The Need for Revising the MRCP Regulations

Sir,

The current MRCP regulations are particularly detrimental to overseas doctors who want to have this qualification.

With several centres now opened by the Colleges in Asia and Africa for Part I of the MRCP, the examination has been truly and genuinely internationalized. However, during the framing of the Part II regulations no deep consideration has been given to the employment situation and training capacity of these countries as well as other countries of the Third World from whence come the candidates for this purely British style examination.

First, a 5-year limit has been set to clear Part II—from the date of passing Part I. This ensures that overseas doctors, at that stage, must ignore the rural health service of their respective countries (including both voluntary and mandatory) and must gravitate to large hospitals at the first opportunity. Even this may not solve the problem of training as the specific MRCP courses are either non-existent or only one or two in number.

These are in abundance in the UK and provide examples of a range of cases and styles required for the clinical section of Part II. Therefore, not surprisingly, such candidates try to obtain scholarships for the UK. The standard duration of such scholarships is 3 years and the candidate tries his/her best to pass, but in the process has no choice but to appear again and again. This means that if he or she completes the maximum allowable six attempts, then the Third World scholarship is wasted. Ultimately, more than 25% never pass Part II because of this limit on attempts.

Those overseas candidates who cannot get a scholarship go as private students, and have the option of either passing PLAB (which takes 1 year and has a maximum pass-rate of 28%) and get a job in geriatrics or remain in the UK to do MRCP courses and take examinations again and again.

One might ask ‘why so often?’ The reason is that the UK Home Office does not allow extension of stay unless shown the entrance examination letter each time. Even with such a plea the maximum time allowed for private study candidates is often 2 years, so the pressure to sit examinations often has been present for years. This explains why so many overseas doctors have finished their six attempts in the last few years and are back home doing private practice in ENT and ophthalmology after spending 6 or 7 years getting their training in medicine. So much for the services which the Royal Colleges of Physicians (UK) are rendering for the causes of internal medicine beyond the frontiers of the UK.

As the Colleges cannot ensure equal training opportunities to the 3000 MRCP candidates who appear every year, and as the Colleges do not run any long MRCP training programmes, and because the Royal Colleges of Surgery and Gynaecology and Obstetrics maintain high standards without such restrictive regulations, then the bar on time and number of attempts should be lifted. The Australian Royal College allows two chances in clinicals to those who pass the written section, in each attempt.

The second point in favour of lifting these restrictions is that the examination itself is inadequate in terms of time, range and fixed reproducible standard. For example, it is extremely common to pass the written section once and fail it the next time and pass it again just to fail it the next time. This game goes on and on and even if a candidate obtains the usually maximum score of 17 points out of 20, he or she has to take the written section again. In an average attempt about 37% of candidates stop at the written part as they are not allowed to proceed to the clinical. One is not allowed to defend the answers and opinions in the written part. Disputes about the marking of the paper are settled by college employees and the standard practice of referring to two independent examiners is not followed. Also, unlike the ECFMG authorities in the USA who allow hand re-checking of computer marked answers at extra charge—no such facility exists for the candidates taking MRCP Part I. Furthermore, a whole system is frequently represented by a 4-minute short case in the Part II examination—such is the depth and time given to test the candidates.

These restrictions on Parts I and II will one day make these examinations, together with internal medicine as a whole, an unattractive and risky option for candidates. At present, the pass rate of foreign doctors at Part II is a little over 20% compared with the pass rate of UK qualified doctors of a little below 40%.

The regulations of the Royal Colleges of Physicians, the Home Office, and the General Medical Council do not help to provide equal opportunities. There is perhaps a need for a new Royal College co-ordinating with the Department of Health and the universities and institutes of specialized training to impart real training to candidates. Such a proposed institution might be more sympathetic to the cause of Third World doctors and respond to the challenges of ‘glasnost’ and ‘perestroika’ which are not only for Soviets.

Another option is the possibility of co-ordination between existing Asian and African postgraduate bodies into a single unified system. The Arab Board, The West African Board, UNICEF, OIC and OAU could help in the setting up of such a system to provide equal opportunities for all.

E.M.S.

