY

Many changes are involved in the physical recovery of the postpartum woman. Two of these, uterine involution and lactation, are mainly driven by hormones. It is both fascinating and exciting to see and understand the interplay of the hormones involved in these two changes.

At the time of the birth of the baby, the uterus weighs an average of 2.2 pounds. After the birth, the uterus can be felt by palpation just above the level of the umbilicus. The uterus then begins its dramatic return to its normal weight of two ounces, and it descents into the pelvic cavity at the rate of one cm/day. The fundus, or muscular top part of the uterus, can no longer be felt at continued on page 12
The effects of postpartum hormone levels

Ten days postpartum. At this time it has receded under the level of the symphysis pubis. It returns to its nonpregnant size by four to six weeks postpartum (Dickason, Silverman, and Schult 1994; Hilton 1992). This dramatic change is designed to save the mother's life, as the powerful uterine muscle clamps off the vascular network that has been feeding and nourishing the baby. Uterine atony, or the lack of uterine muscle contraction, can be caused by overdistention of the uterus from multiple gestation, a large fetus, an exhausted uterine muscle, or a full bladder. If the bladder is full, the uterine fundus will be palpable above the umbilicus and to the right in the mother's abdomen. Postpartum hemorrhage is the major cause of maternal morbidity and mortality in the postpartum period. Uterine involution is encouraged by the hormone oxytocin. Pharmacologically, oxytocin can be given through an IV or an IM injection immediately after birth. Optimally, a mother will be able to breastfeed immediately after birth and continue nursing on demand for natural bursts of oxytocin, which act to stimulate uterine involution. The mother should be warned that she might notice cramping during the breastfeeding session and for up to twenty minutes afterward for several days postpartum. If this becomes a problem, her care provider can recommend an appropriate analgesic to make the experience more comfortable.

Lactation is the second major hormonally-driven physiologic change in the postpartum woman. The rapid drop in progesterone and estrogen at delivery is the hormonal trigger for milk production (Lawrence 1994; Love and Lindsey 2000; Riordan and Auerbach 1993). Both hormones involved in lactation, prolactin and oxytocin, come from the pituitary gland. Oxytocin, from the posterior pituitary gland, is involved in milk ejection as well as uterine involution. The posterior pituitary gland has a large store of oxytocin (3,000-9,000 mU); however, only 50-100 mU are required to elicit the milk ejection reflex (Riordan and Auerbach 1993). In a breastfeeding woman who does not supplement infant feedings at all, increased oxytocin levels have been detected over time. By contrast, prolactin levels of women who breastfeed, with and without supplementation, have been found to decrease over time (Riordan and Auerbach 1993).

Prolactin is the hormone of milk production and comes from the anterior pituitary. Plasma prolactin levels rise and fall in proportion to the frequency, intensity and duration of nipple stimulation (Love and Lindsey 2000). Peak response to suckling is usually found between fifteen minutes and sixty minutes after the initiation of the session. As previously stated, although prolactin levels decrease through the postpartum period, they remain elevated in a lactating woman when compared to baseline levels of non-lactating women (Lawrence 1994). In a non-lactating woman, prolactin levels will return to normal within seven days postpartum. There is no relation-continued on page 13
ship between the level of engorgement and the amount of circulating prolactin (Riordan and Auerbach 1993). One group of researchers (Gratten, et al. 2001) found increased prolactin receptors in the brain during pregnancy and lactation. Based on the portions of the brain where these increased receptors were found, they felt that increased prolactin levels in the brain could be responsible for changes in the mother such as increased appetite, the suppression of the stress response, and the acquisition of “maternal behavior” (Gratten, et al. 2001).

**POSTPARTUM EMOTIONAL DISORDERS**

Researchers are only beginning to understand the many effects of hormones on the brain, especially during the postpartum period. Estrogen helps to control the production of serotonin in the brain. Serotonin is a neurotransmitter known for its ability to cause elevated mood and an overall sense of well-being. The effect of estrogen on mood has been gaining popularity as a study topic and as a treatment modality.

Estrogen affects serotonin in three ways: First, estrogen removes tryptophan, an amino acid building block used to make serotonin, from its binding sites. Second, estrogen decreases the enzyme monoamine oxidase (MAO), which is known to destroy serotonin. Third, estrogen increases the ability of serotonin to travel throughout the body on the surface of platelets in the bloodstream (Romoff and Yaloff 1999). There is a range of postpartum emotional disorders that could potentially have an endocrine component in addition to, or in lieu of, a psychosocial component. To simplify this discussion, only endocrine changes which may accompany these disorders will be listed.

It is difficult to call the “baby blues” a “disorder” when as many as fifty to seventy percent of all postpartum women report suffering from a mild, transient mood disturbance. This generally peaks at the fourth postpartum day and resolves by the tenth postpartum day, although it could take several weeks. The symptoms include weepiness, sadness, irritability, anxiety, and confusion. These changes coincide with the precipitous postpartum drops of estrogen and progesterone (Wisner, Parry, and Piontek 2002).

