HOW DO THE PILL AND OTHER CONTRACEPTIVES WORK?

Part A

The birth control pill is currently being used by over 10 million women in the US[1]. A number of physicians and researchers have noted that the **birth control pill** (BCP) (also called an *oral contraceptive*) is actually an abortifacient (ie, an agent that causes an early abortion; specifically, any agent that causes death of the zygote, embryo or fetus after conception has occurred). Others have stated that they do not believe the BCP (birth control pill) is an abortifacient as noted in the recent publication (1998), written by several physicians entitled: *Hormonal Contraceptives: Are they Abortifacients?* [26]

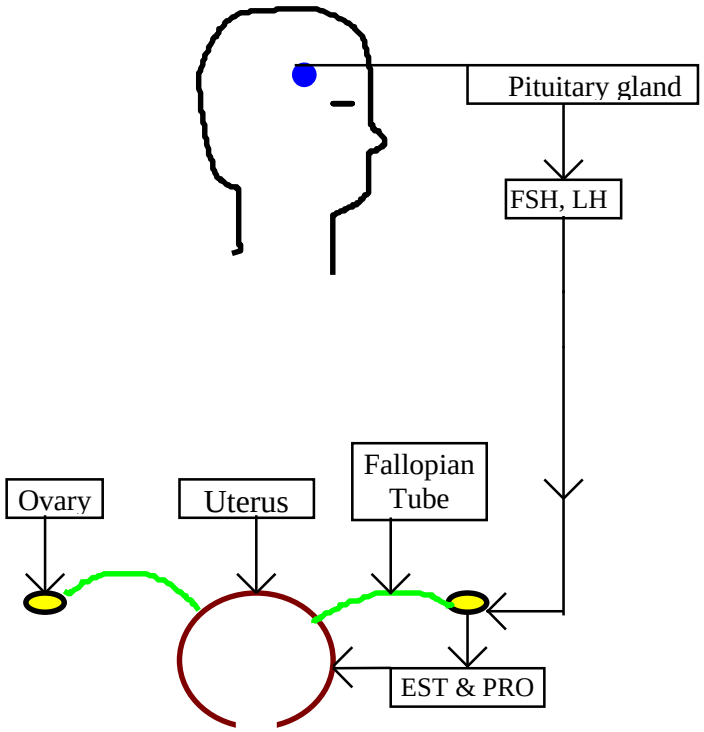
The ethical question of whether contraception is morally permissible has varied among the Catholic Church and Protestant churches. Both agreed on the "sin of contraception" before 1930 [2], while both differ in general on the issue today. This paper will focus on the medical and technical aspects concerning the cited questions regarding the pill's abortifacient qualities.

In order to answer the question of whether the BCP causes early abortions a number of basic questions need to be answered such as:

A) What is an birth control pill (BCP) and how does it work?

Normally, as we can see in diagram A, the pituitary gland produces two hormones called FSH (Follicle Stimulating Hormone) and LH (Luteinizing Hormone). These hormones serve to stimulate the ovary to produce an egg each month (ie, to ovulate). The ovary is also the site of production of the woman's two central female hormones, *estradiol* (EST), a type of estrogen, and *progesterone* (PRO), a type of progestin. Birth control pills (BCPs) are a combination of ***synthetic*** estrogen and progestin. Oral contraceptives "fool" the pituitary gland so that it produces less follicle stimulating hormone and luteinizing hormone. These two hormones are needed for ovulation to occur, therefore, BCPs suppress, **but do not eliminate** ovulation.

Diagram A



Oral contraceptives have two other main effects:

1) they thin the inner lining of the uterus (called the *endometrium)*, depleting it in glycogen (ie, a type of sugar) and decrease its thickness. A thinner endometrium has a decreased blood supply.
2) they may thicken the cervical mucus, making it more difficult for the sperm to travel up through the cervix. The evidence for this is weak [3,4] and not strongly supported by the rabbit model [5].

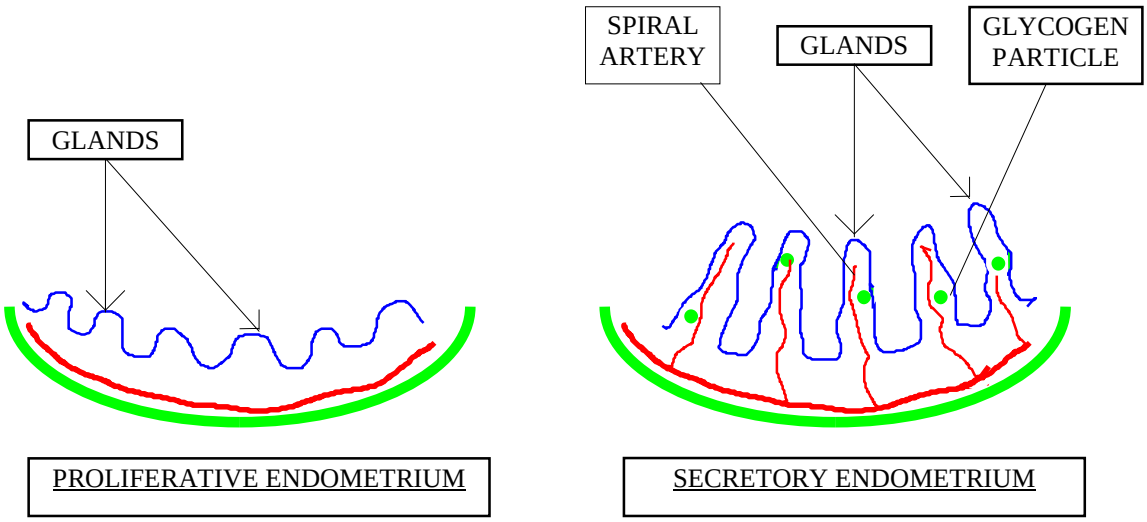
Of course, BCPs could not cause abortions if they always stopped ovulation so this needs to be the first issue that is raised. A clear proof of the occurrence of ovulation is provided by noting what the drug companies which manufacture BCPs state. If one opens up the *PDR* (*Physician's Desk Reference*, ©1998) one will find a table describing the "efficacy rate" of the BCP. In every table listed under each BCP one notes a "typical failure rate" of 3%. The *PDR* defines this as the rate of annual pregnancy occurrence noted in "typical couples who initiate use of a method (not necessarily for the first time) and who use it consistently and correctly during the first year if they do not stop for any

