

## Birth Control Pills: Contraceptive or Abortifacient?

Currently the claim that hormonal contraceptives [birth control pills, implants (norplant), injectables (depopovera)] 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-gynecologists, perinatologists, and reproductive endocrinologists who are committed to the protection and preservation 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 conceals 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 sperm 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, severe cramping, passage of tissue, etc.) is not substantiated in any literature we are acquainted with. The "hostile endometrium to abortion" proponents theorize that the losses are pre-implantation, and thus would have no tell-tale clinical or laboratory picture. If the pill roughly approximates the pregnancy rate for women on the pill, this type loss would seem extremely unlikely.

It is impossible to say. Ovulation suppression rates vary from about 95% for the pill to 99% for the Norplant. Cervical mucus factors enter in. Most pill literature estimates 3 to 5% pregnancies per year for combined OCs, less for Depo-Provera, more for Norplant, and minipills.

One may get an idea of the frequency of conception on hormonal contraceptives by considering the ectopic (tubal) pregnancy rates. The frequency of ectopic pregnancies in the US is about 1% of all pregnancies. If pregnancy involves a pre-implantation mistake, then the "on-pill" "ectopic" and normal "on pill" conceptions" rates should be the same - about 1% (unaffected by whether the endometrium is "hostile" or "friendly" to the fetus). Comparing rates of ectopic pregnancies in women on hormonal contraceptives with rates in women not on any type of birth control (except for the minipill) are rarely reported. This would suggest

conception on these agents is also quite rare. If there are millions of "on pill conceptions" yearly, producing millions of abortions, (as seems to be the case) an increase in ectopics in women on hormonal birth control would be expected.

By the way, do you think that the "on pill" contraceptives may be causing a loss of blastocysts in some instances? In Medicine, anything is possible. Do you think that women on hormonal contraceptives would be more likely to have a higher rate of blastocyst loss than normal "on pill" conceptions? We believe the answer is "No."

There are 1,200,000 medical and surgical abortions performed each year in the United States.

The "hormonal contraception is partly abortifacient" theory is not established scientific fact. It is speculation, and 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 embryos into the uterine surfaces a great deal more "hostile" than "hostile endometrium" (e.g., fallopian tube lining). "Hostile endometrium" is not a demonstrated clinical reality.

3. The low incidence of reporting of ectopic pregnancies associated with hormonal contraception would indicate the rarity of actual conception by patients using these methods. (Mifepristone and Norplant apparently are less effective in preventing pregnancies and ectopics).

4. Many factors, including the use of abortifacients, are associated with children. It is not the purpose of this paper to promote nor to oppose

the use of abortifacients in a family, weighing all the factors affecting their own circumstances; besides, unless this is clearly indicated, we are confident that they are not using an abortifacient.

5) This paper is not meant to be the "final word" on the matter. If a 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

control to be used or prescribed by those who hold to the sanctity of human life from the time of conception.

January, 1998

... (1984-1985) ... specialists in obstetrics and gynecology, and a number have subsequently emigrated and/or are on the faculty of teaching hospitals or Universities. This information may be distributed freely to Crisis Pregnancy Centers or other individuals or groups who may have an interest in the subject matter.

Watson, A. Bowes, Jr MD, Professor, Maternal-Fetal Medicine, Chapel Hill, N.C.

Matthew J. Bulfin, MD, general OB-GYN, Ft. Lauderdale, Florida

Byron Camoch, MD, Maternal-Fetal Medicine, Jackson, Mississippi

Steve Calvin, MD, Asst. Prof. Maternal-Fetal Medicine, Minneapolis, Minnesota

Denis Cavanagh, MD, Professor, Gynecologic Oncology, Tampa, Florida

Curtis Cook, MD, Maternal-Fetal Medicine, Asst Clin. Prof, Grand Rapid, Mi.

Steven Cruikshank, MD, Professor OB-GYN, Dayton, Ohio

Joseph E. DeCook, MD, general OB-GYN, Holland, Michigan

Bill Doods, MD, Reproductive Endocrinology; Assistant Professor

Dr. Dan Gambrell, Jr., MD, Clinical Professor, Augusta, Georgia

Donna Harrison, MD, general OB-GYN, Berrien Springs, Michigan

George J. Hays, MD, general OB-GYN, Holland, Michigan

Anthony Paul Levino, MD, Assoc. Prof. OB-GYN, Holland, Michigan

Robert W. Lobel, MD, Assistant Clinical Professor, Urogyn, Albany, N.Y.

