enable easy communication with the children's parents. The parents, on receipt of the information, could then arrange for any necessary treatment with the family dentist. Lawson et al. in 1978 reported on the information obtained from these screening examinations during the period 1961–1974. They clearly stated: "These surveys do not represent a random selection from all children within this community, and *the summary data cannot be used for detailed studies*" (my emphasis).

Although it is not readily obvious from the above letter, the correspondent's sole explanation for the decline in dental caries is the increased use of the toothbrush by the young. This is spelt out in Sutton's reference 5, which is not a scientific paper but a letter to the *New Zealand Medical Journal*. Our 1984 paper showed such a hypothesis to be baseless: here, studies were cited which showed that, amongst the young, brushing teeth without a fluoride toothpaste had little or no effect on dental caries. It was pointed out that this was probably due to the inability of a toothbrush to remove plaque completely from the prime sites of caries development.

Finally, readers wishing to examine summaries of the voluminous literature attesting to the decay-reducing properties of fluoride would be well advised to consult standard texts such as those listed below.[1,2]

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1. Stewart RE, Barber TK, Troutman KC, Wei SHY, eds. Pediatric dentistry. St Louis: Mosby, 1982 (Chapter 45).
2. Stallard RE, ed. A textbook of preventive dentistry, 2nd edn. Philadelphia: W.B. Saunders, 1982 (Chapters 9 and 10).

The economics of general practice

To the Editor: *The Medical Journal of Australia* is not an economics journal; this is probably why you allowed Dr Deeble's commentary on the economics of general practice[1] to be published in your journal, containing as it does an elementary error in economics.

The valuable contribution made by Dickinson et al.[2] was to point out that, on reasonable assumptions, given the current fee structure, it is impossible for a general practitioner to earn what the community would regard as an adequate income by working the number of hours which have become the community standard. In attacking this proposition, Dr Deeble asks: "Have tax law distortions so permeated our thinking that 'real' costs are unidentifiable?", implying that "real economic costs" are not measured by the lease value of practice equipment.

Provided it is the subject of an "arms-length" transaction, the lease value of equipment is a good approximation — indeed, the closest possible approximation — to the "real economic costs" of owning practice equipment. If a doctor has purchased equipment, cars or premises, and has not borrowed the money to do so, his taxable income *from the practice of medicine* would be higher than if he leases them from a finance company, but the "real economic costs" of owning the equipment would be unchanged. That is, they would be depreciation and the forgone earnings on the capital used to purchase the equipment. More generally, as economists, we state without fear of contradiction that the "real economic costs" (Dr Deeble's words) of using an asset for a particular purpose are independent of who owns the asset (although maintenance costs may vary according to the user).

Of course, where leasing arrangements are not at arms length, "tax figures" may be a poor indicator of costs. A careful reading of Dr Deeble's comments, however, leads us to believe that it is the *practice of leasing itself* and the additional costs that he imagines arise from this to which he takes exception.

It is important not to confuse income earned solely from practising as a doctor with income earned from owning equipment. Doctors, like everyone else, have a right, when they invest their savings, to seek the highest rate of return. Dr Deeble, of all people, should not complain if that is not in doctors' own practices, or if doctors are not rich enough to purchase all their equipment outright.

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1. Deeble JS. Money, medicine and management. The economics of general practice. *Med J Aust* 1984; 140: 638-639.
2. Dickinson JA, Hall J, Logan J, McDonald ML. An economic model of general practice. *Med J Aust* 1984; 140: 652-658.

To the Editor: Congratulations to James Dickinson et al. for their excellent paper. It is the clearest exposition I have seen of the basic economics of general practice. In particular, it makes indelibly clear that the better medicine you practise, the more time you spend with your patients, the less will be the reward (financially) — and the poorer the receptionist service and the scruffier the premises, equipment, etc, the greater the financial return.

This paper also highlights the ridiculous situation caused by crazy government regulations for GP consultations. The Medicare benefits fee schedule reads:

short consultation (less than 5 minutes) — $10.80 (NSW);

standard consultation (anything from *5* to *25* minutes) — $15.

It does not take much mathematics to show that a doctor who sees a patient every 7½ minutes sees 8 per hour and receives $120 at full rate; a doctor spending 10 minutes per patient sees 6 and receives $90 an hour; one with a 12-minute schedule sees 5 for $75; and those with 15 minutes (often with appointments) collect $60.