other reason." This means that even couples who used the pill consistently over the course of a year had a pregnancy rate of 3%. A 1996 paper by Potter [6] gives an excellent overview of the matter. She notes that the most recent data point to a rate of pregnancy for "typical use" as being 7%, which is probably the more accurate statistic given the immediacy of her research and the fact that today's BCPs are lower dose, theoretically permitting a higher rate of breakthrough ovulation. From these estimates of BCP failure and the common experience of on-pill pregnancies, it is clear that both ovulation and conception occur in couples who use the BCP.

B) Could you present the evidence that some physicians and researchers give to support their claim that the pill is indeed an abortifacient?

Before presenting the evidence, the normal anatomy and ***histology*** (ie, the study of the body's tissues on a microscopic level) of the inner lining of the uterus, (ie, the *endometrium*) needs to be explained (see diagram B).

Diagram B



The endometrium slowly gets built up *before ovulation* (the ***proliferative phase***) and then reaches its peak in the ***secretory phase*** (shortly after ovulation {and conception if it has occurred}). The endometrium is "ready for the newly conceived child to implant" when it reaches its peak in the secretory phase a few days after ovulation. The blood flow, specifically the oxygen and nutrients to the glandular cells of the endometrium, increases

through the cycle as the ***spiral arteries*** enlarge during the secretory phase. The size of the ***endometrial glands*** also enlarge in the secretory phase. The glands contain important nutritional building blocks for the unborn child who is about to implant, including ***glycogen*** (a type of sugar), mucopolysaccharides (ie, they supply certain building blocks for a cell's growth) and lipids (fats)[7].

C) What does the phrase "ready for implantation" mean?

The author of a histology text designed for medical students notes: "Thus, the various changes that take place in the endometrium during the second half of the menstrual cycle may be regarded as preparing the uterine lining for the nourishment and reception of the fertilized ovum (blastocyst)"[7]. It would appear that God perfectly designed a woman's body and the lining of her uterus to be "optimal for implantation" a few days after ovulation and conception have occurred.

D) Does the BCP cause changes in the lining of the uterus that could be detrimental to the newly conceived child's ability to implant himself or herself?

It would appear so. Since we know that the birth control pill allows ovulation and conception to occur at times, if the pill causes unfavorable changes in the endometrium it would make it difficult for the unborn child to implant, and would support the conclusion that it is an abortifacient.

E) What are some of those changes?

The first change that the BCP makes is to *markedly decrease the thickness of a woman's endometrial lining*. Women who take the pill know this because *they can tell you* that the volume of menstrual contents lost in their monthly cycles significantly decreases once they start taking the pill. *Obviously if a woman is losing less menstrual contents each month, the layer of endometrium that is being shed must be thinner and less well developed*.

F) Is there a technical or quantitative way to measure how much thinner a woman's endometrium becomes when she uses BCPs?

Yes, in 1991 researchers in the US performed MRI scans (Magnetic Resonance Imaging) on the uteri of women, some of whom were taking BCPs and some of whom were not [8]. The BCP users had endometrial linings that were almost two millimeters thinner than that of the nonusers. Although this may sound like a small difference, it represented a 57% reduction in the thickness of the endometrial lining in women who used BCPs in this study.

G) But is there really any evidence that a thinner endometrium makes it more difficult for implantation to occur?

Yes. A number of different research papers have studied this issue and it has been widely described in the medical literature concerning in vitro fertilization where it has been noted that the newly conceived child is much less likely to implant on a thinner uterine lining than a thicker one. Originally an older smaller study (Fleisher et al [9], 1985) did not find that the thickness of the endometrium played an important role in in vitro implantation rates, however, other studies have found a positive trend (Rabinowitz et al [10](1986); Ueno et al [11] {1991}) or a statistically significant effect (Glissant et al [12], 1985) of the decreasing thickness of the endometrium in relationship to a decreased likelihood of implantation. Larger and more recent studies (Abdalla et al [13](1994); Dickey et al [14](1993); Gonen et al [15](1989); Schwartz et al [16] (1997); Shoham et al [17]{1991}) have reaffirmed this important connection. Most studies have found that a decrease of even one millimeter in thickness yields a substantial decrease in the rate of implantation. In two studies, when the endometrial lining became too thin, no implantations occurred (Abdalla [13]; Dickey [14]).