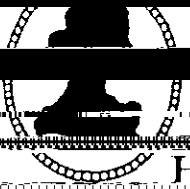
Joe McIlhenny, MD, Obstetrician-Gynecologist, Austin, Texas

Rosenbom, Patterson, Hobbs, MD, OB-GYN, Clinical Assistant, Vienna, Va.

William Staker, MD, Associate Clinical Professor, OB-GYN, Dayton, Ohio

Roy Stringfellow, MD, general OB-GYN, Colorado Springs, Colorado

Robert L. Weeldreyer, MD, general OB-GYN, Holland, Michigan



# HOLLAND GYNECOLOGY & OBSTETRICS, P.C.

Joseph L. DeCook, M.D.

L. Carl Jurgens, M.D.

November 9, 1999

D D Dear Mr. Ure: Enclosed are some pertinent identified literature issues.

We have previously sent you our literature research paper entitled "Hormone Contraceptives:

it that we made on page 2, paragraph 2, 1999. In about 1987, published in 1980 which challenges a point  
of ovulation 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 information is important because it has the potential of the endometrial lining that may  
be an ovulatory site. We have reviewed the original article and commented on it in

our paper. Appendix 2 is enclosed for your consideration. Once again, this material has reference to  
the second paragraph on page 3 of 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,

Joseph L. DeCook, M.D., F.A.C.O.G.  
Donna Harrison, M.D., F.A.C.O.G.  
Camilla Hirsch, M.D., F.A.C.O.G.  
Susan Crockett, M.D., F.A.C.O.G.

JDC/jmu

## Hormone Contraceptives: Controversies and Clarifications

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

communications which might be helpful in clarifying the phenomenon of breakthrough ovulation and the subsequent response of the androgenic levels of the cycle. Chowdhury's article has been added to the appendix frequently to give his testimony.

We have reviewed his 1980 article "Escape ovulation in women due to the missing of low dose combination oral contraceptive pills" by M. Chowdhury et al (2) and have also been in personal correspondence 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 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 combination O.C. The women were asked to monitor their menstrual cycles 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 treated ovulation induction treatment

by measuring serum progesterone in the midluteal phase (i.e. day 25 +/- 2 of cycle) of cycles that conceived. He studied conception cycles because those cycles which conceive occurred; they are the only documentably proven ovulatory cycles. Let's look at his findings:

"I measured progesterone levels in 621 women during the luteal phase of cycles that had been observed with a mean progesterone value of 40.7 nmol/ml (12.7 ng/ml), 95% confidence limits 32.8-50.6 nmol/ml (8.8-16.7 ng/ml) and in 607 women during the luteal phase of cycles that had not conceived with a mean progesterone value of 10.9 nmol/ml (3.3 ng/ml), 95% confidence limits 6.9-14.9 nmol/ml (2.1-4.5 ng/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 (9.4 ng/ml). It provided



... findings in treated conception cycles suggest that a higher value may be needed of treatment with clomiphene and data are being because of the combination of the over-stimulated follicles" (6)

Hull defined the lower limit of progesterone produced in a cycle where ovulation was possible. Below that level of progesterone, ovulation is not possible. Others have suggested the same lower limit of 8.0ng/ml of progesterone as the lower limit of a 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 is already published. Therefore we must conclude that we have no idea how many of his 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 than 8ng/ml, that none of them were actually ovulating. This renders the endometrium was hostile to implantation in an ovulatory cycle on the OCP unless you

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

However, Mazur showed that excessive stromal hypertrophy was present in inadvertent endometrial biopsies performed in early gestation (8) and postulated that this was a

can occur from sampling of the lower uterine segment instead of the mid-uterus (LZ), 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 Estradiol 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 suitable surface for the implantation of the embryo could not be obtained. However, in these women reasons as well why a tissue sample cannot be obtained, and it does not always mean "scanty endometrium". In fact, sampling of the lower uterine segment instead of the mid-uterine part of patients and in patients who are inexperienced are all reasons of insufficient 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 ovary syndrome).

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

4) Important ovulation detection methods 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 assays and assessment of ovarian function and evaluation of the endometrium using LH and TSH surge testing and radiolabeled progesterone assays. We would propose a new study to reexamine this issue, and are currently seeking support to implement this.

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