As the authors show, the overhead is probably about $25–$30 per doctor. The net GP earning is anything from about $90 an hour for the poorest service to about $35 an hour for the best.

After about 40 years' experience in practice, practice management and, lately, visiting GPs throughout New South Wales, I concur fully with the paper and its authors' findings. A better plan to discourage excellence is hard to imagine!

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Orthomolecular medicine

To the Editor: In view of the letter by Dr Chris Reading (MJA, June 9, 1984), it needs to be emphasized that just simply calling somebody odd is not a scientific way of proving them wrong. To do this you have to examine carefully what they say and do. Nor is it enough to rely on hearsay, which in this context is the equivalent of an anecdotal story.

In his editorial of May 12, Professor Mark Wahlqvist writes: "A particularly disturbing error in thinking has been the view — even among medical graduates who ought to know the principles of physiology and pharmacology — that if a little of something is good, more of it is better. This view has become part of the dogma known as 'orthomolecular medicine' ".[1] The term "orthomolecular medicine" was originated by Linus Pauling. Linus Pauling has won the Nobel Prize twice. One does not get such accolades simply for dogma. As I understand the term, it means an attitude of mind which says that if the parameters of physiology, biochemistry and metabolism are kept normal then one is more likely to have good health. Put another way, "orthomolecular medicine" is simply a statement that all the mechanisms of homeostasis need nurturing if health is to be maintained. We, as a profession, have for a long time refused to admit that nutrition has anything to do with homeostasis. Current research work is proving this to be a very narrow view. Thankfully, directors of intensive care units have cottoned on to this fact. Lives are being saved because of it.

Because it was written in an editorial, because Professor Wahlqvist is an eminent nutritionist, and because both these call for a truly scientific attitude, may I be so bold as to ask Professor Wahlqvist to define in scientific terms what he thinks of as "orthomolecular medicine"?

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1. Wahlqvist ML. Nutrient supplementation in Australia. *Med J Aust* 1984; 141: 573-574.

In reply. — Dr Alan Lane asks me to define "orthomolecular medicine". As I am not one of its protagonists, I do not plan to define it but I can comment on its history and the

way in which, by this or any other name, it is being applied in patient care.

The term "orthomolecular psychiatry" was first used by Linus Pauling in 1966, and broadened to "orthomolecular somatic medicine" in 1968. He took the view that a number of metabolic functions of vitamins required doses much larger than the Recommended Dietary Allowances (RDAs). The facts are that, although metabolic blocks are very occasionally seen in inherited disorders of metabolism, they are not common; they are even less common in multiple forms. Moreover, no matter how novel and interesting Pauling's suggestions might have been, they have not been supported by other workers in peer-reviewed journals.

It does not help that Linus Pauling has received two Nobel Prizes (one in chemistry and the other in peace) — nutrition was not one of Pauling's fields of expertise. We must be wary of experts pronouncing outside their field of training, while not denying the value of bright ideas. But bright ideas are not enough, and should not form the basis of medical practice without due evaluation.

In practice, "orthomolecular medicine" has come to be the label for the field of megavitamin therapy for a host of different illnesses in which evidence of effectiveness is lacking. It is an approach based on belief and dogma, and therefore alien to the principles of Western scientific medicine, where it is necessary to respond to changing evidence. I would go so far as to say that "orthomolecular medicine" is unethical. The practice of the dogma will not be helped by what I now perceive to be an effort of its practitioners to alter the description to more orthodox designations, such as "nutritional medicine".

The sooner scientifically based nutrition appears in Australian medical school curricula and postgraduate nutritional education, the better. Our profession will then have filled the vacuum at present occupied by pseudoscience and, through peer review, ensure acceptable standards for the application of nutritional knowledge in clinical practice.

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Chronic subacute hypoglycaemia

To the Editor: There is a condition that can occur in treated diabetes which, to my knowledge, has never been reported as a specific entity. Many physicians must be aware of it, but a lack of reference to it in standard texts results in a failing in the inexperienced to recognize it when it occurs. The condition I refer to could best be termed chronic subacute hypoglycaemia, and the following case history serves as an illustration.

