INHIBITION OF LACTATION BY QUINESTROL

by

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The need to suppress lactation is not so common in our country, although in the West breast feeding is becoming increasingly unpopular (Iliya *et al,* 1966) so much so that according to Daniel *et al,* (1967) in U.K. breast feeding is an exception rather than the rule. Over two-thirds of American women today prefer artificial feeding to nursing whereas breast feeding is universal with our patients. With us the occasion for inhibiting lactation arises only when there is a perinatal loss or a contra-indication for breast feeding. Little wonder then that hardly any work is done in our country on inhibition of lactation.

An ideal lactation suppressant is still being looked for. Estrogens are the most widely used agents for the purpose although combinations of estrogens and androgens are finding increasing favour (Jones and Tanner, 1962; Schneider *et al,* 1964; Iliya *et al,* 1966). Quinestrol (Estrovis), the cyclopentylenolether of ethinyl estradiol claims to suppress lactation with a single oral dose. A double blind study was undertaken by us to compare the efficacy of quinestrol with that of traditionally employed ethinyl estradiol. When this established the efficacy of a single oral dose of quinestrol in inhibiting lactation the study was further extended to find out whether doubling its dosage improved its efficacy.

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Material and Methods

Part I

Sixty patients who had a stillbirth were taken up for the initial study without regard to age, parity or previous nursing experience. Patients with an odd serial number in the study were given a tablet labelled S_1 within 2 hours of delivery to be followed by tablets labelled S_2 given 8 hourly for 14 doses over the next 5 days. Patients with an even serial number were given a tablet labelled T_1 within 2 hours of delivery to be followed by tablets labelled T_2 given 8 hourly for 14 doses over the next 5 days. At the end of the study when the code was revealed it disclosed that tablets S_1 and S_2 contained 0.2 mg ethinyl estradiol whereas tablets T_1 contained 2 mg of Quinestrol and tablets T_2 were placebo. Thus one group of patients received 0.2 mg. ethinyl estradiol thrice a day for 5 days whereas the other received a single tablet of 2 mg. of Quinestrol followed by placebo tablets. Breast binders and diuretics were not

employed nor was the intake of fluids restricted.

Patients were examined every morning to evaluate the degree of lactation inhibition by assessing pain, discomfort, engorgement, congestion and leakage. Pain and discomfort were assessed as follows:

0—absent
1—admission on direct questioning
2—voluntary admission without being questioned.

Engorgement and congestion were scored as 0, 1 or 2 on the basis of tissue tension on palpation of the breasts. Leakage was designated as 0, 1 or 2 depending upon the amount of milk flow, if any, following a single gentle kneading of the areola.

Inhibition of lactation was considered good if the total score (i.e. pain and discomfort, engorgement and congestion, and leakage) on the 5th day was 0 and was not more than 2 on any single day, fair if 5th day score was either 1 or 2 and the score was not more than 3 on any single day and poor if 5th day score exceeded 2 or any single day score exceeded 3.

Patients were followed up in the postnatal clinic during the 3rd or the 4th postpartum week. A specially trained social worker was sent for the follow up study to the homes of those patients who did not turn up to the postnatal clinics. The results of the follow-up study were assessed as good if leakage, pain and engorgement were absent, fair if there was slight leakage but no pain or engorgement and poor if leakage was accompanied by pain or engorgement.

Part II

Sixty patients who had a stillbirth were taken up for this study without any selection as regards age, parity, previous nursing experience. They were given 4 mg. of Quinestrol orally within 2 hours of delivery. Breast binders, diuretics and fluid restriction were not employed.

Evaluation of the degree of lactation inhibition was exactly similar to that in Part I of the study.

Results

Part I

Table I shows the lactation inhibition achieved during the first five days after delivery in the two groups. There is no significant difference between the two groups and the immediate results are good or fair in 93 per cent and poor only in about 7 per cent. Table II shows the results of follow-up study in the two groups. There is no significant difference between the two groups and the follow-up results are good in about 75 per cent of the cases.

Part II

Table III gives the immediate and follow-up study results with 4 mg. dosage of Quinestrol.

