group as a whole ($P < 0.025$). The antipyrine half life of the 20 non-deficient people was 11.3 ± 2.1 h, which was significantly shorter than in either the group as a whole ($P < 0.05$) or the 16 people with low vitamin levels ($P < 0.001$).

We conclude that poor nutrition in the elderly is at least in part responsible for the decline in microsomal enzyme function with age.

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national interests at heart and by those responsible for the public health.

If the Royal College of Physicians and other medical authorities take up the challenge as they have done effectively in regard to smoking much can be accomplished to control the incidence of alcoholism and to instruct the public in the facts of this increasing major national health problem.

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¹ O'Malley, K, *et al*, *British Medical Journal*, 1971, **3**, 607.

² Vestal, R E, *et al*, *Clinical Pharmacology and Therapeutics*, 1975, **18**, 425.

³ Briant, R H, *et al*, *New Zealand Medical Journal*, 1975, **82**, 136.

⁴ Griffiths, L L, *et al*, *Gerontologia Clinica*, 1967, **9**, 1.

⁵ Smithard, D J, and Langman, M J S, *British Journal of Clinical Pharmacology*. In press.

⁶ McLennan, W J, *et al*, *Quarterly Journal of Medicine*, 1973, **42**, 1.

⁷ Baylis, E M, *et al*, *Lancet*, 1971, **1**, 62.

⁸ Colby, H D, *et al*, *Biochemical Pharmacology*, 1975, **24**, 1644.

The cowardice continues

SIR,—Under the above title (4 June, p 1428) you berate the Government for its lack of effective action against the lethal effects of smoking. I am in complete agreement with the tenor of your article. However, a further reaction was to ask myself when we as a profession are going to overcome our cowardice and take up the cudgels against alcoholism as we have done quite effectively against smoking.

Having worked among alcoholics in New Zealand in the past and been president of the National Society on Alcoholism for seven years I have had the opportunity to study the many facets of the problem as it affects the individual and his family, the national economy, the cost to medical services, and the effect on crime and accident rates. Competent surveys initiated by the society and the public health authorities give some impression of the extent of the problem in New Zealand—a small country with approximately 3 million inhabitants. In a Sir Charles Burns lecture Dr Fraser McDonald, a leading psychiatrist, gave a small sample of the statistics as follows. (1) Thirty per cent of all male admissions to both general hospital, psychiatric units, and mental hospitals are due to alcoholism. (2) It is estimated that 30% of acute surgical beds in general hospital are occupied by patients in whom a major contributory factor is alcohol. A study in Christchurch Hospital showed that 70% of road accident victims would not have been able to pass the breath test. (3) One out of 15 adult social drinkers will become an alcoholic. (4) It is estimated that there are 35 000 alcoholics in New Zealand and some 200 000 people will have their lives severely disrupted by alcoholism.

These statistics indicate some distressing personal aspects of the problem but do not touch on the many medical aspects of the problem, the part alcohol plays in the increasing crime rate, and the impact on the economy of the country in lost man hours and loss of efficiency. Clearly alcoholism is a great national health problem and the adverse effects of social drinking and of alcoholism on such a large section of the community cannot be continually ignored by those of us who have the

Prolactin in anorexia nervosa

SIR,—Dr A D B Harrower and his colleagues (16 July, p 156) report finding raised serum prolactin concentrations in anorexia nervosa. Our own results, to be published in detail shortly, are in marked contrast. We examined basal serum prolactin concentrations and the response to thyrotrophin-releasing hormone (TRH) in 11 female patients with anorexia nervosa, both in the acute stage and after supervised weight gain in hospital. No psychotropic medication was given.

The basal prolactin level (mean $\pm$ S.D.) before weight gain was 4.8 ± 2.5 μ g/l, and after weight gain 6.6 ± 3.1 μ g/l. These results do not differ significantly from each other nor from the mean level in a group of 23 healthy female controls (6.3 ± 2.1 μ g/l). The ranges of prolactin concentrations were: before weight gain 2–11 μ g/l; after weight gain 3–13 μ g/l; healthy controls 2–12 μ g/l. There was thus no evidence of hyperprolactinaemia in the anorexic patients—if anything the results were marginally lower before weight gain.

