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Contraception: An Embryo's Point of View

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Introduction

Issues surrounding the creation and care of children touch almost every married couple. The marital one flesh relationship forms the context in which God creates new human beings. These new beings make demands of the parents, challenging and stretching their ability to love, to forgive, to be consistent, and to provide for their children's physical, emotional, and spiritual needs. In this practical, immediate care for their children, parents are given an opportunity for character growth.

In the last century, the ability to separate the act of physical intimacy from the birth of children has brought difficult theological and scientific issues into the heart of every marital relationship. Until the 1930s, couples generally understood and accepted that sexual intercourse could very likely lead to conception and birth of children, and married couples mainly faced the challenges of faithfulness to one's spouse and the willingness to give oneself in sacrificial love to each other and to their children. Today's married couples face even more questions: "Do we want to have children?" "Why should we have children?" "How many?" "When is the right time?" "Is there any God-pleasing way to structure our family size and timing?"

Faithful couples faced with these theological and practical questions turn to pastoral counselors for answers. The pastoral counselor seeking scientific information about contraception will find a lot of information designed to sell a particular device or pill. The marketing discussion centers on how effective the drug or device may be in preventing a recognized pregnancy. Little or no discussion is given to the way in which such pills or devices alter or change the natural physiological processes of a woman's body. Rarely is any information given on the effect of that drug or device on interpersonal relationships, long-term fertility, or the effect on an embryo created when the drug fails. The pastor is then left to counsel couples about decisions that have a profound impact on their interpersonal

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relationships, without the foundational knowledge of normal physiological processes, how those processes are altered by contraceptives currently on the market, and the effects such alterations could have on interpersonal relationships.

The purpose of this essay is to equip the reader to better understand how different methods of contraceptives function in relation to the working of the reproductive system. Although each method affects not only the reproductive system and the woman's long-term health, but also the couple's relationship as a whole, this essay will focus only on what is currently known about the effects of contraceptive methods on embryos. Whether or not an embryonic human being faces harm from the use of a contraceptive method is often the single most important question to faithful couples. When pastors and parishioners understand the biological reality of various contraceptive methods, they can then begin to address the deeper theological issues and provide those answers urgently needed to bridge the gap from "What we know" to "What should we then do?"

Normal Physiology of Reproduction: The Symphony of Procreation

In the symphony of human procreation, God designed the bodies of both the husband and the wife to bring new human life into the world in the most protected and nurturing way possible. The biology of procreation is a marvelous illustration of the reality of the one-flesh relationship: It is the only instance in which cells from one person's body fulfill their physiological function inside another person's body![1] The woman's body provides the safe and nurturing context to receive procreative cells from a man, to stimulate procreation, and for new human life to begin and flourish.

Understanding human procreation starts with understanding a woman's monthly cycles. A woman's monthly cycle is counted from the first day she bleeds (day 1) and ends on the day before she starts to bleed again, approximately 28 days later (range 26–32 days). Between day 1 and day 7 (the first week) of the woman's cycle, her brain[2] releases Follicle Stimulating Hormone (FSH) which causes the egg

[1] Sperm cells cannot fertilize an egg as they are when they leave the man's body. Sperm cells become capable of fertilization as they travel through the woman's body, and only here can these male cells finish their maturation and fulfill their purpose.

[2] A special part of the woman's brain (hypothalamus) conducts this symphony. The hypothalamus drums out a beat [GnRH pulse frequency] which evokes the release of specific hormones from her pituitary gland, which in turn controls the woman's ovary. The rate of this hypothalamic beat in the first two weeks of her cycle causes release of Follicle Stimulating Hormone (FSH). FSH causes eggs to mature and make estradiol, and a different hypothalamic beat frequency in the second half of her cycle causes release of Luteinizing Hormone (LH) from her pituitary that then tells the woman's ovary to release an egg and make progesterone. The precise timing of estradiol and progesterone production by the ovary in the woman's body not only causes the egg to be released at a precise time, but also prepares the lining of her womb to embrace and nurture a

sacs (follicles) in her ovaries to grow and produce the natural hormone "estradiol." Estradiol makes the lining of her womb begin to grow again (after being shed in her monthly period).

In the second week of her cycle, one of these follicles will grow faster, as the egg inside gets ready to be released and fertilized. For this reason, the first two weeks of a woman's cycle spent preparing for egg release is called the "follicular phase" of the cycle.

Near the end of the follicular phase, the woman's brain senses that one (or sometimes two) eggs are nearly ready to be released. Her brain stops releasing so much FSH, and begins a different hormone called Luteinizing Hormone (LH). Then, around day 12, her brain releases a very large amount of LH, called the "LH surge." The "LH surge" is what causes the woman's ovary to release an egg. If there is not enough LH, the egg will not be released from the ovary, but rather will stop growing, and eventually be reabsorbed into the ovary. But, if the LH surge is high enough, the egg will be released and gently swept up into the fallopian tube, ready to meet sperm.

In addition to causing the ovary to release an egg, LH has another very important job in the symphony of procreation. LH reprograms the cells of the ruptured follicle into cells that become yellow (luteus) and begin making a very important natural hormone called "progesterone." In this way, LH transforms the follicle that once held the egg into a "corpus luteum" (yellow body). The amount of LH in the LH surge determines the number of cells in the follicle that will later go on to produce progesterone. So the amount of LH in the LH surge and the amount of progesterone produced later in the luteal phase of the cycle are directly related. Once egg release happens, the second half of a woman's cycle is called the "luteal phase" because all the action depends on the corpus luteum producing the right amount and timing of progesterone.

Just as its name implies ("pro" = for; "gest" = pregnancy; "one" = hormone), progesterone is the "for-pregnancy-hormone" that changes the woman's reproductive tract to be able to care for the embryo. Progesterone causes the woman's fallopian tube to gently sweep the newly formed embryo toward and into the womb. Progesterone also prepares the lining of the woman's womb to embrace and nurture the embryo, allowing the embryo to implant. After the embryo has implanted, progesterone causes the womb's tissues to be able to feed the embryo. So progesterone is absolutely essential to the survival of the early embryo,

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new human being. Knowing the basic details of this symphony reveals the implications of interfering at different points with contraceptive drugs, devices, and surgeries.

and LH is the hormone that enables the ovary to produce progesterone. Both LH and progesterone are the targets for hormonal contraception.