H) What happens to the actual endometrial lining in women who take BCPs when one looks at it under a microscope?

As we saw in diagram B, the uterine lining is at an "optimal state for implantation" when the glands and uterine arteries are at their maximal size. This makes intuitive sense since at this point the blood supply and glycogen and lipid levels that the tiny unborn child needs to survive are at their maximal state. It has already been stated that it becomes significantly thinner but what does it look like on the microscopic level?

Researchers who study the histology of the endometrium find that the BCP causes a number of effects. First, ***the spiral arteries regress significantly,*** becoming much smaller and even difficult to find when one looks under a microscope [18-21]. This of course is important, since an adequate blood supply is critical to the existence of the implanting unborn child. A loss of blood flow means a drastic curtailment in the food and oxygen supply that the child needs to survive. The blood flow to the endometrium is so important that in 1996 one researcher wrote directly about it as concerns its relationship to an unborn child's likelihood of implantation [22]. She first discovered that the blood flow through the spiral arteries peaks at day 16 to 18 of the menstrual cycle and then noted that: "It seems that endometrial perfusion presents more accurate noninvasive assay of uterine receptivity than uterine artery perfusion alone. Therefore, ***blood flow velocity waveform changes of spiral arteries may be used to predict implantation success rate*** to reveal unexplained infertility problems and to select patients for correction of endometrial perfusion abnormalities..." [22] (emphasis added). In layman's language, Kupesic is stating that the efficacy of implantation correlates with the blood flow through the spiral arteries.

I) Are there any other changes on the microscopic level in addition to the reduced blood supply from the spiral arteries?

Yes, the second prominent effect is that ***the endometrial glands become much smaller*** and the "mitotic rate" (rate of cell division) of the cells of the glands decreases [18-21]. Obviously if the glands which supply the glycogen (sugar), mucopolysaccharides or lipids (fats) are compromised, the preborn child who needs those nutrients will have a more difficult time implanting and/or surviving.

J) Many of the studies that examined the endometrial lining are older and were performed when BCPs contained a much higher level of estrogen content (100 micrograms or more). Would the same effect be occurring with more recent BCPs?

Yes. First it should be mentioned that if you ask a woman who is taking lower dose BCPs about the amount of monthly menstrual contents that she loses, she will note that she loses significantly less after she starts taking the BCP. Obviously if she is losing less menstrual contents then she is shedding less each month because the lining of the uterus has become thinner. But what about at the histologic level? Even studies which looked at BCPs which contain 50 micrograms of estrogen (a medium dose) and 0.5 mg of a progestin (eg, norgestrel) found that the spiral arteries and the endometrial glands "shrivel up." [19, 20]

K) Some researchers [50] have argued that if a breakthrough cycle does occur while a woman is taking the pill, her endometrial lining would become similar to that of the non-OCP user for that cycle. Is this an accurate statement?

To the best of this author's knowledge, that statement has no support in the literature. If the above statement were true, it would mean that each time a woman had a breakthrough cycle while taking the OCP (if she does not become pregnant), she should experience as heavy a cycle as if she were not taking the pill. This phenomenon has not been described in the medical literature either.

L) Is there any other <u>new evidence</u> that support the argument that BCPs act by causing an early abortion?

Yes. In 1996 a researcher names Stephen Somkuti published an article concerning the endometrium and a group of molecules called "integrins." [23] ***Integrins*** are a group of adhesion molecules that have been implicated as playing an important role in the area of fertilization and implantation. There are different types of integrins and it is believed

that the endometrium is most receptive to implantation when it expresses certain types of integrins. Birth control pills change the type of integrins that the endometrial lining produces theoretically making it more difficult for the unborn child to implant. In the words of Dr. Somkuti: "These alterations in epithelial and stromal integrin expression suggest that impaired uterine receptivity is one mechanism whereby OCs exert their contraceptive action." [23]

M) Has anyone proven that the BCP causes early abortions?

In order to prove if and how often women are having abortions while taking BCPs one needs to be able to measure how often women become pregnant while taking them. But early pregnancy tests are currently not accurate enough to confirm pregnancy within the first week (although some researchers have been able to detect the hormonal changes in pregnancy as early as four days after conception [24,25]). Until a very early test is developed that can detect pregnancy in women in spite of being on the pill, or until researchers physically measure how many abortions are occurring in women who take BCPs, one cannot state with absolute certainty how often BCPs cause early abortions. New ultrasound technology, might which is capable of detecting ovulation, may give new insights in the future (see answer to question O). As of today, the most accurate description of the current evidence is as follows:

All of the evidence on a microscopic, a macroscopic and an immunological level strongly support the argument that the BCP causes an early abortion at times. Until further studies are done, we should take heed and act upon the current data.

N) Recently a group of physicians, many of whom are experienced Ob/Gyns, wrote a booklet entitled: Hormonal Contraceptives: Are they Abortifacients? [26] In it they write: "The 'hormonal contraception is abortifacient theory is not established scientific fact. It is speculation…" Could you comment on why a group of physicians would hold this view and on the nature of their arguments?