Postpartum depression is usually characterized by a depressed mood with or without anxiety, appetite and/ or sleep disturbances, physical agitation or lack of energy, feelings of worthlessness or guilt, and decreased concentration. At its worst it can include thoughts of death or suicide. If a woman has any of the following risk factors, she is considered to be predisposed to postpartum depression: a history of premenstrual syndrome, stressful life events, past episodes of depression, a previous episode of postpartum depression, or a family history of mood disorders (Wisner, et al. 2002). Several very recent studies have examined the effects of a lack of estrogen on mood, as well as treatment effects of estrogen for women with postpartum depression.

In a small pilot study done in the United States (Bloch et al. 2000), sixteen women were separated into two groups: eight had a history of postpartum depression and eight had no history of postpartum depression. Both groups were placed into a hypogonadal state using Lupron. FSH and LH were suppressed, thus suppressing the ovaries from secreting estrogen and progesterone. Next, the women were given supraphysiologic doses of estrogen and progesterone for eight weeks, to simulate pregnancy, and then suddenly were withdrawn from the hormones, to simulate delivery. Five of the eight women with a history of postpartum depression developed mood symptoms. For these women, their mood changes did not occur until they were removed from the hormones abruptly (the simulated delivery). In the group without a history of postpartum depression, none of the women developed mood problems at any time during the study. The researchers were left to conclude that some women, mainly those with a history of postpartum depression, are susceptible to changes in estrogen levels during the postpartum period. This is likely to be genetic, but that has not yet been determined (Bloch et al 2000).

Researchers in Europe looked at estrogen deprivation in postpartum depression patients and used estrogen replacement therapy as a treatment for their depression (Ahokas et al. 2001). This study group consisted of twenty-three women with clinically diagnosed postpartum depression, with no control group. When the study subjects’ physiologic levels of estrogen were measured, they were found to be at or below the levels found in menopausal women. At the beginning of the trial, four women were taking antidepressants without adequate effect. Three had attempted suicide. Based on their physiologic deficits, 1 mg of estradiol was given sublingually three to eight times per day. These women were informed that they could continue to breastfeed but that the estradiol might decrease their milk supply. However, none of the thirteen breastfeeding patients reported any adverse effects. The treatment response to the estradiol was defined as at least a fifty percent reduction in the initial depression score. The researchers decided that there was no change in depression score in two weeks, the patients would also be given an antidepressant. In twenty-one of the twenty-three patients, there was a treatment response reported by the end of the first week. Only two patients needed additional antidepressant therapy at week three (Ahokas et al. 2001).

This same group of researchers in Europe also studied the effects of estrogen on postpartum psychosis (Ahokas, Aito, and Rimon 2000). Postpartum psychosis is the most severe of all of the postpartum mental illnesses. It is often resistant to psychopharmacology. Approximately four percent of women with postpartum psychosis will kill their infants or children. For this study, ten women with clinically diagnosed postpartum psychosis were enrolled. Again, physiologic estrogen levels were continued on page 14
measured. Based on the woman's estrogen deficit, 1 mg of estradiol was given sublingually three to six times per day. Four patients were taking psychiatric medications at the beginning of the study. They were able to stop their medication during the first week of estradiol treatment. Within two weeks, the patients were almost completely symptom free; a group of women, who had previously been hard to treat, was being treated with hormones alone during the study (Ahokas, Aito, and Rimoin 2000). This was a pilot study, and further research with a larger group will continue, to spotlight the effects of estrogen on brain chemistry and mood during the postpartum period.

Within the woman's body is a constant ebb and flow of hormone levels. A woman's body is designed with intricate feedback systems: perhaps when one hormone is high, another hormone is no longer released. It is also designed so that the presence (or absence) of one hormone leads to the release of a second hormone. For example, this is precisely what happens when estrogen and progesterone levels fall after the birth of the baby and prolactin exerts its effects on the breast to increase milk production. This fall in estrogen and progesterone, however, can be troublesome to some women who are particularly sensitive to changes in estrogen levels. Research is leading us to believe that this could precipitate a postpartum depressive event, particularly in women known to be sensitive to hormonal changes, such as women with premenstrual syndrome. It is helpful for the childbirth educator, lactation consultant and any other persons working with postpartum women to be knowledgeable of the many hormonal changes occurring which affect them physically, and could also affect them emotionally. With this knowledge the caregiver can be proactive and anticipate changes women may experience.

References


Additional Web Site Information:

www.clinicaltrials.info.nih.gov — lists current clinical trials, funded through the National Institutes of Health or National Institutes of Mental Health, in the areas of hormones and postpartum depression, perimenopause, and menopause, as well as many others; most of these trials take place around the Washington, DC area.

www.clinicaltrials.gov — clinical trials which receive federal funding are listed at this web site. These trials are throughout the US; information is given on how to contact the investigative agencies

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