One note about the "LH surge": It is possible to have enough LH to cause the egg to be released, but not enough LH to change a sufficient number of follicular cells to make progesterone, leading to insufficient progesterone to prepare the lining of the uterus to receive and nurture an embryo. This condition is known in infertility research as "Luteal Phase Defect" (LPD). Women who have Luteal Phase Defect have a very difficult time with embryo implantation, and early miscarriage.

Once the egg is released, the egg must be fertilized within twenty-four hours; after this time the egg can no longer be fertilized, and no embryo will be created. Understanding the changes that happen in a woman's body in preparation for egg release forms the basis of Fertility Awareness Based Methods (FABMs) of avoiding fertilization. By identifying the time of egg release, and knowing that sperm can last up to five days in a woman's body, couples can time intercourse to either achieve or avoid pregnancy. Since most women have very predictable physiological changes that are relatively easy to recognize, FABMs can be highly effective in preventing fertilization, but require the cooperation of both husband and wife to time intercourse.

If there are sperm present in the fallopian tube to meet the egg when it is released, then fertilization and the procreation of a human embryo can occur. At the moment of fertilization, the sperm and egg fuse together. At the moment of sperm-egg fusion, which happens in milliseconds, an entirely new human being is formed. This new human being has the special scientific name "zygote," another name for a one-celled embryo. This embryo exhibits all the scientific characteristics of a unique human organism[3] and he or she will continue through all the stages of human existence until death.

In order to avoid the reality that a new human being exists at sperm-egg fusion, and to allow for in vitro fertilization research, some researchers began in the 1960s to refer to the zygote as a "fertilized egg." Scientifically speaking, there is no such thing as a "fertilized egg." The moment that the sperm fuses with the egg, the sperm cease to exist and the egg ceases to exist. Scientifically, what exists is a unique human embryo at the beginning of that human being's life.

After fertilization, the one-celled embryo is brushed gently down the tube toward the womb. On the way through the tube, the embryo grows rapidly[4] and

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[3] Maureen Condic, "When Does Human Life Begin? A Scientific Perspective," *Westchester Institute White Paper* 1 no. 1 (2008): 1. Available at www.bdfund.org/whitepapers. Reprinted in: *National Catholic Bioethics Quarterly* 9 no. 1 (2009): 127–208.

[4] All the stages of embryonic development are available at www.visembryo.com and www.ehd.org/prenatal-images-index.php (accessed July 22, 2016).

prepares to implant in the womb. The trip through the fallopian tubes into the womb takes about five to seven days. The cells of the tube move the embryo response to precise amounts of the hormone progesterone made by the corpus luteum. When the embryo arrives in the womb, he or she arrives at a perfect time, ready to implant. The lining of the womb has also been perfectly prepared to embrace and nurture the embryo as he or she emerges from the covering that protected him or her while travelling in the tube. This precise timing comes from the correct amount and timing of progesterone made by the corpus luteum of the ovary.

Once the embryo has implanted, the embryo herself or himself starts to make a special hormone called Human Chorionic Gonadotropin (hCG). Pregnancy tests available at pharmacies and grocery stores test for the presence of hCG in a mother's urine. When a woman has a "positive pregnancy test" that means that her embryo has produced enough hCG to be detected in the mother's urine. This usually occurs about a week after implantation, when the embryo is around fourteen days old.

By the time hCG is high enough to give a positive pregnancy test, the mother misses her period. A missed period is usually the signal to the mother to consider her embryo might be present. Often at the point of the positive pregnancy test, the mother begins to emotionally bond to her unborn child, weeks after the physical bonding has already occurred.

But hCG does more than trigger the mom's awareness of her embryo. The hCG produced by the embryo stimulates the corpus luteum to continue making progesterone[5] in exactly the same way that LH stimulated the corpus luteum. As the LH signal from the woman's brain slowly disappears, hCG takes its place to ensure continuous production of progesterone so that the embryo can survive and grow.

Progesterone continues to stimulate the lining of the mother's womb to provide nourishment to the implanted embryo. Without progesterone produced by the corpus luteum of the ovary, nourishing and sustaining the embryo for the first ten weeks of his or her life, the embryo would die.[6] If for some reason during that time the corpus luteum were damaged or surgically removed, the fetus would die unless the mother is given supplemental progesterone. After approximately thirteen weeks of life, the placenta itself will produce enough progesterone to sustain the unborn child until birth.

During the nine months of pregnancy, and for several months after birth, if she is exclusively breastfeeding, the mother typically will not release additional eggs. If

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[5] Tulay Ozlu, Aysenur Cakir Gungor, Melahat Donmez, and Bulent Duran, "Use of Progestogens in Pregnant and Infertile Patients," *Archive of Gynecology and Obstetrics* 286 no. 2 (2012): 495–503.

[6] Ozlu, "Use of Progestogens," 495–503.

she exclusively breastfeeds for at least six months, and if she does not resume her monthly cycles during that time of breastfeeding, a woman will naturally tend to bear children on average roughly about once every two years. However, a woman can become pregnant during breastfeeding even if she has not resumed her periods.

In summary, the song of human procreation starts with the intricately timed symphony of pituitary and ovarian hormones, which in turn prepare the woman's body to receive and nurture new human life. The ovary responds to the pituitary by maturing an egg during the follicular phase of the cycle and then releasing that egg in response to LH. After egg release, the ovary produces progesterone. Progesterone then stimulates the tube to move the embryo toward the womb at the right time for implantation, and ensures that the lining of the woman's uterus develops with such precision that, at the point when her embryo enters the endometrial cavity, her uterus is perfectly ready to receive, embrace, and nurture that new life. The mother's ovary responds to the presence of her unborn child (as signaled by hCG) by continuing to produce progesterone, which sustains the maternal-fetal embrace, until such time as the unborn baby's placenta has grown and can produce enough progesterone on its own. The placenta will then support the needs of the baby until birth. If she exclusively breastfeeds, a woman's body will tend not to resume making eggs for several months after birth.

Contraception: An Overview

The scientific and medical concept of "contraception" is a curious one. Usually, drugs, surgeries, and implants are given to cure or prevent a disease. But contraceptives do not prevent or cure any disease. In fact, contraceptives prevent our bodies from doing something our bodies were designed to do, i.e., procreate. Contraceptive drugs, devices, and surgeries induce a state in which the normal function of the body is altered. Why would someone want to alter the normal function of his or her body? Perhaps more significant: ought we be inducing an abnormal physical state in our bodies to make our bodies function the way we want as opposed to the way that our bodies were designed to function? Considering some aspects of contraception may give insight into these questions. This chapter will consider three aspects of contraception: the advent of the term *contraception*, the concept of "efficacy," and the reasons for concern about the environment that embryos face when they are created during the use of contraception.

