driven by their evangelical belief that the ills of renal bone disease are best treated by calcium and the avoidance of derivatives of vitamin D.² It is unfortunate that their conclusions are based on their experience—a notorious arbiter of efficacy—rather than on double blind and randomised controlled studies, but they raise some important points.

The transient suppression of hyperparathyroidism with alfacalcidol and the subsequent escape are well described, not only with derivatives of vitamin D but also with cholecalciferol and with calcium whether given in the form of supplements to the diet or supplements to the dialysis fluid.³ As mentioned, the regimen that we used did not adversely affect serum phosphate concentration. This suggests that factors other than hyperphosphataemia, vitamin D metabolites, and extracellular calcium concentrations are important components of the pathogenesis of hyperparathyroidism. Higher doses of alfacalcidol than we gave may possibly have more complete or long lasting effects. The view that alternative treatment regimens would provide more complete effects is a hypothesis that is not yet proved.

Fournier and colleagues also raise the question of whether our patients had a dietary deficiency of vitamin D. Bone histology is the ultimate arbiter of osteomalacia, which was rarely found on biopsy before treatment: only one patient had osteomalacia in the absence of osteitis fibrosa on histological examination of bone. Dietary intakes of calcium and vitamin D (which were measured but not given in our paper) were well above the recommended daily allowance, and the average dietary intake of calcium exceeded 800 mg daily. If data from all patients with prevalent osteomalacia are excluded the same results were observed.

In response to other questions, reference ranges for bone cell counts vary according to the laboratory. Our own are provided elsewhere.⁴ The optimum range of parathyroid hormone concentrations in early renal failure is provided by the reference ranges. We speculate that the same range will operate, irrespective of the degree of renal failure.

JOHN A KANIS
Professor

NAVEEN A T HAMDY
Clinical research fellow

WHO Collaborative Centre for Metabolic Bone Diseases,
Department of Human Metabolism and Clinical Biochemistry,
University of Sheffield Medical School,
Sheffield S10 2RX

IAN T FAIREY
Clinical data manager

Leo Laboratories,
Princes Risborough HP27 9RR

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Psychosis due to "skunk"

EDITOR,—Recently we have become aware that drug misusers are using a variety of cannabis known as skunk. Some users have described a considerable psychotic experience after use of relatively small quantities.

In November 1994 we surveyed 50 drug misusers attending the methadone programme of the

Glasgow drug problem service, asking them about their reported use and experiences of skunk. Eight reported having used skunk, of whom four reported having experienced psychotic symptoms after its use. Three of those who described psychotic experiences agreed to further assessment. All three admitted to regular misuse of cannabis in addition to their prescribed methadone (with one admitting to occasional misuse of benzodiazepines) and so were experienced in the effects of cannabis. All denied concomitant use of other hallucinogens, and none described any important psychiatric history.

All three described psychotic experiences after smoking one "regular sized joint." These consisted of paranoid delusions and visual illusions in all three cases, visual and auditory hallucinations in two cases, and pronounced derealisation and depersonalisation together with thought broadcast in one case. Two users described associated severe anxiety. The experience lasted for a variable period (half an hour, one day, and three days).

All three subjects believed that the skunk that resulted in the psychotic experience originated from the Netherlands rather than Britain.

Skunk is derived from a variety of cannabis plants that have been selectively cross bred to produce high yielding varieties. While the tetrahydrocannabinol content of "normal" cannabis is up to about 5% by weight, skunk may yield 10-15%, with an even higher content being reported from the Netherlands.

Doctors should be aware of the increasing availability of varieties of cannabis yielding a high content of tetrahydrocannabinol and that psychotic episodes may be precipitated by consumption of what would previously have been considered to be relatively small quantities.