An overview and rebuttal to the arguments cited in the booklet entitled "*Hormonal Contraceptives: Are they Abortifacients?"* is found in the addendum. This author

believes that some of their own arguments can be shown to actually support the argument that the pill is an abortifacient.

Part B: Questions Regarding other Contraceptives

O) How frequently do OCPs cause an early abortion?

At this point, no one knows. There are many factors which influence the answer to this question and it is possible that as technology improves, an accurate estimate will be made. One of the determining factors is how often OCPs allow ovulation to occur. If the rate of ovulation is documented to be substantially higher than the pregnancy rate, then one could start to make an estimate of the frequency of abortion in women who take the OCP.

But measuring a woman's ability to ovulate is difficult. Researchers measure ovulation rates in women who are taking the pill by using several parameters including: 1) ultrasound measurements of the ovary, specifically the size of the largest (dominant) follicle (which contains the egg or oocyte), and 2) hormonal assays of progesterone and estradiol levels. Until now, many researchers have arbitrarily accepted that a pregnancy has occurred when the progesterone levels reaches a certain level. But it is possible that OCPs depress the ovary's ability to produce progesterone despite pregnancy as noted as early as 1962 by Holmes et al [27]. It would seem more accurate to measure ovulation rates based on daily pelvic or vaginal ultrasound exams. In 1985, Ritchie [28] wrote in his review of the role of ultrasound in the evaluation of normal and induced ovulation that: "With daily scanning, ovulation can be demonstrated in >80% of cases." This statistic can only improve as technology moves forward.

There are a number of other reasons why determining the frequency of ovulation by such a method is important. First, studies of women who take the pill often show a high rate of "ovarian activity" in their dominant follicles which may reach a size that is consistent with those seen in non-OCP users who ovulate. In other words the ultrasound measurements indicate that these women (ie, the OCP users) are about to ovulate. But these same studies often conclude that ovulation has not occurred because the progesterone level has not reached a critical level [eg, 29, 30]. This is somewhat counter intuitive in light of a recent study [30] that found: "Patients using the lower-dose monophasic and multiphasic pills had follicular activity similar to that of those using nonsteroidal contraception, with the important exception that ovulation rarely occurred." This study, as almost all others, used the criteria that ovulation is confirmed when a progesterone levels reaches a certain level. This may not be accurate.

High-tech ultrasound may reveal that ovulation rates are higher than today's commonly quoted rates of 3-5% [26]. The two reasons for this are that today's OCPs contain far less estrogen and progestin than the early OCPs did and therefore suppress ovarian activity less often. Second, many studies have examined the rate of breakthrough ovulation *in women who have recently started taking the pill* but the question that must be asked is: "Does the rate of ovulation go up in women who have taken OCPs for more

than a year?" This phenomenon occurs with Norplant, where it was noted that the breakthrough ovulation rate in the first year was only 11%, but increased dramatically after that year, so that a 7-year average yielded an annual breakthrough ovulation rate of 44% [31] (although part of the reason for this increase may have been declining Norplant hormone levels with time). But could a woman's pituitary gland "compensate" or "reset itself" to adjust for the presence of the hormones in the BCP so that ovulation occurs more frequently with time? If so, future trials may show that the rate of breakthrough ovulation increases in women who take the low dose BCP for longer periods of time.

It seems likely that a study will be done in the future that measures the rate of ovulation based on serial ultrasounds (although some may claim that such a study might be unethical). If such a study is performed in women who have been taking *low dose* BCPs *for longer than a year*, it could yield information that leads to a more credible estimate of the abortion rate for women taking BCPs.

P) Does the intrauterine device (IUD) cause abortion?

Yes, the IUD does not prevent ovulation[32] and works by changing the inner lining of a woman's uterus so that the newly conceived child cannot implant in the womb.

Q) Do groups who favor abortion admit that OCPs and the IUD work by causing an early abortion?

The abortifacient nature of the BCP and the IUD is openly admitted by the most ardent pro-abortion supporters. In his arguments before the Supreme Court in 1989, in a case that received world-wide publicity—the case of Webster versus Reproductive Health Services—Mr. Frank Susman, arguing for the pro-abortion side spoke to Justice Anthony Scalia stating: "If I may suggest the reasons in response to your question, Justice Scalia. The most common forms of what we generally in common parlance call contraception today, IUD's, and low-dose birth control pills, which are the safest type of birth control pills available, act as abortifacients. They are correctly labeled as both." [*The New York Times, 1989*: [35]]

R) Do other hormonal contraceptives such as the long acting progestins cause early abortions?

Norplant, manufactured by Wyeth-Ayerst, and Depo-Provera made by Pharmacia-Upjohn are made of artificial progestins. Norplant is composed of levonorgestrel and Depo-Provera of medroxyprogesterone. Depo-Provera is a long-acting progestin that is injected every three months intramuscularly—it is used worldwide despite the fact that studies have shown that it increases the risk of breast cancer by at least 190% in women who take it for more than two years before the age of 25[36]! Norplant is an artificial progestin that consists of a series of Silastic (ie, rubber-like) strips which are filled with levonorgestrel and are implanted under the skin of a woman's upper arm, slowly releasing the progestin into the woman's body over a five year time period.

