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# Introduction to Ellynizer – An Advanced Quantum Biological Device for Eliminating Menstrual Problems

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*Keywords: menstruation, menstrual cycle, ellynizer, quantum entrainment, quantum biology, hypothalamic-pituitary-ovarian axis, menstrual problems, ovulation, spotting, advanced quantum biological device.*

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*Strictly as per the compliance and regulations of:*

# Introduction to Ellynizer – An Advanced Quantum Biological Device for Eliminating Menstrual Problems

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*Keywords: menstruation, menstrual cycle, ellynizer, quantum entrainment, quantum biology, hypothalamic-pituitary-ovarian axis, menstrual problems, ovulation, spotting, advanced quantum biological device.*

## I. Introduction

A healthy menstruation of a healthy woman involves the complex interaction of the hypothalamus, pituitary, ovaries, uterus, prostaglandins, and neuroendocrine factors. The ovarian hormones stimulate the target organs of the reproductive tract and exert feedback effects on the CNS-hypothalamic-pituitary unit to influence its hormone secretion. Disruptions can occur at any step in this multi-faceted process, resulting in hormonal imbalance and menstrual irregularities such as dysmenorrhea (painful periods), Pre-Menstrual Syndrome, and impaired fertility. In order to understand the process of Quantum Entrainment on dysmenorrhea, PMS and dysfunctional uterine bleeding, and their potential etiologic factors, it is important to understand the menstrual cycle.

## II. Menstrual Cycle

The median menstrual cycle length is 28 + 3 days and the average duration of menstrual flow is 5 + 2 days. The cycle, which can be divided into a follicular phase and a luteal phase, results from complex interactions between the hypothalamus, pituitary, and ovary.

This cyclical process, which requires clear communication between the participating glands, is regulated in part by complex changes in the concentrations of five hormones: gonadotropin-releasing hormone (GnRH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), estradiol (E), and progesterone (P) (Figure1). The interplay of these hormones is extremely complicated, with the steroid hormones (E, P) exerting both negative and positive feedback effects on gonadotropin secretion (LH, FSH). The release of LH and FSH from the pituitary is dependent on the secretion of GnRH from the hypothalamus, which is modulated by the feedback effects of E and P. LH and FSH, in turn, are important in

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stimulating secretion of E and P. Virtually all hormones are released in short bursts or pulses at intervals of 1 to 3 hours, so constant levels are not observed in the circulation. The frequency and amplitude of the pulses are modulated by steroid hormones and vary throughout the cycle. But when these are not modulated properly due to various causes like physical/mental stress, contraceptive pills etc. it results in the ultimate irregularities and complications of the menses. In this case of unsynchronized modulation of hormones, Advanced Quantum Biological Device – Ellynizer can artificially entrain healthy normal modulation in the frequency and amplitude of the hormonal releases. There are four distinct phases characterized by histological changes that take place in the uterine endometriumand the hormonal release.