A S WYLIE
Senior registrar in general psychiatry
and substance misuse

Gartnavel General Hospital,
Glasgow G12 0YN

R T A SCOTT
Medical director
S J BURNETT
Research officer

Glasgow Drug Problem Service,
Ruchill Hospital,
Glasgow G20 9NB

Rationing is not inevitable

EDITOR,—In the debate about rationing, rationing is often assumed to be inevitable.¹ But is it? As Rawles has pointed out: "The ethical problem of how to apportion limited resources amongst the needy has been forced on us by arbitrary limitation of health care expenditure. Its solution would not be required if health expenditure were higher."²

Those who support rationing often say that resources are finite but demands are infinite. Yet this cliché does not bear examination. It is a version of Malthus's discredited argument that populations would always outrun resources. It would be closer to the truth to argue that resources are infinite while demands are finite. Any sensible definition of the resources involved in health care, as in other human activities, must include the skills, education, and professionalism of those working in the sector. There is no limit to these.

Nor are demands infinite. Firstly, any human life is finite, so demands can be only finite. Secondly, there is not an infinite demand for any particular treatment—for instance, there is not an infinite demand for kidney transplants but a need for about 60 per million population. Even if we all needed a kidney transplant this would not equal infinite demand.

Government ministers all too often tell us that we must cut our coat according to our cloth. Yet they are figuratively closing down the textile industry that produces that cloth. Our NHS is

built on the effort to match resources to needs. It is not our job to cut needs down to match limited resources. Rationing, as Callahan rightly said, is "the deliberate withholding of beneficial medical care."³ It is contrary to the NHS's purpose.

WILL PODMORE
Chief librarian

British School of Osteopathy
London E8 3EE

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Education and prevalence of Alzheimer's disease and vascular dementia

Premorbid ability influences measures used to identify dementia

EDITOR,—Alewijn Ott and colleagues identified 474 cases of dementia in a suburb of Rotterdam and report that the prevalence of Alzheimer's disease is inversely related to educational status.¹ In an accompanying editorial Martin Orrell and Barbara Sahakian conclude that there may be some truth in the age old saying "use it or lose it" regarding mental skills.² In Ott and colleagues' study dementia was primarily diagnosed on the basis of poor performance in tests of cognitive efficiency. The first screen consisted of a geriatric mental status examination and the mini mental states examination. Subjects who were positive on screening were then assessed with the Cambridge examination for mental disorders of the elderly diagnostic interview; those scoring below the recommended cut off on the cognitive subscale were selected for brain scanning and neurological evaluation. In essence, a major determinant of the identification of cases and diagnosis was poor performance in the minimal state examination and on the cognitive subscale of the diagnostic interview. The authors then report that a greater proportion of subjects with low educational attainment fulfilled diagnostic criteria for Alzheimer's disease. They do not think that diagnostic bias influenced their findings.

We wish to add a cautionary note before the relation between education and Alzheimer's prevalence becomes an established fact. Educational attainment correlates to a greater or lesser extent with virtually every cognitive test through the shared variance of general ability and intelligence. To put it simply, some people may perform relatively poorly in tests such as the cognitive subscale of the diagnostic interview not because they have an insidious dementing condition but because they were never particularly intelligent to begin with. Such people often have a limited educational background.

We put this hypothesis to the test in 51 healthy elderly subjects who were cognitively intact (mean age 74.1 (SD 18.2) years; mean total score on the cognitive subscale 95.9 (5.3)). Following Ott and colleagues' method, we classified our group by degree of educational attainment, in our case years of full time education (≤ 10 years, 11-12 years, ≥ 13 years). Analysis of variance of the total scores on the cognitive subscale showed a highly significant effect (≤ 10 years ($n=19$), score=92.2 (6.2); 11-12 years ($n=15$), score=97.4 (4.8); ≥ 13 years ($n=18$), score=98.7 (3.2); $f=8.76$, $P=0.0006$). Scheffé posthoc comparison showed that the group with fewest years of full time education performed significantly worse in terms of the total score than the two other groups. The simple correlation between years of full time education and the total score for the entire healthy, non-cognitively impaired sample was $r=0.48$, $P<0.001$.