arrival of the doctor, and the administration of the thrombolytic agent (either at home or in hospital), and I believe that it might well be possible to show important correlations.

Drs Radford and Richards, and Dr M S Stead,[2] state, as if it was fact, that anistreplase produces an excess of four in 1000 strokes compared with streptokinase. Is it not remarkable that the major study from which this figure derives, for which the analysis is still incomplete, has been announced to the world and yet not published, and that our information is being received at second hand through the medium of a medical journalist, however distinguished and amusing[3]? Without being able to see the original data relating to analysis of the whole of the study it is premature to discuss these findings. I have spoken on the telephone to Dr Rory Collins, the main organiser of the third international study of infarct survival (ISIS-3), and he has made it clear—as also become apparent in Dr Michael O'Donnell's report[3]—that there is no difference in survival at the end of one month between patients who are given streptokinase, tissue plasminogen activator, or anistreplase. As most of the patients who suffered stroke died the disadvantage of the stroke has already been largely discounted four weeks later by a compensatory reduction in mortality from other causes, presumably cardiac causes, among patients given anistreplase. These are curious observations, and they cannot be interpreted without much more information about the patients who suffered the strokes and their particular characteristics.

Dr Stead hopes that nobody will be persuaded to give anistreplase to him should he be unfortunate enough to suffer a myocardial infarction, and his belief that he would be at risk of a four in 1000 increase in stroke is emotionally understandable but needs to be put into perspective. On the basis of the present tentative, interim, and unpublished data, if Dr Stead was administering anistreplase rather than receiving it he might encounter one patient in whom his treatment was associated with a stroke once or possibly twice in 500 years.

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1 Radford J, Richards RG. Domiciliary thrombolytic treatment. *BMJ* 1991;**303**:120. (13 July.)
2 Stead MS. Domiciliary thrombolytic treatment. *BMJ* 1991;**303**: 120. (13 July.)
3 O'Donnell M. Battle of the clotbusters. *BMJ* 1991;**302**:1259-61. (25 May.)

Assessing protective effect of sunscreen products

SIR,—Drs Robin Russell Jones and Ian R White advocate that sunscreens should be labelled with two sun protection factors: one for ultraviolet B and one for ultraviolet A radiation.[1] They suggest that consumers could be told that sun protection factor/B stands for burning and sun protection factor/A stands for aging. We are concerned that this oversimplistic approach has little basis in fact and would confuse the public about the claims of sunscreens regarding protection against exposure to the sun.

We participated in the second international conference on the biological effects of ultraviolet A radiation in San Antonio last June. The papers presented and the ensuing discussion showed that there are conflicting data on the relevant importance of different wavelengths of ultraviolet radiation in causing biochemical, histological, and clinical changes characteristic of photo-aging in animal models. Much more work needs to be done before we can even begin to extrapolate confidently from animal to human skin. To imply that ultraviolet A radiation is the major cause of photo-aging is ill advised at present.

The hope expressed by Drs Russell Jones and White that a method of assessing the protection afforded by sunscreens against ultraviolet A radiation could be agreed at the meeting of the Commission Internationale de l'Eclairage, which met just before the international conference, has not been realised. Again, we attended this meeting, and from the vigorous debate on the merits and otherwise of various in vivo and in vitro methods[2,3] it is clear that further discussions will be needed before a single method can be agreed by manufacturers and governmental regulators.

It should be remembered that when sunscreens are applied for cosmetic purposes the user is not exposed to either ultraviolet B or ultraviolet A radiation but to solar ultraviolet radiation, which always contains both. For this reason the terms ultraviolet B sun protection factor and ultraviolet A sun protection factor have little meaning. As a consequence we suggest that to have a numerical statement specifically of protection against ultraviolet A radiation beside the sun protection factor on product packs would be unnecessary and potentially confusing.

If manufacturers feel compelled to provide information on the broadness of protection against different ultraviolet wavelengths we believe that this can be obtained adequately from the ratio of spectral absorbances in different ultraviolet wavebands measured by in vitro assay. This approach obviates the need for further human studies using clinical end points, some of which are of dubious value in relation to protection from exposure to the sun. The sun protection factor should remain the most important measure of protection against the harmful effects of solar ultraviolet radiation and continue to be determined by phototesting human subjects.