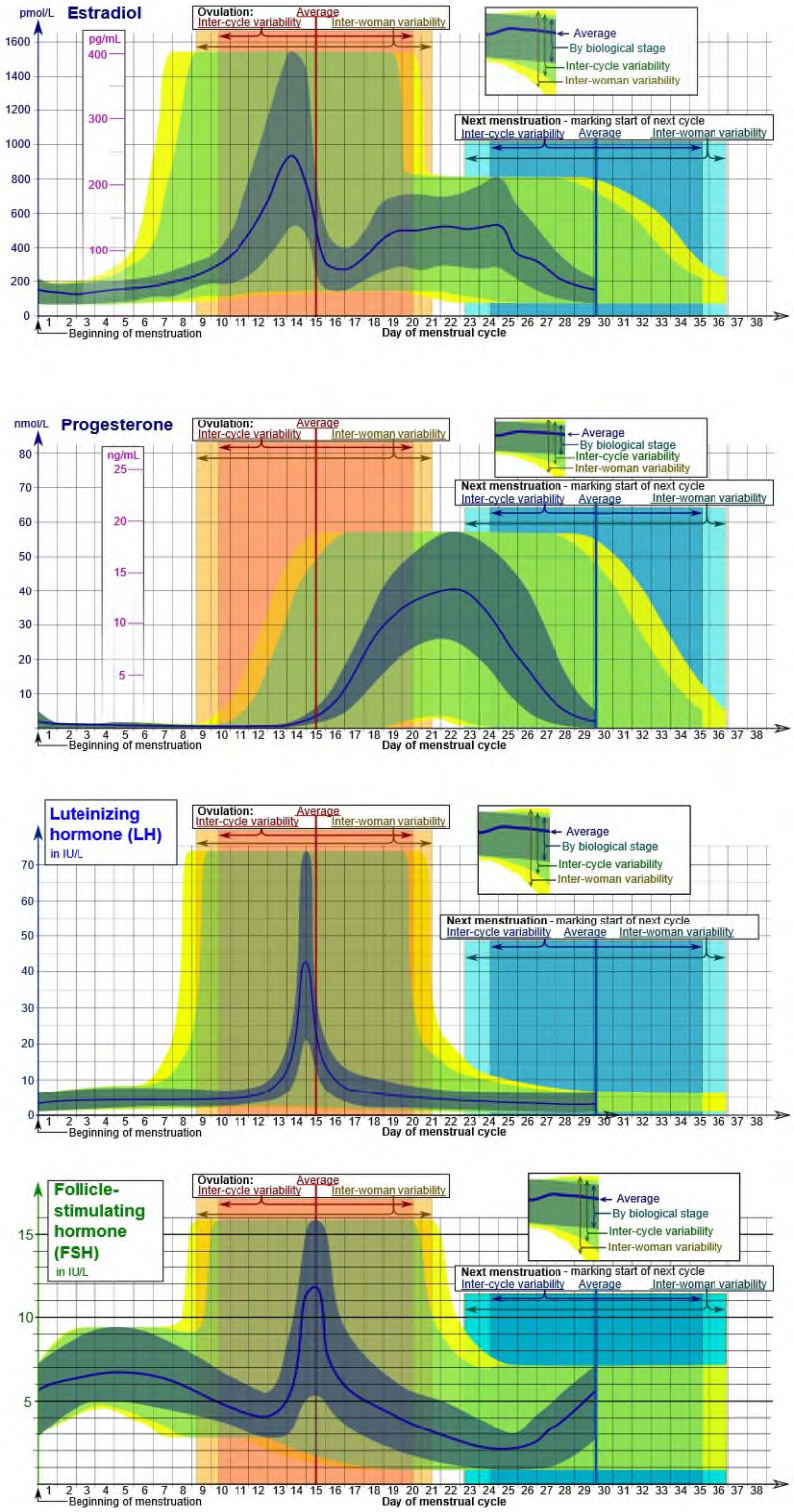


*Figure 1*

The proliferative phase, also referred to as the estrogen phase, begins approximately 5 days after menstruation and lasts for about 11 days. E secreted by the ovary stimulates the growth of the endometrium. The stroma cells and epithelial cells begin to proliferate rapidly, uterine glands begin to grow and elongate, and the spiral arteries begin to grow in order to supply the thickened endometrium. Rising E levels then trigger the midcycle LH surge, which induces ovulation. When ovulation occurs, the endometrium is approximately 3-4 mm thick. At this time the endometrial glands secrete a thin, stringy mucus, which protects and leads the sperm into the uterus.

The luteal or secretory phase, also called the progesterone phase, occurs after ovulation and lasts for about 12 days. The corpus luteum secretes high quantities of P and some E. The E causes slight cellular proliferation in the endometrium. P causes significant swelling of the endometrium and converts it to an actively secreting tissue. P also inhibits myometrial (uterine smooth muscle) contractions, in large part by opposing the stimulatory actions of E and prostaglandins. The endometrium reaches a thickness of 5-6 mm about one week after ovulation. The purpose of this process is to prepare the uterus for implantation of the ovum if fertilization occurs.