Norplant has been noted to allow breakthrough ovulation in over 44% of a woman's monthly cycles[31]. In addition, a study in rabbits conducted by a researcher

named Chang[37] has shown that sperm freely reached the rabbits fallopian tubes—even when the rabbits were given high doses of synthetic progestin. The combination of a high rate of breakthrough ovulation and documented sperm migration to the fallopian tubes (in animals) implies that progestins such as Norplant and Depo-Provera allow a high rate of abortion—most likely, higher than OCPs.

S) Does the "<u>the morning after pill</u>" cause an early abortion?

The "morning after pill" consists of a series of high dose OCPs which some women have taken one or two days after thinking that they have conceived. These high dose hormones act as an abortifacient by unfavorably altering the lining of the uterus, thus preventing the newly conceived child from implanting. The animal model described by Castro-Vazquez in 1971 demonstrated this effect in rats[38]. In addition, the *Medical Letter* states that some studies suggest—and some do not—that Preven (the emergency contraceptive hormone kit) may work at times by interfering with the implantation [39].

T) Some emergency rooms give "hormones" to women who have recently been raped. Can this cause an early abortion?

The woman who has been raped within a few hours of coming to the emergency room, may or may not have already conceived. Some emergency rooms will give such a woman high dose estrogen and progestin hormones very similar to the "morning after pill." [the exception is often found in Catholic hospitals whose physicians are not supposed to give the "post-rape pill"]. In the woman who is near the time of ovulation, the hormones may indeed stop ovulation and prevent conception. <u>But if ovulation and conception has occurred, the hormones may work by causing an early abortion in the same way as has been described for "the morning after pill</u>." Since there is no way to know whether conception has occurred, practicing Christian physicians often refrain from giving the "post-rape pill" .

U) Does artificial fertilization cause early abortion(s).

Every method of artificial fertilization that this author is aware of, whether it be in vitro fertilization, or ZIFT (zygote intrafallopian transfer) or GIFT (Gamete intrafallopian transfer) involve the death of many unborn children during the process. Fewer than one out of 20 conceived children "survive" the process of in vitro fertilization. Even GIFT involves the exposure of more than one egg to multiple sperm—a situation in which multiple early abortions are extremely likely to occur. In addition to these methods, it is possible that women who take fertility pills such as Clomid ® (which work by causing the ovaries to "super-ovulate") may be experiencing early abortion(s) since some studies [40, 41, 42, 43], but not all [44], indicate that this drug thins the lining of the uterus, theoretically making it more difficult for the conceived child(ren) to implant.

V) Can the estrogens that women take "after menopause" cause an early abortion?

Often women are started on estrogen replacement near the time of menopause. This usually has a beneficial effect of reducing the risk of osteoporosis while increasing the risk of uterine and breast cancer. Unfortunately, many women are now starting estrogen replacement before they have completely stopped their cycles—that is, they are not always in true menopause, but are still having occasional cycles. If a woman were to start estrogen at a time in which she were still having an occasional cycle, she could still conceive and have an early abortion. This is something to be aware of and women who wish to avoid this effect should not start hormonal replacement therapy until they have not a cycle for a one-year period.

W) Why was the term "contraceptive" placed in quotations when referring to the various artificial hormones?

Oral contraceptives, Norplant, Depo-Provera, the IUD, the "morning after pill," the "post-rape pill," all work by causing an early abortion at least part of the time. The word "contraceptive" was consistently placed in quotations because all of the evidence points to these hormones or procedures as being abortifacients—that is, they cause an early abortion either some or part of the time. Contraception technically means "to prevent conception"—clearly the hormones which were alluded to cause the death of the unborn child after conception and cannot accurately be solely called "a contraceptive."

Addendum

Response to the arguments put forth in the brochure entitled: *Hormonal Contraceptives: Are they Abortifacients?*

Introduction: In January, 1998, a group of twenty-two physicians (almost all are Ob/Gyns) wrote a collaborative report addressing the question of the abortifacient nature of the pill [26]. Their four main arguments (found on page 7 in their booklet) and a corresponding rebuttal to each are presented:

1: They write: "We know of no existing scientific studies that validate the 'hormonal contraception is partly abortifacient' theory. 'On-pill' pregnancy rates roughly parallel

'on-pill' ovulation rates (about 3-5 percent on 35 mcg pill). Increased spontaneous abortion of on-pill pregnancies is not noted."

Response: [Here, the term "pregnancy rate" refers to the rate of pregnancy as confirmed by a positive pregnancy test, while acknowledging that a woman is actually pregnant before one can measure it {ie, directly after conception}].

The claim that "on-pill" pregnancy rates roughly parallel "on-pill" ovulation rates may appear to be a satisfying argument, but on closer examination this contention actually bolsters the argument in favor of the pill acting as an abortifacient. Why?