The Advent of the Term Contra-ception

Contra-ception is a term coined in the 1960s for drugs, devices, and surgeries that prevent a sexually active woman from obtaining a positive pregnancy test at the end of her cycle. The term was designed to be the opposite of *conception*.

The term *conception* is understood by most people to be the moment of fertilization—the moment when the sperm penetrates the egg. The term *conception* is used interchangeably with the term *fertilization* in many biology textbooks, as well as current medical dictionaries.

In the 1960s, researchers discovered that giving large amounts of artificial estrogens and progestins could interfere with a woman's reproductive system and keep her from having a positive pregnancy test. But some people were concerned about how these artificial hormones actually worked. Could these hormones harm a human life at the beginning? The general public understood that ending an unborn human life is generally called an "abortion." Many people were worried that these drugs and devices were "abortifacient"—things that end the life of an unborn human being. Manufacturers recognized that this thinking could interfere with the marketability of the drugs or devices and felt a need to reassure the public that contraceptives did not cause abortions.

The solution to the problem of public concern about whether or not contraceptives caused early abortions was developed in 1965 by the American Congress of Obstetricians and Gynecologists (ACOG),[7] which is heavily financed by the contraceptive drug industry. ACOG acknowledged that the term *abortion* is understood to be the ending of a pregnancy. Everyone knows that pregnancy begins at conception. But ACOG recognized that the term *conception* had no scientific definition. (The scientific definition for the beginning of any organism, including human beings, is "fertilization"—the moment of sperm-egg fusion.) So, ACOG decided to legally define the word *conception* as "the completion of implantation."[8] Now, legally speaking, pregnancy begins at conception, but conception is an event that takes place ten days to two weeks *after* fertilization.

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[7] "Terms Used in Reference to the Fetus," American Congress of Obstetricians and Gynecologists *Terminology Bulletin* 1 (1965).

[8] ACOG, "Facts are Important," June 12, 2014. www.acog.org/-/media/Departments/Government-Relations-and-Outreach/FactsAreImportantEC.pdf?dmc=1&ts=20160722T1510227981. Note that ACOG commits many logical fallacies in this document, and also includes some factually inaccurate statements, such as the timing of fertilization which actually takes place in milliseconds, not twenty-four hours. See footnote 3. Rachel Benson Gold, "The Implications of Defining When a Woman Is Pregnant," *Guttmacher Policy Review* 8 (2005), www.guttmacher.org/about/gpr/2005/05/implications-defining-when-woman-pregnant.

Using the ACOG definition of conception as the beginning of pregnancy, the accusation against contraceptives as abortifacient is easily dismissed. Since abortion is defined as the ending of a pregnancy and since pregnancy does not begin until the completion of implantation, then legally, drugs and devices that kill an embryo *before* implantation is complete, cannot be classified as abortifacients.[9] Voila! Problem solved!

Thus the term *abortifacient* is rendered meaningless in discussions about most contraceptive mechanisms of action. But the fact remains that some drugs, devices, and surgeries labeled as "contraceptive" can and do have actions that harm or kill embryos. Thus the difficult moral question for faithful couples is not whether or not a contraceptive is abortifacient but whether or not the contraceptive is *embryocidal*—does it have an action that can kill an embryo if the embryo is formed during the use of that contraceptive drug, device, or surgery?

The Concept of Efficacy of a Contraceptive

In order to determine whether or not some drug or device works as a contraceptive, scientists see if the user has a positive pregnancy test at the end of the cycle in which she used that particular contraceptive drug or device [i.e., day 28 of her cycle]. That positive pregnancy test is the detection of the hormone "beta hCG" made by a living embryo that implanted in her womb a week before. If no beta hCG is found, then the contraceptive worked. But, *how* did it work? This is the core of the huge controversy surrounding contraceptives. Day 28 of a woman's cycle is two weeks after an embryo is created. Did something happen during the first twelve days of an embryo's life to make the embryo not able to survive to produce a positive pregnancy test?

There are three reasons to consider the environment of embryos conceived during the use of a contraceptive:

[9] The ACOG acknowledges the distinction between fertilization and the ACOG definition of conception and the implications on the difference in their recent press release on personhood amendments: "Although the individual wording in these proposed measures varies from state to state, they all attempt to give full legal rights to a fertilized egg by defining 'personhood' from the moment of fertilization, before conception (i.e., pregnancy/ implantation) has occurred. This would have wide-reaching harmful implications for the practice of medicine and on women's access to contraception, fertility treatments, pregnancy termination, and other essential medical procedures." See www.acog.org/About-ACOG/News-Room/News-Releases/2012/Personhood-Measures.

1. All contraceptive drugs, devices, and surgeries "fail" at a certain rate.[10] As noted in a recent paper: "Unintended pregnancies occur with all contraceptive methods, including IUDs. This provides incontrovertible evidence that fertilization and implantation can occur, albeit rarely, with modern methods of contraception."[11]
2. The fact that pregnancy can happen with all contraceptive drugs and devices proves that embryos can be and in fact are created during the use of all contraceptive drugs, devices, and surgeries, because all pregnancies begin with creation of an embryo.
3. When that embryo is created, the contraceptive drug, device, or surgery will create a certain environment for the embryo that either helps or hinders that embryo's life. Understanding the way contraceptive methods work in the woman's body makes clear the environment that the embryo will face during the beginning of his or her life.

Quantifying the extent of embryo destruction or survival during the use of a particular contraceptive method requires knowing how often eggs are released during the use of a particular contraceptive method, how often those eggs are fertilized, producing embryos, and knowing the environment the embryo will face in the woman's reproductive tract. The question to be answered: Is there likely to be an increase in embryo death for women using a particular method of contraception when compared to embryo death in women not using any method of contraception?

How often are eggs released on a particular contraceptive?