In the premenstruation or ischemic phase, if pregnancy has not occurred, the coiled arteries constrict and the endometrium becomes anemic and shrinks a day or two before menstruation. The corpus luteum of the ovary begins involution. This lasts about 2 days and is terminated by the opening up of constricted arteries, the breaking off of small patches of endometrium, and the beginning of menstruation with the flow of menstrual fluid.

The desquamation of the endometrium, or menstruation, is caused by the sudden fall in blood P and E, which results from regression of the corpus luteum. This deprives the highly developed endometrial lining of its hormonal support. The immediate result is profound constriction of the uterine blood vessels, which leads to diminished supply of oxygen and nutrients. After the initial period of vascular constriction, the endometrial arterioles dilate, resulting in hemorrhage through the weakened capillary walls. The menstrual flow consists of this blood mixed with the functional layer of the endometrium. Prostaglandins are thought to mediate both the initial vasoconstriction as well as the uterine contractions accompanying menstrual flow.

## III. Menstruation Problems

The Menstrual Cycle has been the subject of many traditional tales, myths and mysteries. The hormonal changes during the cycle affect women physically, psychologically and behaviorally. The major problems that women suffer are Dysmenorrhea and Dysfunctional Uterine Bleeding.

### *a) Dysmenorrhea*

Dysmenorrhea, or painful menstruation, is one of the most common gynecological complaints. It is estimated to affect almost half of all women at some time during their childbearing years, usually appearing during adolescence and tending to decrease with age and following pregnancy.Lower abdominal cramping and pain that may radiate to the thighs and lower back is the most prevalent symptom. Headache, nausea, constipation or diarrhea, and urinary frequency are often present, and vomiting may also occur. It is characterized by pain occurring on the first day of menses, usually coinciding with the onset of flow, but may not be present until the second day. The symptoms tend to peak after 24 hours and usually subside after 2 days. While many women suffer mild discomfort during menstruation, dysmenorrhea is present if pain prevents normal activity and requires over-the-counter or prescription medication.

There are three types of dysmenorrhea: primary, secondary, and membranous. Primary dysmenorrhea is characterized by the absence of an organic etiology, while secondary dysmenorrhea is associated with specific diseases or disorders, such as endometriosis, ovarian cysts, pelvic inflammatory disease, adenomyosis, cervical stenosis, fibroid polyps, and possibly uterine displacement with fixation. Membranous dysmenorrhea (uterine cast) is rare and causes intense cramping pain as a result of the passage of the intact endometrial cast through an undilated cervix. The most common misdiagnosis of primary dysmenorrhea is secondary dysmenorrhea due to endometriosis. With endometriosis, the pain increases 1 to 2 weeks before the menses, reaches a peak 1 to 2 days before, and is relieved at the onset of flow or shortly thereafter. The use of intrauterine devices (IUDs) may also cause severe cramping.A majority of women suffering from dysmenorrhea are diagnosed with primary dysmenorrhea.

### *b) The Role of Prostaglandins in the Etiology of Primary Dysmenorrhea*

Prostaglandins (PGs) are hormone-like compounds that function as mediators of a variety of physiological responses such as inflammation, muscle contraction, vascular dilation, and platelet aggregation. They are modified forms of unsaturated fatty acids that are synthesized in virtually all cells of the body. Studies have demonstrated that varying PG levels in the female reproductive tract affect the cyclic regression of the corpus luteum and the shedding of the endometrium. PGs may also mediate the effect of LH on ovulation.