If a woman is taking the pill she will experience an artificially regulated cycle that lasts 28 days so she will have about 13 cycles per year (365 days divided by 28). Thus a group of 100 women would be expected to have a total of 1300 cycles per year. If women taking the pill experience a breakthrough ovulation rate (ie, on-pill ovulation rate) of between 3% to 5%, a group of 100 women would be expected to have between 39 to 65 breakthrough cycles in one year (1300 x 3% - 5%). *William's Obstetrics* notes that the average woman has a "natural fecundibility rate" of 28 percent.[32] ["Natural Fecundiblity rate," perhaps more accurately called the *fertility rate*, is defined in this section of *William's Obstetrics* as *liveborn infants per ovarian cycle*]. But *William's Obstetrics* also notes that for every 600 liveborn children, 279 embryos or fetuses are miscarried, 176 of them after a positive pregnancy test and 103 of them prior to being able to detect that a woman is pregnant. This means that the average couple will actually have a detectable pregnancy rate of: 28% + (176/600 x 28%) = 36.2%.* So a group of 100 woman who are sexually active and using the birth control pill, might expect between 14 and 24 detectable pregnancies per year: [{39 - 65} x 36.2%]. But the *PDR* (*Physician's Desk Reference*) notes that a group of 100 women who are using the pill in a consistent manner will have about 3 pregnancies per year [33] and a 1996 study by Potter[6] yields an updated statistic of 7 pregnancies per year (see source 366 above). In other words, if the ***condition*** that "on-pill pregnancy rates roughly parallel on-pill ovulation rates" ***is true***, then ***the conclusion*** that the *pill is not an abortifacient* ***highly suspect***. This is because if the ovulation rate is 3% to 5%, we might expect the pregnancy rate to be 14% to 24%—that is, far higher than the ovulation rate. Since we do not see this

clinically, we must ask*: why is the clinically measurable pregnancy rate far lower than the theoretical rate based on the rate of breakthrough ovulation*? A number of explanations exist including the failure of sperm to reach the egg due to thicker cervical mucus or a change in motility within the fallopian tubes which the pill may cause. But one must also recognize that the difference in rates may be due to a failure of the zygote/embryo to implant due to the pill's effects on the endometrial lining. In short, the observation that "on-pill pregnancy rates roughly parallel on-pill ovulation rates", serves, if anything, to give evidence in favor of the argument that the pill is an abortifacient.

***The *total pregnancy rate* (detectable and non-detectable pregnancies) would be the total number of pregnancies per cycle in the average woman: 28% + (279/600 x 28%) = 41.0%.**

2: They write: "There is regular successful implantation of the invasive blastocyst on surfaces a great deal more 'hostile' than 'hostile endometrium' (eg, fallopian tube lining). 'Hostile endometrium' is not a demonstrated clinical reality."

Response: This argument is specious. It has already been stated in the answers to questions B-K that the sum of the evidence—both recent and old—supports the argument that the pill changes the lining of the endometrium in a fashion unfavorable for implantation. The fact that the unborn child may attach him or herself to a structure such as the fallopian tube lining has little to do with the previous arguments. Although one can make the argument that a rare occurrence or an exception disproves a theory, one cannot deduce the converse, namely, that the exception proves the theory. That is, noting that some unborn children do implant in the fallopian tube, or for that matter in the peritoneal cavity, merely *proves that it is possible for this event to occur*. But <u>it offers no evidence</u> that justifies the claim that a favorable implantation site <u>is just as good as</u> an unfavorable one.

3: They write: "The extremely rare reporting of ectopic pregnancies associated with hormonal contraception would indicate the rarity of actual conception by patients using these modalities."

Response: Once again the noted physicians apparently were unaware that their statement serves the purpose of supporting the pill's action as an abortifacient. Women who take the pill and those who do not, can and do become pregnant. The pregnancy can be an extrauterine pregnancy (EUP) {ie, usually a tubal pregnancy} or an intrauterine pregnancy (IUP) {ie, the normal type of pregnancy}. One can measure the ratio of EUP to IUP in either group. What should happen to this ratio {ie, (EUP)/(IUP)} if one compares women who are not taking the pill to those who are?

The Ob/Gyns would argue that this *ratio should remain* constant and if the reporting of ectopic pregnancy were *"practically unreported,"* as the Ob/Gyns write, one might even expect *the ratio to go decrease,* since the numerator would become smaller. On the contrary, if the pill caused more early abortions (ie, less intrauterine pregnancies), one would expect the number of intrauterine pregnancies (IUPs) to decrease in comparison to the number of extrauterine pregnancies (EUPs) and thus the ***ratio should increase***. What does the literature say?

The studies to date note that women who take the pill have ***an increased ratio*** of EUP to IUP. They note that women who take the pill are far more likely to experience more EUP's per IUP than women who do not take the pill, which supports the argument that the pill is an abortifacient. The odds ratio (eg, an odds ratio of 2.0 is the same as saying a two-fold risk) of the increased risk of EUP/IUP in women taking the pill compared to women who were not taking the pill were as follows: 1) WHO [45] found an odds ratio of 1.7 (1.1-2.5); 2) Mol et al [46] found an odds ratio of 1.8 (0.9-3.4); 3) Job-Spira et al [47] found an odds ratio of 4.3 (1.5-12.6); 4) Thorburn et al [48] found an odds ratio of 4.5 (2.1-9.6); and 5) Coste et al [49] found an odds ratio of 13.9 (1.8-108.3). These clinical studies once again contain evidence which suggests that the pill acts as an abortifacient.

4: They write: "Many factors play a part in how a family plans and spaces their children. It is not the purpose of this paper to promote nor to oppose hormonal contraception."