Researchers can easily answer this question, because the place in the ovary where an egg grows—the follicle—is easily seen by ultrasound. The follicle ruptures to release the egg, and this rupture is also easily seen by ultrasound. So it is very easy to determine how often eggs are released during a normal cycle (normal is about 60

[10] "Failure rate" is a particularly negative term. Each of those "failures" is a unique human being, made in the image of God. Each "failure" is an embryo that has survived the conditions inside his or her mother's womb caused by the contraceptive; survived to the point of being able to be recognized as a pregnancy. The remainder of this essay will consider what conditions the embryo faces in his or her mother's womb when a couple uses a particular kind of contraceptive.

[11] Roberto Rivera, Irene Yacobson, and David Grimes, "The Mechanism of Action of Hormonal Contraceptives and Intrauterine Contraceptives Devices," *American Journal of Obstetrics and Gynecology* (1999): 1263–69.

percent ovulation rate[12]) and compare that to how many are released using a particular method of contraception.

How often are those eggs fertilized, producing embryos, and is there an increase in embryo death for women using a particular method of contraception when compared to embryo death in women not using any method of contraception?

This is a more difficult question. Currently it is not known even how often embryos are formed in natural cycles without contraception, because there is no direct scientific test for fertilization and embryo formation. If a reliable test for fertilization inside a woman's body were developed, it would be simple to determine the rate of fertilization to pregnancy in normal women and then compare that normal rate with the rate of women using various methods of contraception. But money for contraceptive research comes from contraceptive manufacturers, and the answer to this very important question would likely not be financially profitable. There is a tremendous need for funding the basic research to find a marker for fertilization[13] in order to directly answer the question of the effect of contraceptive methods on embryos.

Although a direct test for fertilization occurring inside a woman's body is not currently available, there is an indirect way to provide a crude estimate of embryo formation. For an embryo to form, an egg must be released. Researchers can measure egg release. When an embryo survives through implantation, the woman will have a positive pregnancy test. Researchers routinely measure the number of positive pregnancy tests at the end of a cycle. This gives a ratio of egg release (ovulation) to pregnancy ratio.

In one hundred normal women not using any kind of contraception, sixty of those women will release eggs in any given cycle. That gives a normal ovulation rate of 60 percent. In one hundred sexually active couples who do not use any contraception, eighty-five of those couples will be able to achieve a pregnancy within a year. These facts yield a normal monthly ovulation to yearly pregnancy ratio of 60 percent/eighty-five pregnancies in couples using no contraception.

If contraceptives have no effects after an egg is released, then the ovulation to pregnancy ratio should be the same whether or not a woman is using

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[12] Jerilynn Prior, Marit Naess, Arnulf Langhammer, and Siri Forsmo, "Ovulation Prevalence in Women with Spontaneous Normal-Length Menstrual Cycles—A Population-Based Cohort from HUNT3, Norway," *PLoS One* 10, no. 8 (2015), http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0134473.

[13] See www.melisainstitute.org. The MELISA Institute is actively seeking an easily usable test for women to determine when fertilization occurs in order to facilitate the best environment for early embryonic life, and to allow for healthy interventions at the beginning of life. This fertilization marker would also clearly answer the question of whether or not embryos are harmed during the use of contraceptives.

contraception. It would be expected that she would *ovulate* less frequently than women not using contraceptives, all other things being equal, but should ovulation occur, then fertilization of the released egg should occur at the same rate regardless of the use of contraception. The normal ratio of ovulation to pregnancy (60 percent ovulation/eighty-five pregnancies) should be maintained.

If it were true that there were no other effects of a contraceptive drug, device, or surgery other than preventing egg release, then measuring the degree to which that contraceptive method prevented egg release should reliably predict the number of pregnancies (i.e., "failure rate") in a year. For example, if the contraceptive method results in reducing ovulation by half (i.e., 30 percent ovulation rate) and if that contraceptive drug or device only works by preventing ovulation, the result should be forty-three pregnancies at the end of the year. If there are fewer pregnancies, then the drug or device must work in ways other than preventing ovulation to explain the reduction in pregnancies. Many hormone-based contraceptives have large discrepancies between ovulation rate and the rate of expected pregnancies, leading researchers to conclude that other mechanisms of action must be at work.

What are those other ways that a contraceptive might use to prevent a recognized pregnancy test at the end of a woman's cycle? There are five points where a contraceptive drug or device can interfere with the human reproductive system and prevent the woman from having a positive pregnancy test on day 28 of her cycle:

1. Prevent the ovary from making and/or releasing an egg
2. Prevent the sperm from meeting the egg in the tube
3. Prevent the embryo from surviving the passage through the tube to the lining of the uterus
4. Prevent the embryo from implanting in the lining of the uterus
5. Prevent the embryo who has already implanted from surviving to day 28.

If a contraceptive drug, device, or surgery worked only by mechanism number 1 or number 2, then no embryo would be formed. Action at points 3–5 will kill embryos. But many methods work by interfering at several different points in the reproductive process. The more points of interference, the more "effective" the contraceptive.

For example, the drug Ella (ulipristal) was approved by the FDA as an "emergency contraceptive." The manufacturer claims that Ella works as an emergency contraceptive by delaying egg release from the ovary for up to five days.

And Ella does work this way. But Ella works by this mechanism *only if* taken several days *before the woman releases an egg*.

What the manufacturer *does not mention* is that Ella can also prevent a positive pregnancy test if taken *after* egg release. It is obvious that Ella cannot possibly prevent the release of an egg that has already been released. So, what is the mechanism by which Ella "works" after egg release?

Ella (ulipristal) is a second generation of the abortion drug RU-486 (Mifeprex). Both drugs are "hormone-blockers"/"anti-hormones." Both drugs effectively block the actions of natural progesterone. Without sufficient progesterone, an embryo is unable to implant in the womb, and will also die after implantation due to the uterine lining not sufficiently nourishing the embryo. Ella is as effective in blocking progesterone as is the abortion drug RU-486.[14] Yet, Ella is called an emergency contraceptive because that is the indication for which the manufacturer sought approval from the FDA.

For faithful couples to make an informed decision regarding the use of Ella or any other method of contraception requires the couple to have basic information about how those methods are known to work. The documented fact that all contraceptive drugs, devices, and surgeries "fail" to prevent pregnancy at a certain rate proves that embryos can be and in fact are created during the use of all contraceptive drugs, devices, and surgeries, because all pregnancies begin with the creation of an embryo.

