administration appears to raise the incidence from approximately 1 to 2 per 1,000 deliveries. Again, women having an operative delivery already carry a relatively high risk of thromboembolism; ethinyloestradiol given to them may have a summation effect to increase the risk six times. Fundamental determining factors to puerperal thromboembolism are combinations of relative late age and traumatic delivery and there may well be others such as obesity and hypertensive states complicating pregnancy. It still remains uncertain how often it is these which prompt the administration of oestrgoens to inhibit lactation.

At this stage of knowledge it may be concluded that, provided oestrogens have the inhibitory effect on breast activity which is credited to them, and which our observations lead us to accept as genuine, their administration does not increase the risk of puerperal thromboembolism to an unacceptable degree. This is true for women at low risk, but for those who are advanced in years and who are subjected to operative delivery involving considerable trauma there is some reason to hesitate to add one more predisposing factor, even if it be a minor one. Obesity and a past history of thromboembolic disorders might also sometimes be considered to be contraindications to the administration of oestrogens in the puerperium. When oestrogens are given, however, it would seem wise to use the lowest dose compatible with efficient inhibition of lactation.

A firm conclusion about the role of therapeutic doses of oestrogen in predisposing to puerperal thromboembolism may not be possible without controlled prospective studies, and these are already in progess. Because the incidence of clinically evident disease is relatively low, this will take time. Meanwhile, the results of this retrospective study are reported to add to the evidence which needs to be collected from many sources.

References

Arneil, G. C. (1967). Scottish Health Service Study, No. 6. Edinburgh.

Burns, D. (1957). Med. Offr, 98, 205.

Daniel, D. G., Campbell, H., and Turnbull, A. C. (1967). Lancet, 2,287.

Daniel, D. G., Bloom, A. L., Giddings, J. C., Campbell, H., and Turnbull, A. C. (1968). Brit. med. J., 1, 801.

Dykes, R. M. (1957). Lancet, 2, 230.

Garrey, M. M., Paterson, M. M., and Evans, J. M. (1964). Lancet, 2, 1057.

Hill, G. B., and Wilson, W. A. (1968). Med. Offr, 119, 147.

Hodge, C. (1967). Lancet, 2, 286.

Inman, W. H. W., and Vessey, M. P. (1968). *Brit. med. J.*, 2, 193.

Jeffcoate, T. N. A., Lister, Ursula M., Hargreaves, Betty, and Roberts, H. (1948). *Brit med. J.*, 2, 809.

Jeffcoate, T. N. A., and Tindall, V. R. (1965). Aust. N.Z. J. Obstet. Gynaec., 5,

Ministry of Health (1957, 1960, 1963, 1966). Reports on Confidential Enquiries into Maternal Deaths, England and Wales. H.M.S.O., London.

Ministry of Health (1957a). Annual Report of the Chief Medical Officer.

H.M.S.O., London.

Simpson Memorial Pavilion (1965). Annual Medical and Clinical Report. H.M.S.O., London.

Vessey, M. P., and Doll, R. (1968). Brit. med. J., 2, 199.

[From the British Medical Journal, Oct. 5, 1968, pp. 1-2] THROMBOSIS AND INHIBITION OF LACTATION

An association between oral contraceptives and thromboembolism has now been the subject of many reports, with oestrogens rather than progestogens incriminated as the likely cause of it (1, 2). Moreover, puerperal venous

NOTE.—Numbered references at end of article.