degrees, will be what matters, but they will be quite essential for ensuring proper growth and distribution of scientific resources in the regions. It is not inconceivable that some may have started their scientific careers as pathologists.

You make the point that the pathologist has no need of a new career structure, but the report does not propose that the careers structure of medical-graduate laboratory workers should be altered nor their numbers reduced. It does make it clear that an important aim is to attract more first-class scientists, medically qualified or otherwise. The proposed National Hospital Scientific Council, responsible for keeping an eye on the needs of the whole hospital scientific service, pathologists included, should do much to ensure that clinical and laboratory priorities, as regards buildings, staff, and equipment, no longer have to compete at local level. This, it seems to me, can do nothing but good.

As an admittedly "academic," medically qualified biochemist, who is none the less fully involved in everyday hospital work, I personally welcome the report as a timely and courageous attempt to solve a number of complex and difficult problems before they get out of hand. I can but hope that the unimaginative, not to say prejudiced, tone of your leading article will not be regarded as authoritative medical comment on what the report contains.—I am, etc.,

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REFERENCE

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Inhibition of Lactation with Combined Oestrogen and Progestogen

SIR,—Having followed recent correspondence concerning the inhibition of lactation, we think it of interest to report two trials that compared four methods for suppressing the *initiation* of lactation in 136 patients followed up for six weeks after delivery.

Quinestrol (Estrovis) given as a simple 2 mg. or 4 mg. dose immediately after delivery was compared with an untapered course of stilboestrol, 10 mg. thrice daily for five days (total dose 150 mg.), in a random, but not blind, trial. In a second, double-blind trial a tapered course of norethynodrel with mestranol (Enavid)—30 mg. initial dose followed by 10 mg. thrice daily for three days, 10 mg. twice daily for three days, and 10 mg. daily for three days—was compared with a tapered course of stilboestrol—15 mg. initial dose followed by 5 mg. thrice daily for three days, 5 mg. twice daily for three days, and 5 mg. daily for three days (total dose 105 mg.).

The results are shown in the Table. A failure implied that additional suppressive measures were required either for engorgement with severe discomfort or because of abundant lactation. The failures are divided into those occurring within eight days of delivery, and those occurring after the patient had left hospital.

	Total No.	Early Failure	Late Failure	Total Failures
Untapered stilboestrol	21	2	15	17 (81%)
Quinestrol	27	18	3	21 (78%)
Tapered stilboestrol	46	4	14	18 (39%)
Tapered Enavid ..	42	—	7	7(16·7%)

Quinestrol failed to suppress lactation within eight days of delivery in 5 of the 8 patients who received 2 mg., and 13 of 19 patients given 4 mg. We therefore abandoned the use of a single dose of quinestrol as a method of suppressing lactation. The tapered course of stilboestrol was considerably better than the untapered course, though the total dose given was less. The combined use of oestrogen and progestogen (Enavid) gave the lowest failure rate, and this confirms a previous report.[1]

There is no reason to believe that the usual absence of lactation before labour is solely due to inhibition by oestrogens.[2] Whether the apparently more efficient combination of oestrogen and progestogen might also reduce the risk of puerperal thromboembolism which appears to be associated with the use of high doses of oestrogens alone for the suppression of lactation in certain women[3–4] remains to be seen.—We are, etc.,

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3 Daniel, D. G., Campbell, H., and Turnbull, A. C., *Lancet*, 1967, 2, 287.
4 Jeffcoate, T. N. A., Miller, J., Roos, R. F., and Tindall, V. R., *Brit. med. J.*, 1968, 4, 19.

Oestrogens and Puerperal Thrombosis

SIR,—The publication of the paper by Daniel and his colleagues[1] on the possible relation between puerperal thrombosis and oestrogens given to suppress lactation led us to look into the records at University College Hospital for the years 1954–64. During this time the standard of recording is believed to have been high and the hospital's policy was to readmit patients who developed any illness related to childbirth after going home. All oestrogen therapy was recorded with dates.

In our series, as in those reported by Daniel and his coworkers and by Jeffcoate and his (5 October, p. 19), the incidence of thrombosis was found to be related to both maternal age and operative delivery. Two control groups have been selected: the first—unmatched controls—was made up of "next deliveries" drawn from the delivery register, the second—matched controls—consisted of "next deliveries of the same age." Out of 103 recorded cases of thrombosis among 15,472 deliveries in 11 years, thrombosis arose only as an antepartum or intrapartum condition in 25. Five patients suffered thrombosis after two or more deliveries in the period. When all but the first were excluded, 71 puerperal cases remain; two further cases were excluded because their notes were incomplete.

The day of recorded onset of thrombosis, given in the Table, shows that superficial thrombosis is almost only seen in the first week; deep vein thrombosis in this series occurs predominantly after the first week. It seems important that in future surveys on puerperal thrombosis some means must be found to have adequate records of patients with thrombosis and their "controls" for a month after delivery.

Day of onset	1	2	3	4	5	6	7	8	9	10	11	12	12+	
Superficial	—	8	9	9	4	7	2	5	1	—	1	—	2	48
Deep	—	1	2	1	—	—	1	2	5	1	1	1	6	21
Total superficial	39							9						48
Total deep	5							16						21

Daniel and his colleagues took as their measure of oestrogen administration the patient's method of feeding her baby on the seventh day. In the U.C.H. series only 12 women suffering from superficial thrombosis had had oestrogens by the seventh day, and of them five had started their course after the onset of thrombosis. Two more had started taking stilboestrol on the eighth and ninth days, when anticoagulant treatment was already started. It seems that some women decided to give up breast feeding on finding themselves unwell. Analysing all cases of thrombosis, including those arising after the seventh day, seven patients had received oestrogens by the day of onset of the thrombosis, whereas six matched controls had had it by the corresponding day. There were, however, nine patients who started oestrogens after thrombosis had occurred, while there were only five among matched controls. Only six of the 21 cases of deep vein thrombosis or embolism had had oestrogens before the onset of the disease, and six of the matched controls by the corresponding day.

The figures provided by this survey show that, although estimating from the records of the unmatched controls about 5,000 women received oestrogens in the first week for suppression of lactation in the 11 years, the number of women developing superficial thrombosis after oestrogen therapy was too small to show any effect compared with matched controls. Deep vein thrombosis was found to occur at a time when recording is too unsatisfactory for firm deductions. There was, however, in this series no measurable effect of oestrogens on thrombosis rate. There were no deaths.

The dosage of oestrogens given is based on the supposition that it is the *rate* of change of oestrogen level which induces the onset of lactation, and, therefore, that the method of choice is to use the minimum effective first dose followed by slow withdrawal for suppression. Our total dosage was only one-third to one-fourth of that used at Cardiff, and administration was spread over 12 days rather than nine. Pain, withdrawal bleeding, and oestrogen-release filling of the breasts at the end of the course was seldom seen.—We are, etc.,

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1 Daniel, D. G., Campbell, H., and Turnbull, A. C., *Lancet*, 1967, 2, 287.