Exit examination

I was interested to read Professor Greaves's letter (January 1991), in which he advocates a dermatology examination for specialist accreditation, as well as the MRCP. As a recent emigrant to Australia from the UK, I would fully support this.

The Australian registrars have to sit an exit examination after four years of training in dermatology, and as a result they generally know more dermatology than their British counterparts. In view of the fact that they do not undergo any formal training in general medicine or have to sit an exam in this subject, the Australian trainee dermatologists do not know as much medicine. I believe that the British system would become ideal if candidates sat the MRCP after three years general medicine as at present, and had an exit exam after four years dermatology training.

The main criticism that may be posed for such an examination is the fact that there would be less time for registrars to perform research. I cannot see that this should be a major problem, because with the present competition for registrar and senior registrar posts in the UK, those who perform research and write papers, and also work for the exit examination successfully, will be those who get the consultant posts.

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Clinicians and information technology

Sir—As clinicians in a resource management initiative site grappling with the problems of introducing case mix management system, we would like to comment on Dr Severs' article (October 1990). We do not agree that it is a clinicians' job to assign the code. The clinicians' job is to assign the diagnoses, co-morbidities and procedures in each patient episode and to validate these. NHS in Wales published an All Wales information and technology strategic direction which produced a report on the All Wales medical record assessment. We found this report helpful in planning our own local strategy. The top priority was to have a believable clinical data base in which the diagnoses and procedures were to be validated by the consultant concerned.

Deficiencies in our establishment of coders were revealed; steps have been taken to recruit more coders and to ensure that they are properly trained. Training of our coders was enhanced by the development of a medical terminology course at the North East Wales Institute of Health Studies, in which the coders from the three acute units in North Wales participated and found extremely helpful. The number of coders recommended in the above report was one WTE for 6000 annual discharges where coders were working from the entire case record and completing the Körner data information. Resource management project has provided computerised assistance with coding in the form of the 3M computer package, which has allowed a much more detailed extraction of information from the case record. With regards to coding accuracy, the Welsh Office medical records consultant who reviewed 60 case records found that the selection of the proper code for the diagnosis, given the documentation available, was appropriate in 98% of cases. It is proposed that a permanent record of diagnoses and their codes, assigned to that episode are to be posted in the case records for the purpose of audit.

Other problems have been highlighted. The medical record and the information contained in it is at the heart of any case mix management system. Years of neglect in this vital area need to be redressed. We would like to put in a plea for OPCS 4 to be replaced by the ICBM 9CM to give a much more detailed, relevant procedure code. However, none of these systems will deliver a data base of the type which many clinicians want. Our policy is to extract as much clinical information as possible from the patient administration, ward order entry and coding systems into case mix management. Where clinicians identify specific deficiencies, data fields may be derived to include information required for the purposes of research, audit etc.

The resource management initiative is supported by a potential benefits and benefits realisation study and will end with a full project evaluation programme. Information technology is expensive and investment must be justified by utilisation in terms of patient benefits.

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Intrauterine growth retardation does not cause cardiovascular disease

Sir—Professor D. Barker has proposed that retardation of growth during fetal life predisposes to cardiovascular disease in middle age (April, p129). However, evidence from two sources casts doubt on this hypothesis.

First, retardation of fetal growth occurs in twins. In a North American study of 2,449 live born twins, the 50th percentile for mean birth weight of those born at 40 weeks was about 2.7 kg [1]. Despite this, cardiovascular disease is no more prevalent in twins than in singletons.

Second, there are no reports of increased cardiovascular disease in adults who experienced famine prenatally or during infancy. In the Dutch famine of 1944/45 the average daily distribution of food rations in the western Netherlands in the first quarter of 1945

fell to 700 calories. This was followed by a fall of about 0.5 kg in mean birth weight [2].

Nevertheless the adult survivors of this intrauterine exposure to famine appear to be largely indistinguishable from control cohorts [2] whereas significant differences in blood pressure related to birth weight in well nourished communities have been identified in children as young as five years [3].

Although twins do not have a higher risk of cardiovascular disease than singletons, the concordance rate for coronary heart disease and hypertension is higher in monozygotic (MZ) twin pairs than dizygotic (DZ) twin pairs. In 705 twin pairs in which at least one member had reported hypertension, there was a concordance rate of 0.34 in MZ and 0.09 in DZ pairs [4]. In 1,028 middle aged male twins the concordance rate for dyslipidemic hypertension was 0.44 in MZ pairs and 0.14 in DZ pairs [5].

Genetic factors are also believed to explain the higher rate of coronary heart disease in immigrants from Asia who are now living in the United Kingdom [6].

There are features of Professor Barker's data which also suggest that the relationship between weight in infancy and risk of cardiovascular disease is a genetic phenomenon. First, the correlation between ischaemic heart disease and weight was stronger at the age of 12 months than at birth [7]. The correlation between genetic factors and weight is also stronger at 12 months than at birth [8,9]. Second, the relationship was only found in breast fed infants and not in bottle fed infants [7]. Bottle fed infants are more vulnerable to overnutrition and weight gain above their genetic norm [10]. Third, the correlation between birth weight and ischaemic heart disease was equivocal [7] whereas the correlation between birth weight and blood pressure was clear cut [11]. In twin studies the genetic correlation also appears to be stronger for hypertension than ischaemic heart disease [4] and also for their primary risk factors [12]. It is probable that the reported lack of correlation between weight at 12 months and adult blood pressure in a different and smaller cohort born about 20 years later than the cohort used to study ischaemic heart disease, is an aberration or a consequence of higher incidence of artificial feeding. The Hagerstown Study found that the prevalence of 'hypertensive vascular disease' in adults was strongly and inversely related to weight in childhood [13].

