

diethylstilboestrol administered in divided doses up to a total of 210–330 mg. during the course of nine days. This dosage, it may be noted, is higher than is usually given, and work carried out many years ago indicated that a total of 45–50 mg. of diethylstilboestrol during four to seven days is generally adequate to inhibit lactation (Jeffcoate, Lister, Hargreaves, and Roberts, 1948).

There are some who question the efficacy of oestrogen therapy to inhibit lactation, irrespective of dosage, and the literature pertaining to this was reviewed by Hodge (1967). Differences in experience and opinion may in large measure reflect the timing of the administration of the hormone. The evidence suggests that the sooner after delivery oestrogen is given the more likely is it to inhibit breast activity. Oestrogens will not suppress lactation which is already established. Whatever be their efficacy, there is no question that oestrogens are widely used with the object of inhibiting or suppressing lactation, and it is therefore of considerable importance to determine whether all or certain of the preparations available predispose to puerperal thromboembolism, and whether suppression of lactation by itself (without any hormone being administered) has any effect.

So many possible agents are concerned in the aetiology of puerperal venous thrombosis and embolism that it is difficult to assess the significance of each. The presence or absence of breast-feeding cannot readily be separated from factors, such as age, parity, difficult delivery, and general health, which may have governed a decision to inhibit lactation.

This difficulty is emphasized by an analysis of the hospital inpatient inquiry returns for 1962–4, which was published by Hill and Wilson (1968). After correcting for age, parity, and mode of delivery, they concluded that puerperal thromboembolism occurs twice as often after a stillbirth as after a live birth. Information about the administration of oestrogen in their cases was not available, but lactation must inevitably have been inhibited by one means or another after the birth of a dead baby. This still leaves open the question of whether the circumstances of the stillbirth may in themselves have played some part in predisposing to thromboembolism.

In a previous report (Jeffcoate and Tindall, 1965) based on a study of the experience at the Liverpool Maternity Hospital during the years 1953–63, it was shown that of the associated factors examined those most significantly related to the occurrence of puerperal thromboembolism were (1) the age and parity of the patient and (2) the mode of delivery. The incidence was found to be three times higher after caesarean section than after any form of vaginal delivery. At the time of the original inquiry, however, the lactation habit of affected women was not studied. When the possible importance of breast-feeding was raised by the Cardiff workers it was decided to re-examine our original material for the years 1956–63, and to add to it (1) the experience of the same hospital during the years 1964–6 and (2) the experience of Mill Road Maternity Hospital, Liverpool, during the years 1958–66 inclusive. Cases of puerperal superficial phlebothrombosis, a condition which presents a different clinical picture and has other aetiological factors, were not included.

METHOD AND CRITERIA

The statistics presented here are based on the examination of case records, a method which has the disadvantages attached to any retrospective survey. In this instance it means that only *recorded* cases of deep venous thrombosis and embolism are included. The condition therefore had to be clinically evident, and the diagnosis, which is always subject to individual clinical opinion, beyond reasonable doubt. Similar diagnostic criteria were in use at both hospitals at the times in question. The figures are made more incomplete by the fact that they do not include those cases in which women, having developed deep venous thrombosis after discharge from hospital, were either treated at home or were referred to another hospital for treatment. Though we have reason to believe there were not many of these, it has to be accepted that the true incidence of clinically evident puerperal thromboembolism is somewhat higher than is here stated. The lack of follow-up studies, however, applies equally to breast-feeding and non-breast-feeding women.

To provide control series a study was made of the records of one out of every 100 consecutive women delivered in the same hospitals during the same period.