The pill is not an abortive agent

Tom K.A.B. Eskes*

Department of Obstetrics and Gynecology, University of Nijmegen, Sint Radboudziekenhuis, Geert Grooteplein Zuid 14, 6525 GA Nijmegen, Netherlands

The original work by Pincus and Chang [1] on the effects of progesterone and related compounds has had a great impact on women's fertility awareness. The birth of the ‘pill’ has led to the use of oral contraceptives (OCs) by millions of women throughout the world. Eventually, changes in the dose and composition of the pill were necessary in order to minimize side effects. This has given rise to the development of the sub-50 pills (less than 50 mcg of ethinylestradiol) and of various progestagens.

A different ‘side effect’ of the pill can also be noted when one opposes the use of the pill from a religious point of view [2]. It appears that one of the arguments increasingly heard from pro life organisations concerns the possible abortive action of OCs.

When contra-arguments exist for such action they have to be found in biological data on: (1) the working mechanism of OCs; (2) pregnancy during OC exposure; and (3) the morning after pill.

1. The working mechanism of OCs

The menstrual cycle is initiated and regulated by the hypothalamus and pituitary gland [3]. The hypothalamus activates the pituitary gland through luteinizing hormone releasing hormone (LHRH). The pituitary gland produces luteinizing hormone (LH) and follicle stimulating hormone (FSH).

FSH stimulates the growth of the ovarian follicles also called Graafian follicles containing the eggs. The granulosa cells of the follicle produce oestrogenic hormones in increasing quantities. Due to these oestrogens LH is suddenly produced in a peak value. This results in ovulation and thereafter in corpus luteum formation, progesterone and oestrogen production.

The oestrogenic component of the oral contraceptives inhibits FSH release very strongly. This action induces the most important contraceptive property of OCs. The progestational component of the oral contraceptive inhibits the LH-peak resulting in anovulation.

Ovarian follicles can be visualized with ultrasound techniques. During the use of low dose OCs ovarian follicles can be shown with a diameter of up to 18 mm [4,5]. These follicles however, do not produce oestrogens and they can be considered as inactive. In addition, these follicles have a delayed growth and an LH peak is not observed. This can also be explained from the fact that the progestational component of all sub-50 OCs induces a delay of the release of hypothalamic LH-RH [6].

2. Pregnancy during OC exposure

OCs are reliable contraceptive agents. From prospective studies in which one or more pills were not taken at various moments of the ‘cycle’, one can conclude that the first and last days of the ‘cycle’ are crucial in allowing recovery of the gonadostat (hypothalamus and pituitary gland) or not. Such a recovery results in the initiation of the process of ovum maturation due to FSH and LH release.

Pregnancy is not only possible when pills are forgotten, but it can also occur in women by regular and accurate pill use. In these women the bio-availability can be disturbed for instance by pathologic resorption in the gut, concomitant use of medication, or increased enzymatic metabolism. In all these instances pregnancy can occur despite pill exposure.
After fertilization of the ovum a series of mitotic divisions occur. From the inner cell mass the embryo will develop. The outside blastomeres form the chorionic villi and placenta. The first product of these villi is the glycoprotein: human chorionic gonadotrophin (hCG) which is already produced before implantation. HCG has a strong stimulating action on the ovarian corpus luteum. This produces steroid hormones in increasing amounts which in turn makes the endometrium receptive for implantation during the so called decidua reaction. The effects of OCs on the endometrium can be summarized as: proliferative changes by oestrogens and secretory pseudodecidual changes through progesterational agents.

In addition, the embryo itself has a strong hormonal signal function [7], thus making implantation possible during the use of OCs when an escape-ovulation occurs. Endometrium is not even necessary for implantation when one observes ectopic pregnancies.

An early exposure of embryo and fetus to teratogenic effects of reproductive steroids only holds for the synthetic stilbestrol or DES hormone and for masculinizing progestational agents with an androgenic action such as ethisterone and norethindrone. The currently available OCs do not have teratogenic or mutagenic action.

3. The morning after pill

An increased dose of reproductive steroids is used for postcoital contraception. This medication known as the morning after pill, is based on the observation in monkeys that reproductive steroids can interfere with ovum implantation and development [8]. One has to realize however that a placebo-randomised trial has never been performed in the human. The only way to distinguish myth from reality in postcoital contraception is to calculate statistical chances. Silvestre et al. [9] in analyzing several studies on postcoital contraception have expressed serious doubt on the efficacy of the morning after pill.

In contrast, antiprogesterones (Mifepristone) are very effective as postcoital contraception [10].

It is clear that OCs are effective for contraception. It is also clear that the working mechanism of OCs consists mainly of action on the hypothalamus/pituitary gland by inhibiting FSH and LH production and not primarily on the endometrium. Another argument for the non-abortive action of OCs is the fact that pregnancies are observed during the irregular or even regular use of OCs. The embryo itself does have a strong signalling function permitting implantation. Even without endometrium implantation is possible as seen in ectopic pregnancies.

No arguments can be found for an abortive action of OCs even when these are used in high doses. If one would oppose the use of OCs in general this can not be done with arguments of non-existing biological data.

References