When an intravenous injection of 200 μ g of TRH was given before weight gain the mean prolactin level rose to 20.5 ± 7.7 μ g/l after 20 min and was 16.2 ± 5.4 μ g/l after 60 min. After weight gain the corresponding values were 35.5 ± 11.0 and 21.5 ± 6.7 μ g/l. The difference between the 20-min levels is highly significant ($P = 0.001$), while there is no significant difference between the 60-min levels. These results are consistent with the delay in peak prolactin secretion after TRH in anorexia nervosa shown by Vigersky *et al*¹ and the recovery in pituitary prolactin reserve after weight gain is similar to that demonstrated for gonadotrophins.²

On examination of Dr Harrower and his colleagues' data it is clear that the apparent hyperprolactinaemia of their group with anorexia nervosa is really due to the effect of the one subject with a considerably elevated serum prolactin concentration of 90 μ g/l, which requires investigation in its own right and rigorous exclusion of the possibility of drug administration. If this subject is excluded the mean serum prolactin concentration falls to 16.6 μ g/l, not significantly different from that of their obese group. Similarly, the report of Travaglini *et al*³ of hyperprolactinaemia in five out of six anorexic patients is unsatisfactory in that therapy with dopamine antagonists was not excluded.

We would therefore conclude that there is no unequivocal evidence of hyperprolactinaemia in anorexia nervosa. This is important to resolve as it may help to elucidate the central mechanisms involved. Both depletion of brain dopamine⁴ and increased activity of dopamine at central receptors⁵ have been postulated as mechanisms for the development

of anorexia nervosa. The evidence from prolactin studies is against the dopamine depletion hypothesis. Most reports suggest normal basal prolactin levels in anorexia nervosa, but our finding of impaired pituitary prolactin reserve and the study of Kalucy *et al*⁶ showing decreased nocturnal prolactin secretion are more consistent with the second hypothesis.

¹ Vigersky, R A, *et al*, *Clinics in Endocrinology and Metabolism*, 1976, **5**, 517.

² Warren, M P, *et al*, *Journal of Clinical Endocrinology and Metabolism*, 1974, **40**, 601.

³ Travaglini, P, *et al*, *Acta Endocrinologica*, 1976, **81**, 252.

⁴ Mawson, A R, *Psychological Medicine*, 1974, **4**, 289.

⁵ Barry, V C, and Klawans, H L, *Journal of Neural Transmission*, 1976, **38**, 107.

⁶ Kalucy, R S, *et al*, *Journal of Psychosomatic Research*, 1976, **20**, 595.

Starting on the pill

SIR,—Women starting on oral contraceptives are advised to take the first pill on the fifth day of their menstrual period and told that additional contraceptive precautions are essential for the first 14 days.

I suggest that the time has come to question whether this is the most sensible regimen to adopt. Three years ago I took part in a clinical trial of a pill containing 150 μ g of D-norgestrel combined with 30 μ g of ethinyl oestradiol (Ovranette). Patients were instructed to start taking the pill on the first day of the next menstrual period and told that the contraceptive effect was immediate, so that no additional contraception was necessary. No pregnancies occurred in the first cycle among 178 patients for whom the pill was prescribed in this way. Since then it has been my practice to advise patients starting oral contraception to follow this regimen and not one has become pregnant. The first cycle on the pill is shorter (22–23 days), but provided patients are warned about this they do not mind. The incidence of spotting and breakthrough bleeding is not increased. Subsequent packets of pills are started as usual after the standard number of pill-free days and normal cycle length is re-established.

It is also routine practice to advise patients to use additional contraceptive precautions for the first 14 days when changing to a new pill, particularly to one of a lower dose. This advice was reiterated in a recent circular from one of the drug firms, Schering. However, we find that many of our patients object so strongly to this that they refuse to change over to a pill containing 30 μ g of oestrogen, even when we explain to them that such pills are now to be preferred. I believe that the need for additional contraceptive precautions under these circumstances could safely be avoided if patients were advised to start their new pill on the first day of withdrawal bleeding or even proceed to take it on the day after finishing their present preparation—that is, to take two packets of pills consecutively without a break. I have used both these regimens successfully and find that they prove effective and acceptable to the majority of women.

At present patients attending family planning clinics who require additional contra-