The Pill and the IUD: Birth Control through Abortion

by Robert Babecka (Technical review provided by Dr. Kathleen M. Raviele, OB/GYN.)

Both the birth control pill and the IUD (intra-uterine device) prevent births after conception has occurred. This is true about every brand of birth control pill and IUD. If you would not willingly terminate the life of an unborn child after conception these products are not for you. If you know of others using these products you should inform them of these facts.

The pill consists of synthetic hormones. There are two types of birth control pills. One contains the hormones estrogen and progestin and is referred to as the "combined pill." The other contains only progestin as the birth control agent.

The combined pill works in two ways, prevention and abortion. It prevents conception by inhibiting ovulation and by making it more difficult for the sperm to reach the egg--this is how the combined pill works most of the time. Sometimes ovulation does occur and the sperm is successful in fertilizing an egg (conception). The new life is then aborted by making the uterus unreceptive to the fertilized egg.

When the combined pill was introduced in the 1960s, medical experts estimated that abortions were responsible for between two and ten percent of prevented births.(Peel and Potts) Since then, pharmaceutical companies have reduced the concentration of estrogen in the combined pill, making it less successful at preventing ovulation. As a result, the percentage of births prevented by abortion in today's pills are higher. Today's pills are worse.

The progestin--only pill prevents birth by abortion most of the time. This pill stops the fertilized egg from implanting in the uterus wall. It only prevents conception a small percentage of the time.

The IUD is a foreign body made of plastic and inserted into the uterus. The IUD does nothing to inhibit ovulation or prevent fertilization. It functions solely to prevent the newly conceived child from implanting in the uterus.

These birth control methods rely on abortion to achieve success. Many women who would not use abortion as a means of birth control are not aware of these facts. As a pro-life individual you should actively spread the word about these birth control products.

References

John Peel and Malcolm Potts, *Textbook of Contraceptive Practice* (Cambridge University Press, 1969) p. 99.

Information Provided with Birth Control Pills Describing How They Work

Information Provided with Intra-Uterine Devices Describing How They Work

Resources

"Abortifacient Brief: The Birth Control Pill," by Brian Clowes, Ph.D.

"Abortifacient Brief: The RU-486 Abortion Pill," by Brian Clowes, Ph.D.

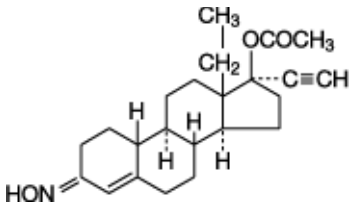
"Abortifacient Brief: Depo-Provera," by Brian Clowes, Ph.D.

"Abortifacient Brief: Implants," by Brian Clowes, Ph.D.

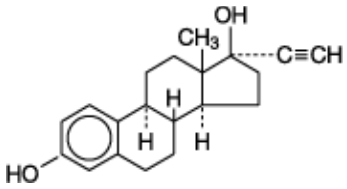
"Abortifacient Brief: The Intrauterine Device," by Brian Clowes, Ph.D.

Information Provided with Birth Control Pills Describing How They Work <BACK>

Each green tablet in the ORTHO-CYCLEN 28 package contains only inert ingredients, as follows: D & C Yellow No. 10 Aluminum Lake, FD & C Blue No. 2 Aluminum Lake, lactose, magnesium stearate, microcrystalline cellulose and pregelatinized starch.



Norgestimate



Ethinyl Estradiol

CLINICAL PHARMACOLOGY

ORAL CONTRACEPTION

Combination oral contraceptives act by suppression of gonadotropins. Although the primary mechanism of this action is inhibition of ovulation, other alterations include changes in the cervical mucus (which increase the difficulty of sperm entry into the uterus) and the endometrium (which reduce the likelihood of implantation).

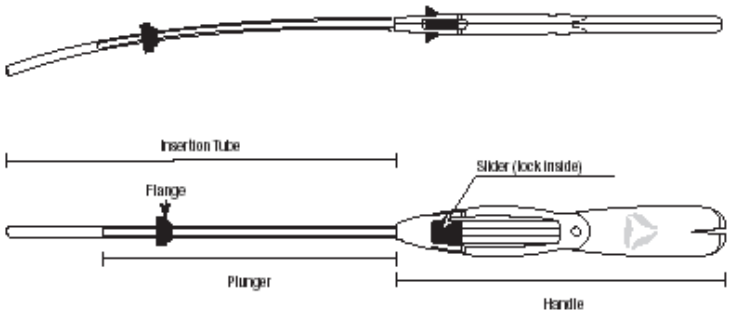
Receptor binding studies, as well as studies in animals and humans, have shown that norgestimate and 17-deacetyl norgestimate, the major serum metabolite, combine high progestational activity with minimal intrinsic androgenicity (90-93). Norgestimate, in combination with ethinyl estradiol, does not counteract the estrogen-induced increases in sex hormone binding globulin (SHBG), resulting in lower serum testosterone (90,91,94).