Name and address supplied


Sir,

Your correspondent criticizes the MRCP examination on the following grounds:
1. It is UK oriented.
2. Marks in Part I (multiple choice, marked by computer) are not re-checked on request.
3. Part II must be completed within 5 years of passing Part I.
4. The number of attempts allowed at Part II is only six.
5. Disputes about the Part II written section are settled by ‘college employees’.
6. Insufficient time is given to short cases in the clinical examination, ‘a whole system is frequently represented by a 4-minute short case’.

The MRCP(UK) is an examination of the Royal Colleges of Physicians of Edinburgh, Glasgow and London, but I shall try to answer the points.

1. It is perfectly true that the Membership is a UK-oriented examination. Given its history, it is difficult to see how it could be otherwise. The Colleges have for more than 10 years encouraged other countries to set up their own postgraduate examinations in medicine, which might be more suitable to local conditions than is ours. The fact that they have done this to only a limited extent may be due to a feeling among junior doctors that such a local examination would not carry the prestige in other countries that the MRCP does, and to the continued popularity of the Membership. I do not see that the UK Colleges can be blamed if a large number of overseas candidates want to take their Membership examination.

2. The numbers of candidates taking Part I is about 5000 a year. To check all results would be a heavy burden but we do run checks on a random selection of papers and in no case has the computer been found to have made a mistake.

3/4. The purpose of the 5-year attempts limit is intended to be a kindness to candidates. Five years is a long time in a junior doctor’s career; we thought it reasonable to limit the number of attempts and the time during which they can be made so that a young doctor does not spend excessive time, money and effort on attempting Part II. The fact is that candidates have a decreasing chance of passing the examination with each attempt. After three or four failures success becomes unlikely. Surely it is better that failure should be accepted sooner rather than later?

In cases of hardship when, for example, a candidate is unable to come to the UK to complete his attempts in time because he cannot leave his own country or is in military (or rural) service, the Colleges have the power to extend the time limit and frequently do so. The candidate has only to write and explain why he cannot complete his attempts in time and he will be treated sympathetically.

5. ‘College employees’ (meaning, I assume, the non-medical staff) do not make any decisions about the examination. They administer it and act as spokesmen for the Examining Boards.

6. In the short case section of the clinicals not all systems can be examined. It is surely better that a candidate should see a variety of cases than only one or two. Each case must, therefore, be examined for only a short time, hence the term ‘short cases’.

I have done my best to answer your correspondent’s complaints but I can easily see why he makes some of them. It is hard to have to come to the UK to take clinicals and then to be examined in what may be unfamiliar surroundings. It is certainly wise to work and study in this country before taking the examination and that involves time and money. I do not believe it would be a kindness on our part to offer more of the first — I wish we had more of the second.

Having said all this, we do keep the Membership examination continuously under review. Although it is, I think, a searching and fair examination it can always be improved. There is a working party sitting now considering all aspects of the examination to see if any improvements can be made. Nothing is fixed permanently. The examination is continuously evolving. We welcome questions and criticisms when we will, as I have attempted to do here, explain the reasons for our actions and what changes are proposed or rejected.

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Natural Honey Prevents Indomethacin and Ethanol-induced Gastric Lesions in Rats

Sir,

We read with interest the paper by Ali et al. (Saudi Med J 1990; 11: 275) regarding the effect of honey on experimental gastric ulceration, and would like to raise the following points regarding its content:

1. The dosage of honey used to produce almost 100% protection against gastric damage was rather high (5 g/kg). If this dose was to be given to a human patient (average weight 70 kg) it means that the patient should receive 350 g of honey at one time! It is interesting to recall here that some honey samples can be toxic. A patient experienced severe bradycardia and hypotension after ingesting about 30 g of honey.1

2. It would have been of interest to find out if administering small doses of honey over a long period would be as effective against gastric ulceration as the bolus dose used by the authors. The problem of ulcer recurrence could have been investigated after stopping the honey treatment.

3. No reference drug (a known cytoprotective drug or H2-blocker) was included in the study. This would have facilitated comparison and evaluation of the anti-ulcer effect of honey.

4. Although this may be the first report on the effect of honey on gastric ulceration, honey has long been known to aid in the healing of wounds.2

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References


Sir,

We would like to thank Dr Badreddin Ali and his colleagues for their interest in our paper and should like to clarify some points raised by them.

