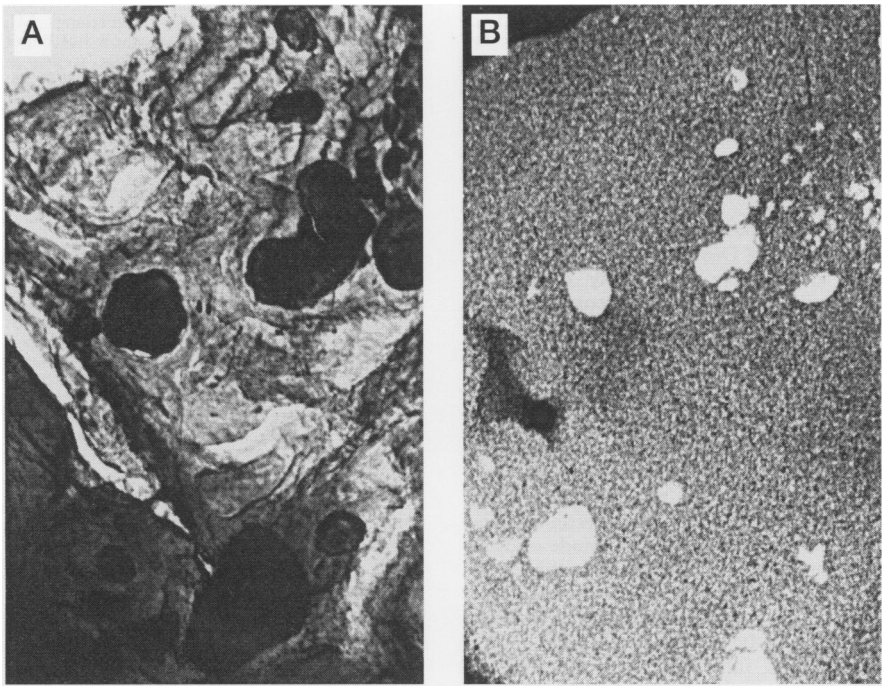

Figure 2 Calcified comedo-type ductal carcinoma in situ of breast in a thick section (1200 μm deep) stained with 0·0001% (w/v) alizarin red S (A), and a corresponding sliced specimen radiograph of the same tissue (B).

for staining other components of thick tissue sections.[3 4]

Literal in-depth assessment of microcalcification and the changes in macromastia[5] may also facilitate appropriate analysis of mammographic lesions and the diagnostic histological dilemmas resulting from reduction mammoplasty[6] for macromastia.

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Prospective comparative study of computer programs used for management of warfarin

Poller *et al* refer to their experience with, among others, the Hillingdom system for computer-assisted warfarin maintenance.[1] Their numbers are small, and we should like to record our current experience with a larger series. Nearly all of our patients are given a target INR of 2·8. To compare these as nearly as possible with those of the above authors, who used a range of 2·0 to 3·0, we have used 2·3 to 3·3. The ranges used by these authors are narrow, and divergence from them does not necessarily mean that anticoagulation is at a level which is either ineffective or dangerous. We followed their division into the first 26 weeks of treatment and the period, if any, after that point. The results are shown in the table.

With the important exception of the interval between visits, our experience is not significantly different from that of Poller *et al* as shown in their table 4. Our mean intervals, both for early and later periods of treatment, are longer than theirs: this may be partly attributable to the maximum permissible interval having been increased from eight to 10 weeks during the period under consideration. We can add that the average interval at the latest visit was 7·04 weeks.

We have only six patients (187 visits) to compare with those who had a higher target INR. Having so few, we would only say tentatively that the average intervals before and after 26 weeks were 2·24 and 3·36 weeks, respectively, and that the interval at the latest visit averaged 3·83 weeks.

The mean interval is important, both for the convenience of patients and economy in the use of hospital resources. Our data support the inherently probable propositions that intervals become longer as treatment proceeds, and are much shorter with higher INR targets. Because they require more frequent attendances, and also because they are more difficult to achieve, high targets require much justification on grounds of clinical necessity.

	No of visits	*% visits in range*	*% visits above range*	*% visits below range*	*Average interval (weeks)*
Up to 26 weeks	2058	44·1	24·8	30·9	3·11
After 26 weeks	5332	50·5	24·8	24·6	5·81

361 patients; 7390 visits; target INR 2·8; range 2·3–3·3.

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Vasculopathy and antiphospholipid antibodies in systemic lupus

We read with great interest the article by Ellison and colleagues showing intramural deposition of platelet derived material in small cerebral vessels in four patients with systemic lupus erythematosus (SLE).[1] The authors recall the strong association between the presence of antiphospholipid antibodies (aPL) and the occurrence of ischaemic cerebral events in SLE. The results of tests for aPL (lupus anticoagulant or anticardiolipin antibodies), however, are not mentioned in their patients with SLE. Such information could probably be obtained from the patients' charts or from stored serum or plasma. At least the full description of extra-neurological aPL related events—arterial or venous thrombosis, recurrent fetal loss, or thrombocytopenia—could suggest the presence of this peculiar family of antibodies. Such data could allow the hypothesis that intramural deposition of platelet derived material is a feature of a aPL-associated non-inflammatory "vasculopathy" to be tested. The absence of such material in the two patients with SLE and active vasculitis[1] is consistent with this hypothesis, due to the lack of an association between aPL and vasculitis in SLE.[2 3]

Furthermore, the search for intramural deposition of platelet derived material should be performed in other forms of vascular lesions encountered in patients with antiphospholipid syndrome, either "primary" or secondary to SLE, such as non-inflammatory non-atheromatous large artery lesions[2 3] and heart valve thickening,[4 5] the latter being mainly present in patients with SLE, with long-lasting disease.[4] The pathogenesis of these lesions remains unknown: it could involve a complex aPL mediated interaction between platelets and endothelial cells, resulting in platelet derived material incorporation into vessel or heart valve wall, which would explain the "mysterious" thickening frequently observed. Similar remarks could also apply to Sneddon's syndrome, a condition closely related to aPL,[6] the pathological basis of which has been recently detailed,[7] but the pathophysiology remains obscure.

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