When that embryo is created, the contraceptive drug, device, or surgery will create a certain environment for the embryo that either helps or hinders that embryo's life. The important question facing faithful couples is: When embryos are created during the use of a certain method of contraception, is there any reason to suspect that the particular method creates conditions inside the mother's body that will make it more likely that these embryos will die? The unique way each contraceptive method functions determines whether an embryo "unintentionally" created will find his or her mother's body a safe haven or a hostile environment.

Contraceptive Methods: Evaluating the Risk to the Embryo

There are basically five different methods currently used to avoid a positive pregnancy test at the end of a woman's cycle: (1) Fertility Awareness Based Methods [FABMs], (2) Barrier methods, (3) Hormone-based methods, (4) IUDs, and (5)

[14] Jerry Reel, Sheri Hild-Petito, and Richard Blye, "Antiovulatory and Postcoital Antifertility Activity of the Antiprogestin CDB-2914 When Administered as Single, Multiple, or Continuous Doses to Rats," *Contraception* 58, no. 2 (1998): 129–36. (During development of the drug ulipristal, the original experimental names for ulipristal were CBD-2914, VA2914, HRP2000, or RTI 3021-012.)

Emergency Contraception. Some methods work exclusively by preventing fertilization and have no effect after fertilization. Some work by sometimes preventing fertilization and sometimes making it difficult or impossible for the embryo to survive. Some methods will directly kill the embryo. Many methods work by a combination of all three. We will look at these five methods from the least to the most harmful for the embryo.

1. Fertility Awareness Based Methods (FABMs)

Based on the biological fact that the egg can only be fertilized during the twenty-four hours after egg release, FABMs time intercourse so that sperm are not present during the twenty-four hours after the egg has been released. Since sperm can live in a woman's reproductive tract for up to five days, the timing of intercourse takes into account sperm survival times. The details on these methods are easily available online,[15] and space does not allow a full discussion here of details. But a brief review below will help with understanding of their effects on embryos conceived during the use of FABMs.

Most women have many physical signs that the egg is beginning to mature and getting ready to release. One example is a woman's vaginal mucus, which is produced in direct response to the amount of natural estrogen produced by the follicles in the woman's ovary during the first half of the cycle. When the follicles are small, very little estrogen is produced, and the woman has very little mucus. As the follicles mature, more natural estrogen is produced, resulting in more clear mucus. Since sperm live up to five days in the woman's reproductive tract, the beginning of clear mucus formation is the signal to avoid intercourse. When an egg is about to be released, copious amounts of clear vaginal mucus are produced. Within two days after ovulation, the consistency of the mucus changes from clear to white and sticky, due to progesterone now produced by the corpus luteum of the ovary after egg release. This change happens more than twenty-four hours after egg release, when the egg is no longer able to be fertilized. So, intercourse after this time under normal circumstances does not result in embryo formation.

Embryos formed during the use of FABMs do not face any increased risk of death. There is no interference with the woman's reproductive tract. The embryo will not face any interference with transport through her fallopian tube, or interference with implantation, or interference with the ability of the ovary to make progesterone to support the early pregnancy. Fertility Awareness Based Methods pose no risk to embryos.

[15] See www.factsaboutfertility.org.

2. Barrier Methods

Like Fertility Awareness methods, barrier methods work by trying to prevent the sperm from reaching the egg when the egg can be fertilized. However, unlike Fertility Awareness methods, this prevention is accomplished by putting up a barrier to sperm transport. This barrier can be temporary (condom, diaphragm, cervical cap) or permanent (male vasectomy, female tubal ligation, or tubal occlusion). The location of the barrier is key to identifying whether or not there is potential harm to the embryo.

In general, reversible barrier methods and vasectomy in men are fairly safe for the embryo. These methods do not affect the ability of the ovary to release an egg, because there is no known hormonal interference with the processes of reproduction in the woman's body. Egg release is not affected, LH surge is intact, and the ovary can produce normal amounts of progesterone during the luteal phase of the cycle. The lining of the womb matures normally, and if an embryo reaches the womb, these methods are not known to interfere with implantation.

However, permanent female sterilization procedures place barriers inside the woman's fallopian tubes, which result in permanent damage that increases the risk that any embryo conceived will not be able to successfully travel through the tube to the womb. This damage to the tube increases the risk that the embryo will implant in the tube (ectopic pregnancy), further discussed below.

2a. Reversible Barrier Methods

Condoms, diaphragms, and cervical caps provide a temporary physical barrier that seals off sperm from entering the womb, preventing the sperm from being present when the egg is released. However, sperm are mighty swimmers, and can frequently escape from these barriers. So, most of the time these methods are used with a spermicide; a chemical designed to kill sperm (cream, foam, or gel) in order to kill sperm managing to escape the barrier.

There was some concern in the past that these spermicides might cause an increase in birth defects, by damaging sperm instead of killing them. This would theoretically allow a damaged sperm to fertilize the egg. Some small reports indicated a possible increase in various birth defects after the use of spermicides, but large studies did not find an increase in birth defects in children born to parents who used spermicides for contraception.

2b. Permanent Barrier Methods: Vasectomy

A vasectomy is a permanent male sterilization procedure that severs the tube (vas deferens) that connects the place where sperm is made with the place where sperm is stored. Since no sperm can reach the storage place, no sperm can be released. Most of the time, vasectomy completely blocks sperm from being released

in the seminal fluid. However, occasionally the cut ends of the vas can find each other and a connection can be formed that again allows sperm to pass into the seminal fluid. Since vasectomy only affects the supply of sperm, and does not affect anything in the woman's reproductive tract, there is no known harm to embryos conceived when the vasectomy fails.

2c. Permanent Barrier Methods: Tubal Occlusion Procedures

The fallopian tubes in a woman can be closed in a number of different ways, including surgically removing a piece of tube, burning the tubes, applying clips from the outside, or applying occlusive material to the inside of the tube. All of these procedures close off the tube completely (most of the time) and prevent sperm from penetrating past the point of obstruction. However, occasionally sperm do penetrate past the occlusion and fertilization can happen. When an embryo is formed in a woman who has had tubal sterilization procedure, the embryo often has great difficulty travelling through the tube, past the area of occlusion. When the embryo is still in the tube at the time he or she is ready to implant, then implantation happens in the tube instead of in the womb.