1. It is conceivable that the extrapolation of animal data to the human clinical condition is not always possible. It is not uncommon to use clinically unrelated doses in acute experimental conditions, for example, the therapeutic dose of sucrose is 50 mg/kg whereas doses of 300-1000 mg/kg were used to evaluate its effects on the experimental gastric lesions in rats. At present the anti-ulcerogenic factor present in honey is not known. However, carboydrates (constituting 80% of its composition) were without any protective effects. Thus, the question of volume would not arise, if it was possible to isolate the protective factor present in honey. Furthermore, substantially lower doses have been found to be effective depending on the time of administration of the honey (unpublished observation).

We agree that bees can produce honey which is poisonous. By far the largest number of such harmful honeys are reported to be produced from species of rhododendron, azalea, andromeda and Kalnica. The honey used in this study did not fall in those categories. Furthermore, honey can cause hypersensitivity reactions in susceptible individuals.

2. We completely agree with their suggestions for chronic treatment. However, as far as we know, there is no suitable animal model resembling human gastroduodenal ulcers. Studies are in progress to answer this question in the near future.

3. No attempt has been made to elucidate the mechanism of action of honey in this study. However, it is well established that cytoprotective prostaglandins afford protection against both indomethacin and ethanol-induced gastric damage. Similarly, the H₂-blocker, cimetidine (100 mg/kg, i.e. 7000 mg for 70 kg humans) inhibited indomethacin-induced gastric lesions but failed to protect against ethanol-induced damage at a dose of 200 mg/kg in rats. Thus, it was thought that inclusion of these reference substances would not provide further new information unless parameters such as acid secretion, mucus determination etc., were studied and the results compared with those obtained using honey.

4. We have no comment on this question.

Finally, we would like to say that our paper was a preliminary report on the effects of honey on gastric damage. It was not possible to investigate and to incorporate every detail in a single paper.

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References


Alkaptonuria: A Case Report

Sir,

Dr Mohay-ud-din's case report of a Yemeni patient with alkaptonuria made interesting reading. We report another case of ochronosis in a Saudi patient.

A 42-year-old Saudi male presented with rupture of the left Achilles tendon during slow running. Physical examination revealed no other positive findings. Past history revealed that the patient had ruptured his right Achilles tendon in January 1986, also during slow running. Both times the tendon was repaired and he had full recovery. The patient had a history of chronic low back pain and renal stones.

Deep blue-black discoloration of the tendon was noted at the time of both surgeries. Histopathologic examination revealed mucoid degeneration of the tendon with intra- and extracellular deposition of light brown pigment granules which stained black with cresyl violet. The urine was analysed for homogentisic acid, was strongly positive and the diagnosis of ochronosis was confirmed.

The patient had suffered low back pain since 1983. Serial X-rays of his spine from 1983 to the present were reviewed in the light of the diagnosis. They revealed changes consistent with chondrotic spondylopathy with calcification of all the intervertebral discs and severe narrowing of the spaces with vacuum phenomenon. The patient has since developed blue-black discoloration of his pinnae. He gets temporary relief of low back pain from NSAID and physiotherapy.

Alkaptonuria/ochronosis is an autosomal recessive, inherited disorder of the metabolism. The enzyme homogentisic acid oxidase is deficient in the liver and kidney. Although most of the homogentisic acid is excreted in the urine, some of it eventually, gradually accumulates in connective tissues, polymerizes and binds irreversibly to the collagen fibres. Although the metabolic defect is present from birth the disease manifests itself during the fourth to fifth decade due to deposition of the pigment in the connective tissue.

The incidence of this disease, as reported in the literature, varies from 0.1 to 5 cases per million worldwide.1 In the Middle East where there is a high degree of consanguinity, the incidence has been shown to be 320 per million births.2 O'Brien et al. in their Review of World Literature in 1963 do not report any cases from Saudi Arabia.3 Physicians in the Middle East
need to be aware of this disorder when dealing with young patients with arthropathy. Diagnosis has only to be thought of to be made. Diagnosis at an early age and investigation of other family members is important for genetic counselling. Although the disease probably does not influence the life span, it contributes significantly to morbidity due to the severe degenerative changes in the spine and large joints. Few cases of a ruptured tendo-achilles secondary to ochronosis have been reported. O'Brien et al. report only two cases. The ruptures in this case were at the insertion of the tendon into the os calcaneum, compared to rupture at a higher level commonly seen.

Here we report alkaptonuria/ochronosis in a Saudi male which, to our knowledge, is the first case reported in a Saudi. A detailed case report is under preparation and will be published later.