The association between the symptoms of dysmenorrhea and intrauterine production of PGs goes back 40 years to the report of Pickles, who first identified a substance in menstrual fluid which stimulated contractions of human uterine smooth- muscle strips. This menstrual stimulant was subsequently found to

contain PGF2α and PGE2, with the PGF/PGE ratio higher in the endometrium and menstrual fluid of women with primary dysmenorrhea. PGF2α and PGE2 have opposing vascular effects causing vasoconstriction and vasodilation, respectively. While PGF2α administration stimulates uterine contractility during all phases of the menstrual cycle, PGE2 may inhibit myometrial contractility during menstruation and stimulate it during the proliferative and luteal phases. Since they are both formed from a common precursor, arachidonic acid, the increase in PGF2α/PGE2 ratio indicates that synthesis can be directed preferentially towards the PGF compounds. Several studies suggest that women with primary dysmenorrhea have elevated concentrations of PGF2α and/or its metabolites in the endometrium, menstrual fluid, and peripheral circulation.

These findings have led to the hypothesis that painful menstruation may be due to hypertonicity of the myometrium with accompanying uterine ischemia caused by the local release of excessive amounts of PGs.Furthermore, escape of PGs from the uterus into the systemic circulation could be responsible for other symptoms of dysmenorrhea such as GI disturbances, faintness, dizziness, and headaches. This theory is supported by several research findings: 1) higher PG levels (especially PGF2α) during the secretory phase than in the proliferative phase of the menstrual cycle; 2) high PG levels and high PGF2α/PGE2 ratio found in the endometrium and menstrual fluid of women with dysmenorrhea; 3) administration of PGs produces symptoms similar to dysmenorrhea; and 4) PG inhibitors successfully relieve symptoms of dysmenorrhea.

PG synthetase inhibitors (non-steroidal anti-inflammatory drugs), such as ibuprofen, mefenamic acid, naproxen, and indomethacin, have been used as analgesic treatment for dysmenorrhea since the early 1970s. Prior to their discovery, women who had dysmenorrhea were dependent largely on narcotics or oral contraceptives for pain relief. PG inhibitors block PG synthesis early in the inflammatory reaction by inhibiting the cyclooxygenase pathway. Once pain has become severe, relief is unlikely. However, these drugs should not be used prior to the onset of menses because of their teratogenic potential.

In a comprehensive review of clinical trials of PG inhibitors in the treatment of primary dysmenorrhea, it was found that significant pain relief was reported for each of the PG inhibitors for the majority of women. However, the authors concluded that 9% to 22% of dysmenorrheic women will not benefit from PG inhibitor treatment, possibly because some of these women may have secondary dysmenorrhea. While PG inhibitors are generally recognized as effective against pain, there are drawbacks. These drugs are not selective in their inhibition of PGs, translating to a reduction of all PGs, good or bad. In addition, possible side effects include dizziness, headache, nausea, vomiting, heartburn, and diarrhea, as well as GI damage with protracted use.

Cyclic administration of oral contraceptives, usually in the lowest dosage but occasionally with increased estrogen, is also used to alleviate pain. The mechanism of pain relief may be related to absence of ovulation or to altered endometrium resulting in decreased prostaglandin production during the luteal phase. Surgery is a rare form of intervention used in women who do not respond to medication.

*c) Dysfunctional Uterine Bleeding*

Abnormal uterine bleeding includes excessive bleeding, irregular bleeding, and absence of bleeding. In about 25% of patients, these menstrual irregularities are due to organic causes. Possible organic causes of abnormal uterine bleeding include, but are not limited to: endometriosis, polycystic ovary syndrome, blood dyscrasias, thyroid dysfunction, pelvic inflammatory disease, anorexia nervosa, diabetes mellitus, pituitary disorders, uterine fibroids, cervical stenosis, cervicitis, endometrial polyps, gynecologic carcinoma, syphilis, vaginal adenosis, adrenal disorders, and corpus luteum cysts. The use of oral contraceptives (as well as their discontinuance), anticoagulants, corticosteroids, and IUDs can also cause abnormal uterine bleeding. For the remainder of patients, there is absolutely no organic pathologic condition but rather a functional abnormality in the hypothalamic-pituitary-ovarian axis, defined as dysfunctional uterine bleeding (DUB). Before reaching a diagnosis of true DUB, the clinician must rule out any underlying pathologic conditions. Patterns of abnormal uterine bleeding and possible underlying medical causes are described below:

Menorrhagia is heavy or prolonged menstrual bleeding that may occur as a single episode or on a chronic basis. Normal menstrual flow lasts about 5 days and produces a total blood loss of 60 to 250 ml. In menorrhagia, the menstrual period is extended and total blood loss can range from 80 ml to overt hemorrhage.