Pregnancies diagnosed in women with previous tubal sterilization procedures are rare (5/1000) but when pregnancies do occur, the embryo is frequently implanted in the tubes (ectopic pregnancy). An ectopic pregnancy in the fallopian tube is a life-threatening problem for women because, as the pregnancy grows, the tube cannot stretch enough so, at some point, the tube ruptures and the woman can easily bleed to death. To date, there is no accepted procedure to replace the ectopic pregnancy into the womb.[16] So embryos implanted ectopically will die, and sometimes the mother will also die from internal hemorrhage unless the ectopic pregnancy is surgically removed. When ectopic pregnancies are diagnosed, the treatment is to surgically remove the embryo or part or all of the tube.

Summary

Barrier methods work primarily by preventing sperm transport to the egg. Most barrier methods have not been shown to interfere with embryos after fertilization,

[16] There are three case reports of successful attempts at transfer of an ectopic pregnancy: (1) C. J. Wallace, "Transplantation of Ectopic Pregnancy from Fallopian Tube in Cavity of Uterus," *Surgery, Gynecology & Obstetrics* 24 (1917): 578–9; (2) Landrum B. Shettles, "Tubal Embryo Successfully Transferred in Utero," *American Journal of Obstetrics and Gynecology* 163 (1990): 2026–7; and (3) J. M. Pearce, "Term delivery after intrauterine relocation of an ectopic pregnancy," *British Journal of Obstetrics and Gynaecology* 101 (1994): 717–18. (However, the Pearce article was retracted in 1995.) Two case reports do not in themselves provide enough information about the risks, and attempts at tubal transfer to date must be considered experimental. The Watson Bowes Research Institute (http://watsonbowesresearchinstitute.org) is seeking funding for research in ectopic transplantation.

nor do they prevent implantation in the endometrial lining. The exception is permanent sterilization procedures which involve the woman's fallopian tubes. Fallopian tube sterilizations carry an increased risk that any embryos created will implant in the damaged portion of the tube (ectopic pregnancy), a life-threatening condition requiring surgery.

3. Hormone-Based Contraceptives (Artificial Hormones and Hormone Blockers)

Hormone-based contraceptives include those drugs and devices that interfere with the ability of the woman to release eggs and prepare her body to nurture an embryo. This broad group includes two major categories: artificial hormones and hormone blockers/anti-hormones.

> **Artificial Hormone Contraceptives**: The artificial hormone category includes common birth control pills, mini pills, shots, vaginal rings, patches, and any other way of getting artificial hormones into a woman's circulatory system in sufficient quantities to interfere with the natural cyclic release of her own hormones.
>
> **Anti-hormone/Progesterone Blocker Contraceptives**: The anti-hormone/progesterone-blocker category includes two drugs—the abortion pill mifepristone (RU-486, Mifeprex), used commonly overseas as an emergency contraceptive, and the second generation abortion drug ulipristal (Ella, Ella-One) approved in the US as an emergency contraceptive.

All hormone-based contraceptive drugs and devices, whether they are artificial hormones or progesterone blockers, work by disrupting the symphony of natural hormone release and response that causes egg release and also prepares a woman's body to receive and nurture an embryo. This disruption occurs by targeting the release and function of two natural hormones in a woman's body: the hormone LH and the hormone natural progesterone.

Recall from the earlier section about normal physiological processes of reproduction, that the "LH surge" is the trigger that causes the egg to be released from the ovary. Recall also that the amount of LH in the LH surge determines the number of cells in the follicle that will later go on to produce progesterone. So the amount of LH in the LH surge, and the amount of progesterone produced later in the luteal phase of the cycle are directly related.

The hormone progesterone is responsible for causing the embryo to travel down the tube into the womb, and for preparing the lining of the womb to receive and continue to nourish the embryo through the first part of pregnancy. So, both LH and progesterone in perfectly timed amounts are necessary for normal embryo

survival. Blocking or decreasing either of these hormones has significant ramifications for the embryo.

All hormone-based contraceptives work primarily by interfering with the release of LH from the brain.[17] The various artificial hormone combinations and the progesterone blockers cause one of three things to happen with LH:

1. Hormone-based contraceptives can cause very little LH to be released. If there is very little LH released, then no egg release will happen. If the egg is not released, then it cannot be fertilized, and no embryo would be formed. If hormone-based contraceptives always prevented the release of an egg, then there would be no concern about harm to embryos. But since we know for certain that women can become pregnant while taking hormonal contraception, we know for certain that some eggs are released and we know for certain that some embryos are formed.
2. Hormone-based contraceptives can allow enough LH surge to allow the egg to be released, but not enough LH surge to change very many cells in the corpus luteum to be able to later produce progesterone. (This situation of insufficient LH surge happens naturally in a disease called "luteal phase deficiency," which is a cause of recurrent miscarriage. Treatment of LPD involves giving natural progesterone as a supplement throughout the luteal phase of the woman's cycle, and throughout the first few months of her pregnancy.)

[17] ESHRE Capri Workshop Group, "Ovarian and Endometrial function during hormonal contraception," *Human Reproduction* 16, no. 7 (2001): 1527–35. See also Rachel Steward, Alexander Melamed, Anna Granat, and Daniel Mishell Jr., "Comparison of Cervical Mucus of 24/4 vs. 21/7 Combined Oral Contraceptives," *Contraception* 86 (2012): 710–15 quoted here: *"The main contraceptive effect of combined oral contraceptives (COCs) is inhibition of the midcycle lutenizing hormone (LH) surge to prevent ovulation. However, several studies have shown that the percentage of ovulatory cycles in women using low-dose COCs ranges between 1.5% and 16.8%. With this high rate of ovulatory cycles in women taking COCs, we would expect the pregnancy rate with COC use to be much higher than the perfect use failure rate of 0.3% were there not other effective mechanisms of contraceptive action in addition to ovulation inhibition. Another potential mechanism of contraceptive action is the suppression of follicle-stimulating hormone secretion during the follicular phase of the cycle, thereby preventing follicular maturation; however, follicular development has been shown to occur in 23%–90% of cycles in women using COCs. There are also many progestin-related mechanisms that likely contribute to the overall efficacy of the combined contraceptives, such as thickening of cervical mucus, impairment of tubal mobility and peristalsis, and effects on the endometrial lining, making it less suitable for implantation."*

Hormone-based contraceptives can allow eggs to be released, but in this situation of "break through ovulation," the LH level is most often abnormally low, mimicking LPD.[18] If an embryo is created under these circumstances, the corpus luteum will not make enough progesterone to allow for the correct travel time through the tube, or allow for correct preparation of the mother's womb for implantation, or allow for the lining of the womb to nurture the growing embryo through the first part of his or her life. This action of the hormone-based contraceptive causes embryos to die. An abnormally low LH is a common occurrence for the 3 percent to 40 percent of women who release eggs on the birth control pill, and in women who release eggs after taking progesterone blockers.