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References


(Reprinted in Am J Med 1963; 34: 813-838.)

Sir,

I am most interested to read the letter from Drs Kagalwalla and Beg reporting a case of spontaneous rupture of tendo-achilles on both sides due to degenerative changes caused by the deposition of polymerized homogentisic acid. The diagnosis of ochronosis is invariably missed in the early stages, therefore, it should be kept in mind in dealing with an unusual musculoskeletal disorder. It must be remembered that a case of alkaptanoluria could also present in an Eye, ENT, Dermatology, or Cardiology Clinic. The simple test of voiding the urine in a glass jar and leaving it to stand overnight helps in making the diagnosis. In a case of alkaptanoluria the urine, if allowed to stand, becomes dark brown. This can subsequently be tested in the laboratory for homogentisic acid.

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Gastrointestinal Complications of Behcet's Disease

Sir,

A 41-year-old Yemeni truck driver was admitted to Asir Central Hospital (ACH) with fever and 30 kg weight loss over 2 months. His past medical history was negative and systemic enquiry was not contributory.

On examination, he was sick-looking with a temperature of 38 °C. He was pale but had no lymphadenopathy. The rest of the examination was normal except for a 0.5 cm tender ulcer on the left scrotum and a smaller one over the adjacent thigh.

On further questioning at this point, he reported that he had scrotal ulcers for 2 weeks and mouth ulcers 6 months before.

The complete blood count was normal except for a haemoglobin of 10.6 g% and an ESR of 122 mm/h. Several blood smears and cultures were negative. Blood chemistries including liver function tests were normal. Urinalysis was normal. Stools for ova, parasites and occult blood were negative. The chest radiograph, ultrasonound of abdomen and the echo-cardiogram were normal. Tests for VDRL and HIV were negative. Serologies for brucella, typhoid and toxoplasma were negative. Anti-ANCA and anti-DNA were negative. A Mantoux test was positive and so was the serology for kala-azar.

Three weeks after admission, he developed painless melena requiring blood transfusions. No source of bleeding could be detected by either upper gastrointestinal endoscopy or sigmoidoscopy.

He developed acute abdomen 4 weeks later and emergency laparotomy revealed peritonitis and multiple perforations along the 20 cm length of the terminal ileum which was resected. Histopathology revealed transverse ulcers and non-specific enteritis. Antibiotics and later antituberculosis medications were administered but patient continued to be febrile. As the diagnosis of Behcet's disease was entertained even from the start, prednisone was administered.

While on steroids, he developed a fistula at the previous site of a drainage tube in the right iliac fossa which later bled massively. An urgent ultrasound and CT scan of the abdomen revealed a large abdominal aortic aneurysm.

He was referred to King Khalid University Hospital in Riyadh where he had aneurysmectomy. He did extremely well on steroids until 1 year later when he again presented with abdominal pain and an ultrasound revealed another huge abdominal aortic aneurysm for which surgery was recommended.

Even though Behcet first described the disease with only dermatological manifestations, subsequent reports in the literature have proven that Behcet's disease is a multisystem disease with protein manifestations. During his prolonged hospitalization, the patient described above developed a constellation of dramatic signs and symptoms, particularly related to the gastrointestinal tract. He developed massive GI bleeding with undetermined focus requiring blood transfusions. He later developed an acute abdomen with perforations of the terminal ileum and peritonitis. Non-specific ulcerations of the small intestine occur in Behcet's disease, 75% in the ileum and caecum, and the rest in the jejunum, colon and oesophagus. They may present with signs of bleeding, abdominal pain or mass, obstruction and/or perforation as they did in our patient.
This case illustrates the serious GI manifestations with which patients with Behçet’s disease may present.

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References


**Can Echocardiography Predict Selenium Deficiency in Patients on Home Parenteral Nutrition?**

Sir,

The association between selenium deficiency and congestive cardiomyopathy in humans, has been well documented. 1,2 Patients on home parenteral nutrition (HPN) are a group particularly at risk. 3,4 Unfortunately, measurements of plasma selenium and red cell glutathione peroxidase (RCGP) levels are not widely available tests. We used echocardiography in patients on HPN to assess their left ventricular function and to determine whether this technique can identify patients with selenium deficiency prior to the onset of clinical symptoms. Thirty-two British patients (16 males, 16 females) on HPN for 6 months to 8 years were studied at Hope Hospital. The ages ranged from 18 to 37 years (mean 25 years). Echocardiographic measurements and assessments were carried out without prior knowledge of the selenium and RCGP results. The Mann–Whitney U-test and Spearman correlation coefficients were used for statistical analysis.