Hypomenorrhea is unusually light menstrual flow, sometimes only “spotting.”

Metrorrhagia is uterine bleeding that occurs irregularly between menstrual periods. The bleeding is usually light, although it can range from staining to hemorrhage.

Polymenorrhea describes menstruation that occurs too frequently.

Oligomenorrhea is abnormally infrequent menstrual bleeding characterized by 3 to 6 menstrual cycles per year. When menstrual bleeding does occur, it can be profuse and prolonged or decreased in amount.

Amenorrhea (secondary) is the absence of a menstrual period for 3 or more months in women with past menses, precluding normal physiological causes such as pregnancy, lactation, and menopause.

*d) Etiology of Dysfunctional Uterine Bleeding*

Dysfunctional uterine bleeding occurs most commonly at the extremes of reproductive age, with 20% of cases in adolescence and greater than 50% in patients over age 40. Normal endometrial bleeding occurs as a result of stimulation of the endometrium by the physiologic levels and balance of estrogen and progesterone present in the normal ovulatory cycle and by the subsequent rapid withdrawal of these two hormones. This withdrawal results in complete and rapid shedding of the entire functional layer of the endometrium. Various disturbances in this balanced estrogen-progesterone relationship can result in four clinical etiologies of true DUB:

*e) Nonovulatory DUB*

Greater than 70% of DUB cases are associated with anovulation. The bleeding in anovulatory women is generally the result of continued stimulation of the endometrium with unopposed estrogen, which occurs when there is a dysfunction of the hypothalamic-pituitary-ovarian axis. The endometrium, thickened by the estrogen, then sloughs incompletely and irregularly, and bleeding becomes irregular, prolonged, and/or profuse. The absence of progesterone results in deficient endometrial prostaglandins so that appropriate spasm of the coiled arterioles is lacking. This also results in irregular and incomplete shedding of the endometrium.

*f) Irregular Ripening of the Endometrium (Luteal Phase Defect)*

This occurs in ovulatory cycles where the corpus luteum production of progesterone is inadequate to permit development of a receptive endometrium. Any disturbance of follicular growth and development can produce an inadequate follicle and a deficient corpus luteum. Patients with luteal phase defects can present primarily with DUB manifested as premenstrual bleeding, menorrhagia, or polymenorrhea.

Irregular (or Prolonged) Shedding of the Endometrium – Irregular shedding of the endometrium is apparently due to slow degeneration of the corpus luteum with prolonged exposure of the menstruating endometrium to the waning progesterone. Clinically, irregular shedding of the endometrium manifests itself by cyclic prolonged menstruation, which may be profuse.

*g) Endometrial Atrophy (or Threshold Bleeding)*

The normal amount of estrogen secreted during the proliferative phase of the cycle results in a stable endometrium that is intact and does not bleed. In the absence of estrogen, or with the minimal levels present premenarchally or postmenopausally, the endometrium is so unstimulated and atrophic that no bleeding occurs. However, with persistent intermediate levels of estrogen, irregular bleeding occurs. This is because there is enough estrogen to stimulate the endometrium but not enough to stabilize it, keep it intact, and maintain it.

## IV. Anatomy of Menstrual Cycle and the Pathway of Solving the Menstrual Problems

The initial signals for a menstrual cycle are initiated from the very central nervous system (CNS) that also controls all the biological functions of the body. The two major sites of action within the CNS, which are important in the regulation of reproductive function are the Hypothalamus and Pituitary.