3. In some women who take artificial hormone contraceptives, the LH surge may not be affected much at all. In this case, an egg will be released normally, and the corpus luteum of the ovary will make normal amounts of progesterone. If an embryo is formed under these circumstances, the ovary will form a normal corpus luteum and produce a normal amount of progesterone. This is the situation for most women who become recognizably pregnant using artificial hormone contraceptives, and then go on to carry that pregnancy to term.

   However, progesterone-blockers kill embryos after implantation. Both RU-486 and Ella directly destroy both the corpus luteum and the place where the embryo implants. Thus it is very rare for an embryo to survive[19] during use of progesterone blockers such as RU-486 or Ella because of direct actions on the corpus luteum and the lining of the womb.[20] That is why both RU-486 and Ella are so "effective" as contraceptives.

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[18] L. B. Zapata, M. W. Steenland, D. Brahmi, P. A. Marchbanks, and K. M. Curtis, "Effect of missed combined hormonal contraceptives on contraceptive effectiveness: a systematic review," *Contraception* 87, no. 5 (2013): 685-700, www.ncbi.nlm.nih.gov/pmc/articles/PMC4322904/pdf/nihms653707.pdf. Quoted here: ". . . among women who did ovulate, cycles were usually abnormal (i.e., low progesterone levels, small follicles and/or poor cervical mucus)."

[19] Donna Harrison and James Mitroka, "Defining Reality: The Potential Role of Pharmacists in Assessing the Impact of Progesterone Receptor Modulators and Misoprostol in Reproductive Health," *Annals of Pharmacotherapy* 45 no. 1 (2011): 115–9.

[20] Reel, Sheri Hild-Petito, and Richard Blye, "Antiovulatory and Postcoital Antifertility Activity of the Antiprogestin CDB-2914 When Administered as Single, Multiple, or Continuous Doses to Rats," *Contraception* 58 no. 2 (1998): 129–36. (During development of the drug ulipristal, the original experimental names for ulipristal were CBD-2914, VA2914, HRP2000, or RTI 3021-012.)

How often are eggs released during the use of hormone-based contraceptives?

The answer depends on a host of different factors, including when in her cycle the woman takes the drug, how much she weighs, whether or not she has taken any drugs that interfere with the hormone-based contraceptive, exactly what kind of hormone-based contraceptive she is taking, and more. But, in general, when researchers study one hundred women who are using birth control pills as hormonal contraception, somewhere between 2 percent and 40 percent of those women will have follicle rupture (ovulation) each cycle.[21] Compared to the rate of follicle rupture in women not using contraception (60 percent) a 2–40 percent rate of rupture predicts that there should be around three to fifty-seven pregnancies each year

in that one hundred women using hormonal contraception. But in one hundred women who use birth control pills as hormonal contraceptives there are one to four pregnancies each year. That fact has led researchers to conclude that hormonal contraceptives likely work in some ways other than just preventing egg release.

What other ways could hormonal contraception work?[22] In addition to interfering with the LH surge, artificial hormone-based contraceptives can affect the ability of the tube to transport the embryo. Some hormonal contraceptives that use only progestins (the "mini-pill") can slow down the tube, increasing the likelihood that the embryo will implant in the tube instead of in the womb. Anti-hormones such as RU-486 and Ella can speed up the tubal transport, causing embryos to reach the womb before the embryo is capable of implanting.

Hormone-based contraceptives can also directly interfere with the growth and development of the lining of the womb, which can hinder or prevent an embryo from implanting in the lining. Both tubal interference and direct interference with the growth and development of the lining of the womb can cause embryos to die. These mechanisms of action have been called euphemistically "interception," "endometrial contraception," and "contra-gestation."

Once the embryo has implanted, artificial hormone-based contraceptives do not dislodge the embryo or lead directly to embryo death. In contrast, progesterone blockers—RU-486 (mifeprex) and ulipristal (Ella)—can directly destroy the embryo by directly blocking the action of progesterone in the womb. So, both RU-486 and ulipristal can both prevent implantation and can cause abortion after implantation.

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[21] See footnote 18. Reisman, *Sexual Sabotage*, 45.

[22] See footnote 18.

Both RU-486 and Ella can also directly destroy the corpus luteum of the ovary,[23] so that it cannot make progesterone to support the early pregnancy.

In summary, there are significant concerns about how hormone-based contraception works. Although frequently, these drugs can prevent egg release, when egg release does happen and embryos are formed, the additional effects of the hormone-based contraceptives make implantation and embryo survival much less likely. For couples who care about never causing harm or death to their embryonic children, hormone-based methods are not a safe option.

4. IUDs: Intrauterine Devices

It has been known for hundreds of years in the veterinary world that inserting something into the womb of an animal will prevent that animal from being able to carry a pregnancy to term. The IUD is a piece of metal or plastic of various different shapes placed inside a woman's womb that sets up a chronic inflammation inside the womb. The 2008 international working group on IUDs published this remarkably honest comment: *"There is sufficient evidence to suggest that IUDs can prevent and disrupt implantation. The extent to which this interference contributes to its contraceptive action is unknown. The data are scanty and the political consequences of resolving this issue interfere with comprehensive research."*[24] An embryo created during the use of an IUD would have very little chance of implanting in the inflamed endometrial lining,[25] and embryos who do not implant die.

But, inflamed wombs bleed easily. So, to improve the bleeding problem, IUD manufacturers started adding artificial hormones to the IUD, hoping to shrink the lining of the womb so that it would not bleed constantly. These artificial hormones also thicken the mucus of the cervix and slow down the movement of the woman's fallopian tube.

It has been claimed recently by those marketing IUDs that the hormonal IUD (Mirena) prevents the egg from being released from the ovary. While there is some slight decrease in egg release for women in the first year on Mirena, after a year of Mirena use, follicular rupture rates are similar to the follicular rupture rates of women who are not using any contraception, which are similar to the rates

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[23] Yuval Yung, Ettie Maman, Libby Ophir, Nirit Rubinstein, Eran Barzilay, Gil Yerushalmi, and Ariel Hourvitz "Progesterone Antagonist, RU486 Represses LHCGR Expressionand LH/hCG Signaling in Cultured Luteinized Human Mural Granulosa Cells," *Gynecological Endocrinology* 30 no. 1 (2014): 42–7.