ACNE

Acne is a skin condition with a multifactorial etiology. The combination of ethinyl estradiol and norgestimate may increase sex hormone binding globulin (SHBG) and decrease free testosterone resulting in a decrease in the severity of facial acne in otherwise healthy women with this skin condition.

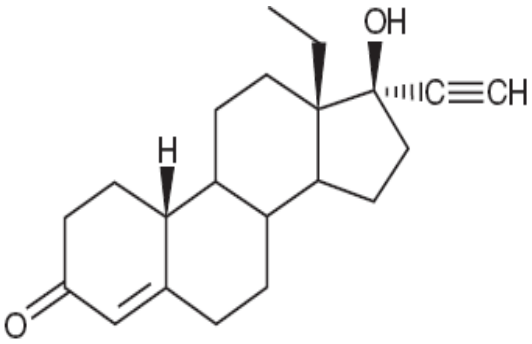
Information Provided with Intra-Uterine Devices Describing How They Work <BACK>

Diagram of Inserter



Mirena is intended to provide an initial release rate of 20 μg/day of levonorgestrel.

Levonorgestrel USP, (-)-13-Ethyl-17-hydroxy-18,19-dinor-17α-pregn-4-en-20-yn-3-one, the active ingredient in Mirena, has a molecular weight of 312.4, a molecular formula of $C_{21}H_{28}O_2$, and the following structural formula:



CLINICAL PHARMACOLOGY

Levonorgestrel is a progestogen used in a variety of contraceptive products. Low doses of levonorgestrel can be administered into the uterine cavity with the Mirena intrauterine delivery system. Initially, levonorgestrel is released at a rate of approximately 20 μg/day. This rate decreases progressively to half that value after 5 years.

Mirena has mainly local progestogenic effects in the uterine cavity. Morphological changes of the endometrium are observed, including stromal pseudodecidualization, glandular atrophy, a leukocytic infiltration and a decrease in glandular and stromal mitoses.

Ovulation is inhibited in some women using Mirena. In a 1-year study approximately 45% of menstrual cycles were ovulatory and in another study after 4 years 75% of cycles were ovulatory.

The local mechanism by which continuously released levonorgestrel enhances contraceptive effectiveness of Mirena has not been conclusively demonstrated. Studies of Mirena prototypes have suggested several mechanisms that prevent pregnancy: thickening of cervical mucus preventing passage of sperm into the uterus, inhibition of sperm capacitation or survival, and alteration of the endometrium.

Clinical Pharmacokinetics

Following insertion of Mirena, the initial release of levonorgestrel into the uterine cavity is 20 μg/day. A stable plasma level of levonorgestrel of 150-200 pg/mL occurs after the first few weeks following insertion of Mirena. Levonorgestrel levels after long-term use of 12, 24, and 60 months were 180±66 pg/mL, 192±140 pg/mL, and 159±59 pg/mL, respectively. The plasma concentrations achieved by Mirena are lower than those seen with levonorgestrel contraceptive implants and with oral contraceptives. Unlike oral contraceptives, plasma levels with Mirena do not display peaks and troughs.

The mean ± SD levonorgestrel endometrial tissue concentration in four women using levonorgestrel intrauterine systems releasing 30 μg/day of levonorgestrel for 36-49 days was 808 ± 511 ng/g wet tissue weight. The endometrial tissue concentration in 2 women who had been taking a 250 μg levonorgestrel–containing oral contraceptive for 7 days was 3.5 ng/g wet tissue weight. In contrast, fallopian tube and myometrial levonorgestrel tissue concentrations were of the same order of magnitude in the Mirena group and the oral contraceptive group (between 1 and 5 ng/g of wet weight of tissue).

The pharmacokinetics of levonorgestrel itself have been extensively studied and reported in the literature. Levonorgestrel in serum is primarily bound to proteins (mainly sex hormone binding globulin) and is extensively metabolized to a large number of inactive metabolites. Metabolic clearance rates may differ among individuals by several-fold, and this may account in part for wide individual variations in levonorgestrel concentrations seen in individuals using levonorgestrel–containing contraceptive products. The elimination half-life of levonorgestrel after daily oral doses is approximately 17 hours; both the parent drug and its metabolites are primarily excreted in the urine.

Pharmacokinetic studies of this product have not been conducted in special populations (pediatric, renal insufficiency, hepatic insufficiency, and different ethnic groups).

Drug-Drug Interactions

The effect of other drugs on the efficacy of Mirena has not been studied.

INDICATIONS AND USAGE

Mirena is indicated for intrauterine contraception for up to 5 years. Thereafter, if continued contraception is desired, the system should be replaced.

Mirena is recommended for women who have had at least one child.