The hypothalamus consists of only 0.3 % of the total brain, measures 4 $cm^3$, and weighs approximately 10 g. Despite its small size, it contains many nuclei that are responsible for endocrine regulation, reproduction, metabolism, temperature regulation, emotional responses, and electrolyte balance. The hypothalamus lays beneath the thalamus, hence, the nomenclature. Laterally, it is bordered by the anterior part of the subthalamus, the internal capsule, and the optic tract. The hypothalamus forms the lateral wall and floor of the third ventricle. The median eminence of the hypothalamus extends to the anterior pituitary and contains neurosecretory neurons that affect hormone production from the anterior pituitary. The hypothalamus is comprised of three zones: lateral, medial, and periventricular. Within each zone lie several nuclei, where the arcuate nucleus is pertinent to reproduction. The arcuate nucleus is responsible for the production of GnRH. GnRH is secreted into the portal pituitary circulation, reaching the anterior pituitary to affect FSH and LH release from the anterior pituitary. The hypothalamus also influences thyroid function via TRH (corticotropin -releasing hormone), adrenal function via CRH (coricotropin-releasing hormone), andgrowth and metabolic homeostasis via GHRH (growth hormone-releasing hormone).

The pituitary gland is a pea-sized gland, also known as the master endocrine gland. It measures 12 × 8 mm and weight approximately 500 mg. It is located beneath the third ventricle and above the sphenoidal sinus in a bony cavity called the sellaturcica. The adult pituitary gland contains two major parts: the adenohypophysis and the neurohypophysis. The neurohypophysis is a diencephalic down growth connected with the hypothalamus, while the adenohypophysis is an ectodermal derivative of the stomatodeum. The pituitary gland can also be divided into two major lobes: anterior and posterior. The anterior lobe is equivalent to the adenohypophysis, while the posterior lobe is equivalent to the neurohypophysis. The difference is that the nomenclature of anterior and posterior lobes does not include the infundibulum, which extends from the hypo- thalamus to the pituitary gland, which contains neural hypophysial connections and is

continuous with the median eminence. The anterior pituitary contains several cell types: gonadotropes (responsible for secretion of FSH and LH), thyrotropes (responsible for the secretion of thyroid- stimulating hormone [TSH]), adrenocorticotropes (responsible for the secretion of ACTH), somatomammotropes (responsible for the secretion of GH), and lactotropes (responsible for the secretion of prolactin). In addition to these hormones, the anterior pituitary secretes activin, inhibin, and follistatin, which play a role in menstrual cycle regulation. The posterior pituitary lobe contains two cell types that secrete ADH (antidiuretic hormone) and oxytocin. The communication between the hypothalamus and the anterior pituitary is vascular; however, it is a neuronal connection between the hypothalamus and the posterior pituitary.

The gonads in the female consist of the bilateral ovaries. The ovaries are located in the pelvis along the sides of the uterus. In reproductive-age women, ovaries measure approximately 2.5 × 3 × 1.5 cm in size. Laterally, the ovary is attached to the pelvic sidewall by the infundibulopelvic ligament, which contains the vascular supply to the ovary (ovarianartery and vein). The ovary consists of an outer cortex and an inner medulla. The ovarian follicles are found in the cortex, while the medulla mainly contains fibromuscular tissue and vasculature. Each ovarian follicle consists of an oocyte surrounded by layers of granulose and theca cells. These layers will vary depending on the maturation stage of the oocyte contained within the follicle. Within the ovarian cortex, follicles can be found in different stages of development. Earlier stages of follicular development are independent of central nervous system hormone production, while later stages of follicular development will depend on reproductive hormones produced by the central nervous system. The growing ovarian follicle will produce estradiol from the granulose cells. After ovulation, the remnant cells of the follicle luteinize and start secreting progesterone. The granulosa cells are also responsible for the secretion of inhibin as well as anti-Müllerian hormone (AMH). The uterus is largely a receptive organ to all the steroid hormones that emanate from the endocrine glands. The uterus is a fibromuscular organ that is bordered anteriorly by the urinary bladder and posteriorly by the rectum. The uterus can be divided into two major portions: an upper body (corpus) and a lower cervix. The hollow portion of the uterus contains a mucosal lining called the endometrium. The endometrium contains several layers of cells: the basal layer and the superficial layer. The basal layer is responsible for the regeneration of the endometrial cells. The superficial layers undergo the cyclic changes of the menstrual cycle. The endometrium normally proliferates in response to the rising estradiol levels in the first half of the menstrual cycle and is converted to a secretory layer in response to progesterone produced by the corpus luteum in the second half of the menstrual cycle. If the cycle does not result in a pregnancy, where there is lack of hCG, progesterone production is not maintained by the corpus luteum, and the endometrium becomes unstable and sloughs in preparation for a new cycle and another attempt for pregnancy. Just outside the uterus, above the skin of the pelvis, is the region for Ellynizer to entrain Quantum Entanglement with the Hypothalamic-Pituitary-Ovarian Axis.

