INTRODUCTION

Induction of ovulation whether medical or surgical is considered to be a management for chronic anovulatory state. Chronic anovulation is a major cause of infertility and in most conditions it is possible to restore normal fertility by careful selection of the appropriate method of induction of ovulation (Franks et al; 1994).

Clomiphene citrate has been available for induction of ovulation for nearly 30 years and still has an important place in the management of women with an ovulatory menses and of estrogenized subjects with amenorrhea. Most women with amenorrhea with a positive progesterone withdrawal test have clinical, biochemical and ultrasound evidence of polycystic ovarian syndrome (Fox et al; 1991).

Gonadotropin-releasing hormone remains the treatment of choice for induction in women with hypogonadotropic hypogonadism of hypothalamic origin (Martin et al; 1993).

Gonadotropin-releasing hormone analogs with gonadotropine in polycystic ovarian syndrome. PCOS is typically associated with hypersecretion of LH and there are now several studies which have shown a link between persistently elevated levels of concentration of LH and a poor outcome of induction of ovulation, so it seems logic to suppress of endogenous LH by administration of GnRH agonist analogs during gonadotropine induced ovarian stimulation may improve the rate of successful pregnancy (Homburg et al; 1990).

It has now been reported that there is a reduced rate of multiple follicular development of purified FSH in very low doses (Polson et al; 1987).

A recent report using human endometrial stromal cells in vitro indicates that FSH, LH and HCG extracted from the urine of either post menopausal women or pregnant women stimulate intracellular Cyclic AMP and induced decidualization (Tang et al; 1993).

Paradoxically, little is known about the pharmaco-dynamics and pharmaco-kinetics of LH and FSH during such stimulation.

Recently, the existence was shown of a sharp FSH threshold concentration above which the ovary responds with gonadotrophins in hypogonadotrophic women, this FSH threshold concentration was independent of the co-administration (Van Weissenbruch; 1990).