TABLE I

Immediate Results in Part I of the Study (Double Blind Trial)

	Good	Fair	Poor	Total
Quinestrol	15	13	2	30
Ethinyl estradiol	13	15	2	30

TABLE II

Follow-up Results in Part I of the Study (Double Blind Trial)

	Good	Fair	Poor	Patients not traceable	Total
Quinestrol	18	5	0	7	30
Ethinyl estradiol	15	4	1	10	30

TABLE III

Results with 4 mg. of Quinestrol (Part II of the Study)

	Good	Fair	Poor	Total	Not traceable
Immediate	50 (83.3%)	4 (6.6%)	6 (10%)	60	—
Follow-up	39 (97.5%)	1 (2.5%)	0	40	20

Side effects: No patient in the entire study exhibited any side effects.

Discussion

Part I

The most important revelation of the present study is that Quinestrol given in a single oral dose within 2 hours of delivery is as effective in inhibiting lactation as the traditional administration of ethinyl estradiol over a period of 5 days. It is a universal experience that patients who have lost their babies during or soon after delivery are psychologically disturbed and are very keen to leave the hospital immediately to avoid the company of other mothers who are happily and proudly nursing their babies. They are also indifferent towards medication during immediate postpartum days. In addition, our hospitals, overcrowded as they always are, are very hard pressed for beds in the postnatal wards. Besides, single dose therapy is a boon to both the patients and the nursing staff. The supreme advantages of a single dose oral lactation suppressant are, thus, very obvious and need no further emphasis.

Kuvu (1968) carried out a double blind comparative trial of quinestrol with ethinyl estradiol and found successful lactation inhibition during the first postpartum week in 67 per cent of cases with quinestrol (2 mg. dosage) and in 63 per cent of cases with ethinyl estradiol. Our contention that lactation inhibition by a single oral dose of quinestrol is as good as by the traditional 5 day treatment with ethinyl estradiol is supported by his results.

Morris (1967) found favourable lactation inhibition by single oral dose of quinestrol in 78 per cent of the patients.

Engineer and Das (1971) found good lactation inhibition by quinestrol in 67% of their cases.

Our results viz. good lactation inhibition in 50% of cases and fair inhibition in 43.3% of cases with a 2 mg. dosage of quinestrol are comparable with the results of other workers. The exact percentages of patients with successful lactation inhibition by single oral dose quine-

strol in the different series are not comparable because the assessment of the degree of lactation inhibition is obviously subject to personal impressions and the finer criteria for assessment vary in the different series. Yet, the conclusion is inescapable that a single oral dose of quinestrol is a highly efficient and most convenient method of inhibiting lactation.

Part II

Once the efficacy of a single dose of quinestrol in inhibiting lactation was established we tried to find out if doubling the dose of quinestrol would give still better results.

Table IV compares the immediate results in 30 cases of the Part I study who had received 2 mg. of quinestrol with those in 60 cases of Part II study who had received 4 mg. of quinestrol while Table V compares the follow up results in the two groups. The superior results with 4 mg. dosage need no emphasis at all.

Engineer and Das (1971) found that when the dose was reduced to 1.6 mg. the percentage of good lactation inhibition dropped to 41 as compared with 76 with 2 mg. dosage. Morris (1967) also found that lowering of the dosage below 2 mg. resulted in unsatisfactory lactation inhibition. He further found that the results with 2 mg. dose and 4 mg. dose were similar. However, our findings demontrate that 4 mg. dosage is positively superior to 2 mg. dosage. It may be mentioned that Morris studied only 19 patient with 4 mg. dosage. Since our results are far better with 4 mg. dosage than with 2 mg. we have no hesitation in recommending a 4 mg. dosage of quinestrol for lactation inhibition in our patients.

Lastly, so far no ill-effects have been reported with the use of quinestrol for inhibiting lactation and no cases of thrombo-embolism are reported. But Hakim *et al* (1969) have recently demon-

TABLE IV

Immediate Results

Dose of Quinestrol	Good	Fair	Poor	Total
2 mg.	15 (50%)	13 (43.3%)	2 (6.6%)	30
4 mg.	50 (83.3%)	4 (6.6%)	6 (10%)	60

TABLE V

Follow-up Study

Dose of Quinestrol	Good	Fair	Poor	Total	Not traceable
2 mg.	18 (78.2%)	5 (21.8%)	0	23	7
4 mg.	39 (97.5%)	1 (2.5%)	0	40	20

strated a rise in plasma Factor IX levels in patients given estrogens for lactation inhibition. Campbell and Turnbull (1967) found significantly higher incidence of thrombo-embolism in patients who had lactation suppressed. In view of these reports the effect of quinestrol on clotting mechanism seems warranted.

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