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1 Russell Jones R, White IR. Assessing protective effect of sunscreen products. *BMJ* 1991;**303**:52. (6 July.)
2 Lowe NJ. UVA photoprotection. In: Lowe NJ, Shaath NA, eds. *Sunscreens—development, evaluation and regulatory aspects.* New York: Marcel Dekker, 1990:459-68.
3 Diffey BL, Robson J. A new substrate to measure sunscreen protection factors throughout the ultraviolet spectrum. *Journal of the Society of Cosmetic Chemists* 1989;**40**:127-33.

SIR,—Drs Robin Russell Jones and Ian R White discuss the problems associated with assessing the protective effects of sunscreen products.[1] The availability of sunscreens that are highly protective against ultraviolet B radiation has resulted in sunbathers receiving very high doses of ultraviolet A radiation, leading to many dermatological problems.[2] To determine a product's sun protection factor the accepted "gold standard" method necessitates phototesting of human subjects. This method is not suitable for assessing the protection afforded by a product against ultraviolet A radiation (320-400 nm).

A new, preferably in vitro, method of assessing a sunscreen's effectiveness that does not rely on recording erythema is required. Diffey and Robson have described such a method.[3] In this, the spectral transmission of ultraviolet radiation (290-400 nm) is measured through a sample of Transpore tape with and without sunscreen applied. The tape was chosen as a readily available membrane that transmits both ultraviolet A and ultraviolet B radiation and has an irregular surface on which to apply the sunscreen in a manner similar to that used on human skin in vivo. This test cannot be used for products that are based on alcohol or oil. It has been suggested that human epidermis would be a better choice of substrate on to which to apply the sunscreen.[4] We believe that it is essential to use human skin in any in vitro test and that the skin must be readily obtainable in a non-invasive manner.

We have prepared samples of human stratum corneum taken on to double ground quartz slides by using the skin surface biopsy method.[5] This "sandwich" of quartz, glue, and skin readily transmits ultraviolet radiation throughout the range 290-400 nm; is an ideal substrate on to which to apply a sunscreen for in vitro testing of the protection factor; and can be used for testing any type of suncreen, including alcohol based and oil based products.

To validate our method we calculated the in vitro protection factor of five sunscreens by using the method described by Diffey and Robson[3] but using skin surface biopsy specimens. We also calculated the in vivo sun protection factor of three of the products by a recognised method for humans. The table gives our results.

Sun protection factors of five sunscreens tested in vivo and in vitro

Product	Base	Sun protection factor	
		In vivo	In vitro
A	Cream	7	7
B	Lotion	17	16
C	Cream	4	3
D	Alcohol	4*	4
E	Alcohol	15*	9

*Manufacturer's stated value.

We believe that an in vitro testing method similar to that described by Diffey and Robson[3] has two important uses. The data generated can be used to prescreen potential sunscreens for the degree to which they block ultraviolet B radiation in a manner similar to the "real" situation; the test is much cheaper and quicker than the test used in humans. More importantly, it is the only practical method available to assess the ability of a sunscreen to protect skin from ultraviolet A radiation. We hope that adding human stratum corneum to Diffey and Robson's method may speed the process of persuading the photobiological community to accept and adopt this in vitro method for assessing potential sunscreens for their ability to block ultraviolet A radiation.

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1 Russell Jones R, White IR. Assessing protective effect of sunscreen products. *BMJ* 1991;**303**:52. (6 July.)
2 Hawk JLM. Ultraviolet A radiation: staying within the pale. *BMJ* 1991;**302**:1036-7.
3 Diffey BL, Robson J. A new substrate to measure sunscreen protection factors throughout the ultraviolet spectrum. *Journal of the Society of Cosmetic Chemists* 1989;**40**:127-33.
4 Protecting man from UV exposure [Editorial]. *Lancet* 1991;**337**: 1258-9.
5 Marks R, Dawber RPM. Skin surface biopsy: an improved technique for the examination of the horny layer. *Br J Dermatol* 1971;**84**:117-23.

Normal antenatal management

SIR,—In his article on normal antenatal management Professor Geoffrey Chamberlain stated that venous blood should be checked for ABO and rhesus groups and, if relevant, rhesus antibodies.[1] It should be standard practice to perform an antibody screen in all pregnant women.[2] Professor Chamberlain seems to have completely discounted the fact that, besides anti-D, other antibodies in the rhesus and other systems, including the Kell, Duffy, Kidd, MNS, and P systems, have been implicated in haemolytic disease of the newborn. Hardy and Napier found approximately 0·2% of

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