good as those obtained with any jelly and the method is far less messy and much cleaner. One does not even need to use a towel to dry the patient off afterwards as the surgical spirit evaporates.

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officialdom, on present-day trends there will be a vast number of the population available for study who have submitted themselves to this form of therapy at their own hands.

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¹ McGibbon, D H, and Briffa, D, *Clinical and Experimental Dermatology*, 1978, 3, 371.

Saxophonist's diverticulosis

SIR,—Further to Dr L D Gardner's letter (26 May, p 1428) drawing attention to the extravagant claim for oral pressure made by the saxophonist with diverticulosis (5 May, p 1217), I am at a loss to understand the pathogenic mechanism proposed for saxophonist's diverticulosis, even if the oral pressures generated had been more physiological.

The incomplete Valsalva manoeuvre performed by the blowing saxophonist would be expected to increase intra-abdominal pressure equally on both sides of the colonic wall. I would find more acceptable a description of saxophonist's hernia, piles, varicose veins, or even periphonic petomania.

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PUVA and solariums

SIR,—While the DHSS continue to bombard dermatologists using PUVA therapy for skin diseases with warnings of potential long-term hazards, of which most of us are aware, it might perhaps be more appropriate for them to direct their warnings to the ever-increasing number of the normal population who are attending commercially run solariums or have their own private units and are receiving doses of ultraviolet A in excess of those given by us to our patients.

I recently examined two commercially available solarium units and measured the UVA output with a Waldmann black ray meter, which is used to calibrate our departmental SAS unit. Both "health lamps" had an emission of approximately 15 mW at nine inches, whereas our own unit gives approximately 10 mW at the same distance. Once the customer acquires a tan he or she is encouraged to attend weekly for half-hourly "treatment" to maintain the tan. On my calculations this involves a dose of approximately 30 joules UVA a week.

The DHSS is also right to draw our attention to the possible side effects of the psoralens themselves, but it is also possible to obtain a therapeutic dose of psoralens from the diet. I saw one patient, a tailor's cutter, who developed friction blisters on the first three digits of the right hand where his scissors caused pressure. He had recently bought a UVA health lamp and was also a food faddist in that he ate large quantities of celery, parsnip, and fresh citrus fruits, all of which contain psoralen. Friction blisters in patients undergoing PUVA therapy have been described.¹ This patient's blisters subsided when he abandoned his UVA lamp.

It is probable that the only way of determining the long-term effects of PUVA therapy will be to administer it to a large number of patients carefully followed over many years, but, should our attempts to give PUVA to a select group be thwarted by

Amitriptyline plasma concentrations and clinical response

SIR,—We agree with Dr T R Norman and others in their letter (31 March, p 894) that investigations of the relation between plasma antidepressant concentrations and clinical response should be conducted in those groups of patients who are most likely to benefit from tricyclic antidepressant therapy. With this in mind, we included in our study (27 January, p 230) only hospital patients with a diagnosis of endogenous depression, as defined by recognised scales, who did not respond during a placebo treatment period. Unfortunately, the concise presentation of our report has led Dr Norman to misunderstand some of our results.

The mean response rate, measured by a decrease on the Hamilton rating scale (HRS), of patients who developed plasma concentrations inside our proposed therapeutic range of 80-200 µg/l amitriptyline plus nortriptyline was not 58%, as stated by Dr Norman, but a satisfactory 72% improvement. In contrast, the mean response rate of those patients with plasma concentrations outside this range was only 42%. The difference in response between these two groups was highly significant irrespective of whether final HRS score, amelioration score, or percentage change in HRS score was used ($P < 0.001$). Furthermore, non-parametric statistical analysis produced similar results.

Reanalysing our data along the lines suggested by Dr Norman, we have compared the clinical response of those patients with plasma concentrations inside and outside the suggested therapeutic range. The criterion for clinical response was set, as in our earlier study,¹ at a final HRS score of 8 or less. Twenty-nine of the 38 patients with plasma concentrations within the 80-200 µg/l range responded (76%), compared with only 10 out of 27 patients (37%) with plasma concentrations outside this range ($\chi^2 = 10.2$, $P < 0.005$). Measurement of drug plasma concentrations was carried out by gas chromatography,² which has been shown by external quality control tests to be extremely reliable, with a variation index of only 0.25.³ The figure shows the mean percentage change on the HRS with increasing plasma concentrations of amitriptyline plus nortriptyline for the 65 patients completing our study. The seven patients whose plasma concentrations were below 80 µg/l had a poor mean response (38%). There was no apparent difference in response for the three groups of patients with plasma concentrations of 80-120, 120-160, and 160-200 µg/l; while the 20 patients with plasma concentrations in excess of 200 µg/l showed a successively poorer response with increasing plasma concentrations.

Where previous studies have included only small numbers of patients there is an increased chance that the plasma drug concentrations

Mean percentage change in HRS ($\pm$ SE of mean) according to plasma concentrations of amitriptyline plus nortriptyline.

observed will form a cluster. Such an occurrence at low concentrations may well produce a positive linear correlation between plasma drug concentration and clinical response,^{4,5} while a preponderance of high concentrations could produce a seemingly negative one.^{1,6} This phenomenon may produce results from different trials which appear to be contradictory but are in fact complementary. When a large, well-conducted study such as ours is completed these apparent contradictions are largely reconciled, and the true curvilinear relation between plasma concentrations of amitriptyline and clinical response is revealed.

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¹ Montgomery, S A, et al, *Clinical Pharmacology and Therapeutics*, 1978, 23, 309.

² Dawling, S, and Braithwaite, R A, *Journal of Chromatography*, 1978, 146, 449.

³ Jones, S, and Turner, P, *British Medical Journal*, 1979, 2, 1217.

⁴ Braithwaite, R A, et al, *Lancet*, 1972, 1, 1297.

⁵ Ziegler, V E, et al, *Archives of General Psychiatry*, 1977, 34, 607.

⁶ Kragh-Sorensen, P, et al, *Lancet*, 1973, 1, 113.

Historical fats

SIR,—Your journal (2 June, p iv) carried an advertisement from the Butter Information Council which could be misleading. The heading of the advertisement states, "If you were convinced that the population is not eating any more animal fat than it was in 1909—would it make you stop and think?" If we accept the truth of the statement, it is still misleading, because the quality of animal fat in 1909 was almost certainly different from the quality of animal fat at the present time. The animal feeding was completely different—for example, the soft fat pig was considered of good quality, whereas today the bacon factories want only hard fat pigs. Also the dripping sold in 1909 contained unsaturated fatty acids as opposed to the present hard cooking fats and lards, which contain only saturated fats.

The point I am making is that within the total amount of animal fat consumed in 1909