Birth Control Pills: Contraceptive or Abortifacient?

Currently the claim that hormonal contraceptives [birth control pills, implants (norplant), injectables (depoprovera)] include an abortifacient mechanism of action is being widely disseminated in the pro-life community. This theory is emerging with the assumed status of "scientific fact," and is causing significant confusion among both lay and

significant weakening of both our credibility with the general public and our effectiveness against the tide of elective abortion.

This paper is meant to provide some clarifying information on the issue based on current knowledge and experience regarding the mechanism of action of hormonal contraceptives. It has been compiled in consultation with, and by cooperative effort of, several practicing obstetrician-

of human life from conception.

We begin with the recognition that within the Christian community there

We would consider this a personal matter of conscience and belief, and this paper is not intended to argue for or against this issue.

In this discussion we accept the time honored definition that conception occurs when a sperm penetrates an egg. Disruption of the fertilized egg after this point constitutes abortion. We consider fertilization, not implantation, to be the beginning of human life.

Most literature dealing with hormonal contraception ascribes a three-fold action to these agents: 1) inhibition of ovulation, 2) inhibition of transport, and 3) production of a "hostile endometrium," which presumably prevents or disrupts implantation of the developing baby if the first two mechanisms fail. The first two mechanisms are true

be abortifacient. (Note: the developing baby at the time of implantation is called a "blastocyst," and will be referred to as such in this paper. "Endometrium" is the lining of the uterus into which the blastocyst implants.)

The typical clinical picture of spontaneous abortion (heavy bleeding

[redacted]

substantiated in any literature we are acquainted with. The "hostile

[redacted]

pre-implantation, and thus would have no tell-tale clinical or laboratory

[redacted]

women on the pill roughly approximates the pregnancy rate for women on the pill, this type loss would seem extremely unlikely

[redacted]

It is impossible to say. Ovulation suppression rates vary from about 95%

[redacted]

norplant in place 3-4 years. Cervical mucus factors enter in. Most pill literature estimates 3 to 5% pregnancies per year for combined OCs, less for depoprovera, more for norplant, and minipills.

One may get an idea of the frequency of conception on hormonal

[redacted]

[illegible]

[redacted]

same- about 1% (unaffected by whether the endometrium is "hostile" or [illegible]

(except for the minipill) are rarely reported. This would suggest

[redacted]

[illegible]

increase in ectopics in women on hormonal birth control. We don't

[redacted]

loss of blastocysts in some instances? In Medicine, anything is possible.

[illegible]

[redacted]

We believe the answer is "No."

There are 1,200,000 medical and surgical abortions [illegible]

is partly abortifacient" theory is not established scientific fact. It is [illegible] the discussion presented here suggests it is error. How happy the abortionists must be to find us training our guns on a presumption, causing division/confusion among pro-life forces, and taking some of the heat off the abortion industry. Ought we not rather be spending our energies to eliminate the convenience destruction of the innocent unborn?

In Summary:

1. We know of no existing scientific studies that validate the "hormonal contraception is partly abortifacient" theory.

2. There is regular successful implantation of [illegible] surfaces a great deal more "hostile" than "hostile endometrium" (e.g., fallopian tube lining). "Hostile endometrium" is not a demonstrated clinical reality.

[illegible] associated with hormonal contraception would indicate the rarity of actual conception by patients using these modalities. (Mirena and norplant apparently are less effective in preventing pregnancies and ectopics).

[illegible] children. It is not the purpose of this paper to promote nor to oppose [illegible] affecting their own circumstances; decides to use this modality, we are confident that they are not using an abortifacient.

5) This paper is not meant to be the "final word" [illegible] scientific study should validate that a hormonal contraceptive agent is partly abortifacient in its action, we would oppose that agent just as we oppose elective medical and surgical abortions.

We must constantly examine valid data as it becomes available in our [illegible] control to be used or prescribed by those who hold to the sanctity of human life from the time of conception.

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Dear Fellow-Physicians, Colleagues & Other Interested Healthcare Issues:

We have previously sent you our literature research paper entitled "Hormone Contraceptives: [illegible] in about 97, published in 1980 which challenges a point that we made on page [illegible] paragraph [illegible] concerning the return of the endometrium in a birth control pill cycle in which there has been escape ovulation, (and thus a different hormone milieu than exists in a birth control pill cycle with no ovulation). This point is important because it bears on the quality of the endometrial lining that may [illegible] an ovulatory bleed. We have reviewed the original article and commented [illegible] our paper. Appendix 2 is enclosed for your consideration. Once again, this material has reference to the second paragraph on page 3 or our original paper. We would be glad to attempt to answer any questions you may have. If you need another copy of our original paper, please let us know.

Sincerely,

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Appendix 3 (Refers to Page 3, Paragraph 2, of our original paper)

[redacted] some insight into the phenomenon of breakthrough ovulation and the subsequent response of the endometrium. Unless [redacted] frequently to give his testimony.

We have reviewed his 1980 article "Escape ovulation in women due to the missing of [redacted] "low dose combination oral contraceptive pills" by V. Chowdhury et al (2) and have also been in personal correspondance with the authors. We have also reviewed a number of newer research articles on the subject of escape ovulation and ovarian activity on the combined oral contraceptive pills (see list of appendix references.) We would like to [redacted] briefly discuss these below.