In communities not in famine there is evidence that genetic factors contribute importantly to birth weight in singleton pregnancies [14,15]. Moreover, in the Collaborative Perinatal Project of the National Institute of Neurological and Communicative Disorders and Stroke, birth weight in 20,000 normal singleton term infants was the single most important determinant of individual growth during the first seven years of life [16]. This and the fact that twins retarded in growth at birth catch up with singletons by seven years [9], and the fact that monozygotic twins are highly concordant in weight at seven years [8,9], underscore the contribution of genetic factors to birth weight in normal singleton pregnancies.

Thus an alternative explanation for Professor Barker's findings is that while acquired obesity is a risk factor for cardiovascular disease, genetic 'overweight' is protective [17–22].

This hypothesis would also explain why birth weight and placental weight had opposing effects on adult blood pressure [11]. Systolic pressure fell by around 10 mmHg with increasing birthweight and rose by around 12 mmHg with increasing placental weight [11]. Heaviness in infancy is a marker for genetic obesity [23] and placental weight is a marker for maternal fatness [24]. Underweight infants of overweight mothers have beeen shown to be at increased risk of excessive weight gain [25] and overweight adults who were underweight during childhood have double the risk of cardiovascular disease compared to overweight adults who were overweight during childhood [13]. Conversely, marathon runners who had formerly been overweight had significantly higher plasma concentrations of protective high-density lipoprotein cholesterol than matched runners who had never been overweight [26].

Thus Professor Barker's results may not be evidence that intrauterine growth retardation increases the risk of cardiovascular disease but rather evidence that genetic heaviness in infancy is a marker for relative immunity against cardiovascular disease.

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Czechoslovakia revisited

Sir—We were delighted by Dr Suri's article published in the April issue of the *Journal.* Dr Suri gave an excellent lecture on The organisation and funding of the geriatric services in the UK at the course on geriatric medicine during his stay in our country. It created such interest that he has been asked to publish it in a Czechoslovak medical journal.

Our Gerontological Society also has very good contacts with the University Department of Geriatric Medicine at the University of Wales in Cardiff. These contacts started five years ago with a visit to Cardiff by Dr Reban who is vice-president of the Czech Gerontological Society.

Eight Czechoslovak geriatricians took part in the first British–Czechoslovak Geriatric Symposium in Cardiff in 1987. The second symposium was held the next year in Prague and the third one in Cardiff in 1990. Five British colleagues participated in the last symposium which was held on 16 May 1991 in Bratislava. The papers will be printed in the fifth volume of *Current Problems of Geriatrics.*

Czechoslovak geriatricians have appreciated the high quality of care for the elderly in the UK for a long time. The British system of care for the elderly is the model for the currently prepared reform of geriatric services in Czechoslovakia.

The Czecho-Slovak Gerontological Society was founded in 1969 and has currently about 1500 members. Its main purpose is to spread knowledge of advances in the field of gerontology and geriatrics. This aim is achieved by holding seminars, conferences and symposia in various towns of our country. The geriatric section is a very active, integral part of the Society.

The structure of Czecho-Slovak Gerontological Society reflects the federal structure of our state. It is composed of the Czech Gerontological Society and the Slovak Gerontological Society. Officers from each society alternate for a period of two years in the administrative charge of the combined society.

S. KRAJCIK,
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S. LITOMERICKY,
President of Czecho-Slovak Gerontological Society

Lumbar Puncture at The National Hospital, Queen Square

Sir—Dr Alex Sakula's article (April 1991) on a hundred years of lumbar puncture reminded me of my past interest in cerebrospinal fluid.

At the 1964 meeting of the Association of British Neurologists in Winchester, Dr William Gooddy, that year's President of the Association, introduced me to Sir Gordon Holmes. I wanted to know who introduced lumbar puncture to Queen Square and when. Sir Gordon, then aged 88, tall and upright, was clearly mentally alert and spoke with a deep, commanding voice. The reply to my question was:

"Let me see, was I on the house in One or in Two?' He peered into the distance and after a seemingly long pause turned to me, slightly bent forward, and then came the explosive: 'Never mind, it doesn't matter. Gowers was against it! So we did not do it.'

Such was the power of the senior physician of former days that no-one was permitted to do a lumbar puncture until Sir William Gowers retired in 1910. Hence, no British book on cerebral spinal fluid appeared until 1925 [1].

Now that I am senior physician, I occasionally regret not to have the power and influence exerted by my predecessors. But not frequently—democracy is preferable.

Some years earlier I had written to Dr Parkes Weber asking the same question. The reply, on a post card signed but not written by him (he was blind), stated that he thought he was the first to perform a lumbar puncture in London at The German Hospital, Hackney in 1896. His invitation to a surgeon on the staff of the hospital to perform the procedure was declined so he did it himself. But clearly Dr Essex Wynter at the Middlesex Hospital preceded Dr Parkes Weber's lum-