Response: As a physician I know that it is common to use a medicine or a type of procedure because previous physicians have done so. It is simply impossible for each physician to "re-invent the wheel" when trying to decide if a particular drug or procedure is the optimal one. Unfortunately, once one becomes accustomed to particular ways of doing things, one tends to continue to do them in a particular fashion because "they have always been done that way," and "new thoughts" on a "standard procedure" are not always appreciated.

How do these statements pertain to the current argument? It has been stated that almost every physician who signed or helped write the booklet *Hormonal Contraceptives: Are they Abortifacients?* is/was an obstetrician. It is common knowledge that virtually all obstetricians prescribe the pill to their patients for contraception, in addition to other indications. Therefore, ***I assume*** (and would certainly issue a retraction were I proven wrong) that nearly every obstetrician who signed or helped write the paper, prescribes or prescribed birth control pills for contraception.

The problem here is that self-proclaimed pro-life obstetricians would have difficulty being unbiased toward the argument that the pill causes early abortions, since each of these physicians most likely has written thousands of oral contraceptive prescriptions in their careers. The admission that the pill is likely an abortifacient amounts to an admission that hundreds of tiny unborn children have likely been aborted by the physicians who prescribed the pill. *Would it not be difficult to expect a pro-life obstetrician to fairly evaluate the pill as an abortifacient when one considers these circumstances*?

In conclusion, the arguments presented by the twenty-two physicians in the booklet entitled *Hormonal Contraceptives: Are they Abortifacients*? lack substance and actually serve to bolster the evidence that the birth control pill causes early abortions.

Footnotes:

1) Faust JM. Image change for condoms. *ABC News Report*. [Internet E-mail]. 6/8/97.

2) Smith, Janet. Contraception, Why Not? ©One More Soul. Dayton, OH (1-513-279-5433)

3) Elstein M et al. Studies on low dose oral contraceptives: cervical and plasma hormone changes in relation to circulating d-norgestrel and 17alpha-ethyniyl estradiol concentrations. *Fertility and Sterility*. 27; 1976: 892-899.

4) Wolf DP et al. Human cervical mucus v. oral contraceptives and mucus rheologic properties. *Fertility and Sterility*. 32; 1979: 166-169.

5) Chang MC, Hunt DM. Effects of various progestins and estrogen on the gamete transport and fertilization in the rabbit. *Fertility and Sterility*. 1970; 21: 683-686.

6) Potter LA. How effective are contraceptives? The determination and measurement of pregnancy rates. *Obstet Gynecol*. 1996; 88: 13S-23S.

7) Snell, Richard. Clinical and Functional Histology for the Medical Student. Little, Brown & Co. Boston; © 1984, 586-591.

8) Brown HK et al. Uterine Junctional Zone: Correlation between Histologic Findings and MR Imaging. *Radiology*. 1991; 1798: 409-413.

9) Fleischer AC et al, Sonography of the endometrium during conception and nonception cycles of in vitro fertilization and embryo transfer. *Fertility and Sterility*. 1986; 46: 442-447.

10) Rabinowitz R et al. The value of ultrasonographic endometrial measurement in the prediction of pregnancy following in vitro fertilization. *Fertility and Sterility*. 1986; 45: 824-826.

11) Ueno J et al. Ultrasonographic appearance of the endometrium in natural and stimulated in vitro fertilization cycles and its correlation with outcome. *Human Reproduction*. 1991; 6: 901-904.

12) Glissant A et al. Ultrasound study of the endometrium during in vitro fertilization cycles. *Fertility and Sterility*. 1985. 44: 786-789.

13) Abdalla HI et al. Endometrial thickness: a predictor of implantation in ovum recipients? *Human Reproduction*. 1994; 9: 363-365.

14) Dickey RP et al. Relationship of endometrial thickness and pattern to fecundity in ovulation induction cycles: effect of clomiphene citrate along and with human menopausal gonadotropin. *Fertility and Sterility*. 1993. 59: 756-760.

15) Gonen Y et al. Endometrial thickness and growth during ovarian stimulation: a possible predictor of implantation in in vitro fertilization. *Fertility and Sterility*. 1989; 52: 446-450.

16) Schwartz LB et al. The embryo versus endometrium controversy revisited as it relates to predicting pregnancy outcome in in vitro fertilization-embryo transfer cycles. *Human Reproduction*. 1997; 12: 45-50.

17) Shoham Z et al. Is it possible to run a succesful ovulation induction program based solely on ultrasound monitoring: The importance of endometrial measurements. *Fertility and Sterility*. 1991; 56: 836-841.

18) Hilliard George D, Norris HJ, *Pathologic Effects of Oral Contraceptives*, Recent Results in *Cancer Research*. 1979. 66;49-71.

19) Ober WB. The effects of oral and intrauterine administration of contraceptives on the uterus. *Human Pathology*. 1977; 8: 513-527.

20) Ober WB. Synthetic progestagen-oestrogen preparations and endometrial morphology. *J. Clin Path*. 1966; 19: 138.

21) Roland M et al. Sequential endometrial alterations during one cycle of treatment with synthetic progestagen-estrogen compounds. *Fertility and Sterility*. 1966. 17: 339.

22) Kupesic S. The first three weeks assessed by transvaginal color doppler. *J. Perinat. Med*. 1996; 24: 301-317.