[24] ESHRE Capri Workshop Group, "Intrauterine Devices and Intrauterine Systems," *Human Reproduction Update* 14 no. 3 (2008): 197–208.

[25] ESHRE Capri Workshop Group, "Intrauterine Devices and Intrauterine Systems," *Human Reproduction Update* 14 no. 3 (2008): 197–208.

of copper IUD users.[26] So preventing egg release cannot possibly be the major mechanism of preventing a recognizable pregnancy in Mirena IUD users.

Those marketing Mirena IUDs also claim that the progesterone component prevents sperm from coming into the womb by thickening cervical mucus. There may be some cervical mucus thickening with progesterone IUD use, but, after a few years, cervical changes disappear. "The presence of good cervical mucus was observed in 69% of the ovulatory cycles studied in the LNG-IUD users. This indicates that effects on cervical mucus cannot be the main mechanism of action of the LNG-IUDs"[27] In fact, both sperm and embryos have been found in the tubes of Mirena and other IUD users, providing direct evidence that sperm can be and are present when eggs are released and that embryos[28] can be and are formed during the use of IUDs. In addition, about 1 in 125 IUD users become pregnant with the IUD in place. For these women, the pregnancy is most often ectopic (where the embryo implants in the tubes) because embryos who make it to the uterus cannot implant. If the embryo does somehow manage to get through the tube and manages to implant in the womb, the trouble is not over. Intrauterine pregnancies which happen with an IUD in place frequently miscarry, become infected, and die, or deliver very prematurely.[29] The chronic inflammation from the IUD disrupts the entire course of a pregnancy for those embryos who do manage to implant in the womb.

In summary, the IUD is designed to work by killing embryos. Any couple who cares about survival of their embryonic children should avoid the use of an IUD.

5. Emergency Contraception

Emergency contraceptives are all capable of killing embryos; the more effective the emergency contraceptive, the more likely it is to kill embryos. Any emergency

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[26] I. Barbosa, S. E. Olsson, V. Odlind, T. Goncalves, and E. Coutinho, "Ovarian Function after Seven Years' Use of a Levonorgestrel IUD," *Advances in Contraception* 11 no. 2 (1995): 85–95. *"In the regularly menstruating LNg-IUD users, according to progesterone levels, 93% of the cycles were ovulatory but just 58% of these 'ovulatory' cycles showed normal follicular growth and rupture."*

[27] I. Barbosa, et al, "Ovarian Function after Seven Years' Use of a Levonorgestrel IUD," 85–95.

[28] Maria Ortiz and Horacio Croxatto "Copper-T Intrauterine Device and Levonorgestrel Intrauterine System: Biological Bases of Their Mechanism of Action" *Contraception* 75 (2007): S16–S30.

[29] American College of Obstetricians and Gynecologists, *Practice Bulletin* 121 (July 2011), reaffirmed 2015. Available at: www.acog.org/~/media/Practice%20Bulletins/Committee%20on%20Practice%20Bulletins%20--%20Gynecology/Public/pb121.pdf?dmc=1.

contraceptive that works *after* egg release must be capable of killing embryos. There are four methods commonly used for emergency contraception:

A. Single-dose progestins (Plan B and Next Choice)
B. Single-dose progesterone-blockers RU-486 (mifepristone, Mifeprex) and ulipristal (Ella, Ella-One)
C. IUDs placed during the luteal phase
D. "Menstrual regulation/menstrual extraction" (i.e., early abortion)

A. Single-Dose Progestins (Plan B, Next Choice)

Progestins are artificial hormones that resemble natural progesterone, but do not have all the actions of natural progesterone. Plan B works poorly as an emergency contraceptive because it does not have much effect after fertilization. Plan B can delay the release of an egg if taken several days before the LH surge. But, if Plan B is taken immediately before the LH surge, egg release will still happen, but the LH surge will be decreased, causing a luteal phase defect as discussed above.

If a woman has already released an egg and takes Plan B, there seems to be little if any effect on her embryo. Single-dose Plan B after egg release probably does not affect either progesterone production or implantation.

B. Anti-hormones/Progesterone Blockers

As discussed previously, Ella (ulipristal, Ella-One) is a second generation abortion drug identical in action to the abortion drug RU-486. Ella can prevent the egg from being released, if taken several days before the egg is about to be released. However, if taken on the day of egg release or after, Ella directly kills the embryo by blocking the action of progesterone to prepare the lining of the womb for implantation, by directly destroying the tissues in the womb that respond to progesterone and nourish the implanted embryo, and also by destroying the corpus luteum of the ovary that makes progesterone to sustain the early pregnancy.

C. The IUD

The IUD is also very effective as an "emergency contraceptive" because the IUD destroys the ability of the womb to allow for implantation by setting up a chronic inflammation inside the womb. If the embryo has already implanted, the IUD will dislodge the embryo, causing an induced abortion.

D. "Menstrual Extraction"

This term is a euphemism for surgically removing the lining of the womb. If the embryo has not yet implanted, then removing the lining will prevent the embryo from implanting. If the embryo has already implanted, then this procedure is an early elective abortion.

In summary, any emergency contraceptive that is effective after egg release must work by killing embryos. This includes Ella, the IUD, and "menstrual extraction." Plan B is a very ineffective emergency contraceptive that can delay egg release if taken several days before the release of an egg. But, if Plan B is taken immediately before the release of an egg, Plan B can lead to the death of the embryo by decreasing the LH surge and inducing a luteal phase defect.

Conclusion

Each one of the five different contraceptive approaches outlined above results in a particular environment inside the woman's womb. Since embryos can be and are created during the use of each of these methods, it is important for couples to understand the basics of each approach, in order to make informed decisions about the risks that their embryonic children will face when created during contraceptive use. For couples who care about never doing harm to their embryonic children, this article will serve as the beginning of a discussion about available methods of spacing children. However, for Christian couples many questions remain; questions about the integrity of the body, the meaning and purpose of the one-flesh relationship, and radical trust in God's promises of sufficiency and provision. These deeper theological issues will remain at the heart of any discussion about the procreation and care of children in a faithful marriage.