A 70-year-old man was admitted to hospital for hip replacement. He was known to be a diabetic, and prior to admission was not taking insulin. However, around the time of surgery, insulin therapy was begun. The surgical procedure was completed satisfactorily and anaesthesia was uncomplicated. Following the operation he did not regain his normal mental health. He was mildly confused, mildly disorientated, and withdrawn. He was seen by a psychiatrist, who diagnosed a depression related to his wife's death 12 months earlier. An antidepressant drug was prescribed. There was no improvement, and he was transferred to a psychiatric unit for further treatment. There he was found to be unaware of the day or date and unable to orientate himself. He was receiving 16 units of insulin (Actrapid MC) in the morning and 8 units in the evening. Capillary blood glucose measurements ranged between 1.8 and 6.5 mmol/L, with most readings between 3 and 4.5 mmol/L. The antidepressant was discontinued, with no change in his mental state. The insulin injections were then ceased, and after a few days his mental state had returned to complete normality. It was clear that he was not depressed, and he involved himself in a rehabilitation programme for his new hip with gusto.

Presumably the lack of obvious classical hypoglycaemic symptoms was considered to exclude hypoglycaemia as the cause of his mental state. Yet there is no doubt that a persistent low blood sugar can cause altered mentation of a persistent, steady type, without the classical symptoms of hypoglycaemia. As illustrated in the case described, the mental state returns to normal rapidly when blood sugars are allowed to rise.

This syndrome is not limited to patients taking insulin. It is, in fact, more common in elderly patients on sulphonylurea drugs. It is particularly likely to occur in institutions such as old peoples' homes, where staff inexperienced in diabetes are supervising therapy. As too often with other diseases, symptoms in the elderly are easily attributed to age.

Chronic subacute hypoglycaemia should always be considered as one of the reversible causes of organic confusion, personality change, or overt dementia in the elderly diabetic. There may be no other clues to its existence, although sometimes unaccountable sweating or excessive hunger will occur.

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We asked Professor Larkins, of the Department of Medicine at the University of Melbourne, to respond to this letter; his comment follows.

Dr Watson has drawn attention to an important and commonly unrecognized manifestation of hypoglycaemia in the elderly. Confusion without the classical autonomic accompaniments of hypoglycaemia may be the major manifestation of hypoglycaemia in this age group. Slow onset of relatively mild hypoglycaemia, together with some degree of autonomic neuropathy which is common in elderly diabetics, presumably accounts for the lack of classical autonomic features. Prolonged impairment of neurological function can follow hypoglycaemia of short duration in the elderly, and the clinical situation that Dr Watson describes is more common than is usually recognized. Increased emphasis on achieving euglycaemia in diabetic patients must be tempered by an awareness of the risk of hypoglycaemia, with its protean manifestations. This caution applies particularly in the elderly. Other neurological manifestations which may not be recognized as being attributable to hypoglycaemia are aggressive and irrational behaviour. Dr Watson is also correct to draw attention to the fact that the risk of hypoglycaemia, including its neurological manifestations, is not confined to patients receiving insulin. Oral hypoglycaemic agents, particularly glibenclamide and chlorpropamide, may also cause significant hypoglycaemia.

The situation Dr Watson describes should not be confused with the commonly made, but usually spurious, diagnosis of hypoglycaemia in non-diabetic subjects. It has become common practice in some circles to attribute a variety of non-specific symptoms, such as tiredness, lack of energy, loss of libido, light-headedness, palpitations and anxiety, to reactive hypoglycaemia. Reactive hypoglycaemia is a rare disorder, best diagnosed by measuring plasma glucose at the time of symptoms. A prolonged glucose tolerance test is not an appropriate investigation in this situation, as many normal people become significantly hypoglycaemic during such a test.[1] Failure to recognize this fact has led to many instances of symptoms being falsely ascribed to hypoglycaemia.[2]

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1. Charles MA, Hofeldt F, Shackelford A, et al. Comparison of oral glucose tolerance tests and mixed meals in patients with apparent idiopathic postabsorptive hypoglycemia. Absence of hypoglycemia after meals. *Diabetes* 1981; 30: 465-470.
2. Yager J, Young RT. Non-hypoglycemia is an epidemic condition. *N Engl J Med* 1974; 291: 907-908.

Medifraud and overservicing

To the Editor: Dr George Repin's reply to Dr Walton's letter in the July 21 issue of the Journal prods me into print — it so eloquently typifies the AMA's attitude and its shortcomings. The Federal Assembly "concluded" and "formally resolved", did it? I suppose we should all be grateful and satisfied — indeed the Secretary General suggests that the correspondent should be "both interested and pleased to know" — that the Federal Assembly so decided; unfortunately nobody else knows.