23) Somkuti SG et al. The effect of oral contraceptive pills on markers of endometrial receptivity. *Fertility and Sterility*. 1996. 65; 484-488.

24) Witt B, Wolf G, et al. Relaxin, CA-125, progesterone, estradiol, Schwangerschaft protein, and human Chorionic Gonadotropin as predictors of outcome in threatened and nonthreatened pregnancies. *Fertility and Sterility*. 1990; 53: 1029-1036.

25) Norman RJ et al. Inhibin and relaxin concentration in early singleton, multiple, and failing pregnancy: relationship to gonadotropin and steroid profiles. *Fertility and Sterility*. 1993; 59: 130-137.

26) DeCook JL, McIlhaney J et al. *Hormonal Contraceptives: Are they Abortifacients:* 1998; Frontlines publishing. Sparta, MI. For contact information call 1-616-887-6256. Email: order@frontlines.org

27) Holmes et al. Oral contraceptives: An assessment of their mode of action. *The Lancet*. June 2, 1962. 1174-1178.

28) Ritchie WGM. Ultrasound in the evaluation of normal and induced ovulation. *Fertility and Sterility*. 1985; 43: 167-181.

29) Van der Vange N. Ovarian activity during low dose oral contraceptives. Contemporary *Obstetrics and Gynecology*. G. Chamberlain. London, Butterworths, 1988, 315-326.

30) Grimes DA et al. Ovulation and follicular development associated with three low-dose oral contraceptives: A randomized controlled trial. *Obstetrics & Gynecology*. 1994; 83: 29-34.

31) Croxatto HB, Diaz S, et al. Plasma progesterone levels during long-term treatment with levonorgestrel silastic implants. *Acta Endocrinologica.* 1982; 101: 307-311.

32) Cunningham et al. *Williams Obstetrics*, 20th Edition. Appleton and Lange. © 1997. Stanford, CT. p 580-1.

33) *Physicians' Desk Reference* : 1997 {The noted information can be found when looking up any oral contraceptive. Failure rate for "typical use" is noted to be 3 percent.}

34) Van der Vange N. Ovarian activity during low dose oral contraceptives. Contemporary *Obstetrics and Gynecology*. G. Chamberlain. London, Butterworths, 1988, 315-326..

35) Alderson Reporting Company. Transcripts of oral arguments before court on abortion case. *New York Times*. April 27, 1989; B12.

36) Skegg DCG, Noonan EA, et al. Depot medroxyprogesterone acetate and breast cancer [A pooled analysis of the World Health Organization and New Zealand studies]. 1995; *JAMA*: 799-804.

37) Chang MC, Hunt DM. Effects of various progestins and estrogen on the gamete transport and fertilization in the rabbit. *Fertility and Sterility*. 1970; 21: 683-686.

38) Castro-Vazquez. Macome JC, et al. On the mechanism of action of oral contraceptives. Effect of Lynestrenol on ovum implantation and oviductal morphology in the rat. *Fertility and Sterility*. 1971; 22: 741-744.

39) An Emergency contraceptive kit. *The Medical Letter*. October 23, 1998; 40: 102-3.

40) Eden JA et al The effect of Clomiphene citrate on follicular phase increase in endometrial thickness and uterine volume. *Obstet. Gyn*. 1989; 73: 187-190.

41) Yagel S et al. The effect of ethinyl estradiol on endometrial thickness and uterine volume during ovulation induction by clomiphene citrate. *Fertility and Sterility*. 1992. 57: 33-36.

42) Fleischer AC et al. Sonographic depiction of endometrial changes occurring with ovulation induction. *J of Ultrasound Med*. 1984; 3: 341-346.

43) Imoedemhe DA et al. Ultrasound measurement of endometrial thickness on different ovarian simulation regimens during in vitro fertilization. *Hum Reprod*. 1987; 2: 545-547.

44) Dickey RP et al. Relationship of endometrial thickness and pattern to fecundity in ovulation induction cycles: effect of clomiphene citrate alone and with human menopausal gonadotropin. *Fertility and Sterility*. 1993. 59: 756-760.

45) The WHO Task Force on intrauterine devices for fertility regulation. A multinational case-control study of ectopic pregnancy. *Clin Reprod Fertil* 1985;3:131-143.

46) Mol BWJ, Ankum WM, Bossuyt PMM, and Van der Veen F. Contraception and the risk of ectopic pregnancy: a meta analysis. *Contraception* 1995;52:337-341.

47) Job Spira N, Fernandez H, Coste J, Papiernik E, Spira A. Risk of Chlamydia PID and oral contraceptives. *J Am Med Assoc* 1990;264:2072-4.

48) Thorburn J, Berntsson C, Philipson M, Lindbolm B. Background factors of ectopic pregnancy. I. Frequency distribution in a case-control study. *Eur J Obstet Gynecol* Reprod Biol 1986;23:321-331.

49) Coste J, Job-Spira N, Fernandez H, Papiernik E, Spira A. Risk factors for ectopic pregnancy: a case-control study in France, with special focus on infectious factors. *Am J Epidemiol.* 1991;133:839-49.

50) DeCook J et al. *Hormonal Contraceptives, Controversies and Clarification*. February, 1999. Pro-Life Obstetrician. PO Box 81, Fennville, MI 49408