In brief, the 1980 Chowdhury article studied "ovulation" in 35 women who were previously sterilized and then asked to take a 30 ug ethinyl estradiol plus norethindrone acetate combination OCP. They were asked to miss a consecutive pills in a cycle and then progesterone levels were measured at day 22 of the cycle, and endometrial biopsies were also obtained. Chowdhury found that 10 out of 35 women had progesterone levels greater than 4 ng/ml. He concluded that these 10 women had ovulated, based solely on this level of progesterone.

But, is a single serum progesterone level of greater than 4 ng/ml sufficient evidence to prove ovulation? Many authors have addressed this question. The answer is: "Clearly, No". Let us look at one of these studies more closely: the 1982 article by Hull et.al. (6): "The value of a single serum progesterone measurement in the midluteal phase as a criterion of a potentially fertile cycle (Ovulation") derived from [redacted] by measuring serum progesterone in the midluteal phase (i.e. day 25+/-2 of cycle) of cycles that concieved. He studied conception cycles because those cycles which conceive [redacted] occurred; they are the only documentably proven ovulatory cycles. Let's look at his findings:

[redacted] observed with a mean progesterone value of 40.7 nmol/ml (12.7ng/ml), 95% confidence [redacted] 80nmol/ml, 6.9 to 25.2ng/ml), which extended significantly above as well as below the conception range, indicating that there is an optimal range for fertility with both an upper and a lower limit. The lower limit is of greater practical importance, and partly to allow for assay variation, we suggest it should be taken as 30 nmol/ml (9.4ng/ml). It provided

[illegible] findings in treated conception cycles suggest that a higher value may be needed after treatment with clomiphene or gonadotropins [illegible] stimulated follicles." (6)

Hull defined the lower limit of progesterone produced in a cycle where ovulation was possible. [illegible] potentially ovulatory cycle (1,3,10,15). Therefore we corresponded with Dr. Chowdhury in order to obtain more precise information about the actual progesterone levels of his study participants. However he replied that all the available information about that study was fully published in the paper, and he has no more detailed information than that which [illegible] 10 patients actually were potentially ovulating (i.e. had a progesterone level high enough to support ovulation.) It is possible that if none of those 10 women had progesterone levels greater that 8ng/ml, that none of them were actually ovulating. [illegible] the endometrium was hostile to implantation in an ovulatory cycle on the OCP unless you [illegible]

The second weakness of the Chowdhury article is the endometrial biopsy histology reporting. Chowdhury states: " The endometrial biopsy showed "hormone effect" as [illegible]

However, Mazur showed that excessive stromal hypertrophy was present in inadvertant endometrial biopsies performed in early gestation (8), and postulated that this was a [illegible] can occur from sampling of the lower uterine segment instead of the fundus(12). Without more description of the actual histology obtained in Chowdhury's biopsies, it is difficult to tell whether or not his specimens actually show "hostile endometrium". (Of further interest is an article by Navot (11) who actually used supraphysiologic doses of Estrogen and progesterone to support the implantation and early pregnancy of women who were without any ovarian function of their own, but who had been recipients of IVF with donor embryos.)

Chowdhury further states: "In 5 out of 35 women in the first cycle treatment group and in 7 out of the 19 in the fourth cycle treatment group the endometrium was so scanty that a [illegible] reasons as well why a tissue sample cannot be obtained, and it does not always mean [illegible] the part of the patient and inexperience are all reasons for insufficient tissue sampling. In fact we are forced to conclude that in 14-35% of his data, the endometrial biopsy material is insufficient for meaningful interpretation.

Thus, the question of whether OCPs produce a "hostile endometrium" with breakthrough ovulations and in such instances are functionally chemical abortifacients remains an unanswered question for the following reasons:

1) Chowdhury's study does not clearly identify a subgroup of patients on the OCP who are clearly ovulating on the OCP. A 4ng/ml progesterone cutoff is inadequate to indicate ovulation , and his raw data is not available for further review at this time.

2) Even if available, a progesterone level >9 ng/ml is only "permissive" of ovulation: i.e. a level < 9 ng/ml precludes ovulation, but a level >9 ng/ml cannot distinguish reliably between ovulatory and nonovulatory cycles. This is because of significant contributions of progesterone production by luteinized unruptured follicles, which are follicles in the ovary which have not released an egg, yet still produce progesterone (6 and others, see literature on polycystic ovarian syndrome.)

3) Chowdhury's endometrial biopsy data are uninterpretable because of the lack of clear documentation of ovulation, and the large number of biopsies with no tissue obtained (ie 14-35% of his endometrial biopsies had no tissue.).

4) Inappropriate ovulation detection measures were utilized in the Chowdhury study (e.g. LH surge testing, ultrasound demonstration of ovulatory follicles or luteal phase endometrial thickening) limiting the study's interpretation and utility.

However, the concept behind Dr. Chowdhury's article is well worth repeating in the context of availability of ultrasound assessment of ovarian and endometrial lining, LH and FSH surge testing, and estradiol and progesterone assays. We would propose a new study to reexamine this issue, and are currently seeking support to implement this.

References, Appendix 3

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