The intricate and beautiful interconnection between the hypothalamus, pituitary, uterus and the ovaries gives a fantastic advantage in entraining harmonizing impulses in the Hypothalamic-Pituitary-Ovarian Axis. Quantum Entrainment over the HPO Axis would reinstate harmony in the menstruation by balancing the proper production ofgonadotropin-releasing hormone (GnRH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), estradiol (E), and progesterone (P). Disruption or imbalance in the production of these five hormones leads to most of the unexpected menstrual problems. With this phenomenon the specifically designed harmonizing mild impulses, called the Elly Pulses induces balanced regulation in the menstrual cycle. For the entrainment Elly Pulses utilizes the feedback mechanism of the HPO Axis. The design of Elly Pulses cannot be disclosed due to patent concern. The feedback mechanism of the HPO Axis will entrain the Elly Pulses to the entire system in order to harmonize the menses. Once the Elly Pulses are entrained with the HPO Axis, the specific fusion of resonating wave forms induces the same harmonizing resonance to the entire network of HPO Axis, hence the halted neural pathways and the stressed pathways get relieved of odd overwhelming neural firings along with neural disruptions. Therefore neural signals for the modulated bursts of those five basic and most crucial hormones achieve proper modulation. Elly Pulses are a fusion of different forms of impulses that will go through the pelvis to the Uterine region of the female body, thereafter entrain throughout the entire Hypothalamic-Pituitary-Ovarian Axis. Once entrained, it'll eliminate the hormonal imbalances within the body that cause the most menstrual problems like Dysmenorrhea, DUB etc. Also, the terrible mood swings in Pre-Menstrual Syndrome are the result of complex imbalance in the level of hormones. So, Ellynizer fixes the mood swings as well. Theneural network of the HPO Axis acts as a pathway of Quantum Entrainment for the Ellynizer. Past cases of Ellynizer's predecessor "Miracurall" had proven the promising impact of Quantum Entrainment to resolve and cure diseases and complications like Diabetes Mellitus and Tinnitus. In the line of Advanced Quantum Biological Devices right after Miracurall - Ellynizer and Raksanizer will be the solutions to some of the mankind's most aboriginal and day-to-day complications like Menstrual Problems and Neurological Disorders.

## V. Summary

Advanced Quantum Biological Devices (AQBD) are the future of mankind's health issues. Without health life is colorless, so no comprise can be made with the health of mankind. Quantum Entrainment has opened a gateway to the future where no health issue will remain unresolved. Neural network of the human body is the most intricate web that handles literally all activities of the human biology. But sometimes due to different causes, the recovery system of this network gets halted, hence it gives birth to varieties of health issues. Quantum Entrainment reinstates the recovery mechanism of the neural network responsible for specific biological function. The more entrainment pathways we discover, the more we cure diseases.

## VI. Acknowledgment

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