Selenium deficiency was common in our patients. Of the 32 patients evaluated at the first clinic visit, 16 (53%) (who had been on HPN for a minimum of 12 months), were found to have selenium deficiency (plasma selenium level <0.8 μmol/l). The mean selenium level in these patients was 0.69 μmol/l (95% confidence limits 0.6–0.78). The mean RCGP was 11.4 IU/g haemoglobin (range 10–12.8). Of the echocardiographic parameters examined, only fractional shortening percentage (FSP), ejection fraction (EF) and mitral valve E-point-septal distance (MVEP) were shown to be useful predictors of selenium deficiency. Both FSP and EF exhibited a significant correlation (r = 0.448; p = 0.005) with plasma selenium level.

**Table 1**

<table>
<thead>
<tr>
<th></th>
<th>Low ejection fraction</th>
<th>Normal ejection fraction</th>
<th>Total</th>
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</thead>
<tbody>
<tr>
<td>Normal plasma selenium</td>
<td>2</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Low plasma selenium</td>
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<td>17</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>13</td>
<td>32</td>
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In addition, MEVP showed a significant correlation with both plasma selenium (r = −0.46; p = 0.008) and RCGP (r = −0.46; p = 0.01).

Seventeen of 32 patients studied had subnormal ejection function (less than 64%) and a low plasma selenium level while 13 patients had normal ejection fraction and normal selenium.

There were only two patients who had a subnormal ejection fraction while their selenium level was normal. This gives a specificity of 87% and a sensitivity of 100% (Table 1).

Examination of the second and third clinic visit data showed that an increase in plasma selenium was accompanied by a corresponding rise in left ventricular contractility (p = 0.006).

Selenium deficiency has been regarded as a cause of heart muscle disease called ‘Keshan disease’ in China. The disease has a regional distribution in agricultural areas where the selenium content of the soil is reduced. 5 The acute form is characterized by sudden onset of impaired heart function, whereas individuals with chronic cases exhibit moderate or severe heart enlargement with varying degrees of heart failure. 6 Addition of selenium to the diet of peasants in Keshan County virtually eliminated the disease. 7

Low selenium status in patients on home parenteral nutrition (HPN) has been associated with cardiomyopathy and muscle pain. 8 Our findings showed that selenium deficiency is common (53%) in patients on HPN. Many of these patients have suboptimal left ventricular function. There was a significant correlation between the plasma level of selenium and both ejection fraction and fractional shortening percentage. Echocardiography appears to form a useful adjunct to the standard biochemistry assay, and in cases where a hospital does not have readily available biochemical services, echocardiography may be used as an indicator of a patient’s selenium status.

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References

Letters to the Editor


Transient Blindness During Pregnancy: Report of Three Cases

Sir,

Transient blindness during pregnancy is a rare phenomenon and only a few cases are reported in the world literature. Pregnancy-induced hypertension is a well known cause of such an entity. We had the opportunity to study three cases with various aetiologies during a period of 5 years out of 6500 deliveries at Al-Shaty Teaching Hospital, Jeddah.

The first case involved a 23-year-old Saudi woman, Gravida four, Para three who had regular antenatal check-ups. When she was admitted in labour she was found to have severe fulminating pre-eclampsia, with complaints of severe headache and blurring of vision. Examination showed puffy face, blood pressure 170/130 mmHg, fundi: normal, urine examination showed 3+ albumin. Her blood pressure was controlled with hydralazine and valium drips. The patient delivered a live baby girl, following which the mother had a generalized convulsion. In the postnatal period she showed postictal psychotic behaviour, aggressiveness, hallucinatory behaviour and loss of vision, and remained so for 78 h. Following this, she made gradual but total recovery. A CT scan showed moderately large attenuated zones in both posterior tempo-occipital areas.

The causative primary lesions described have included spasm of the retinal arterioles, choroidal vascular insufficiency, acute ischaemic optic neuropathy and central retinal artery occlusion.1,2

In the absence of retinal changes one has to look for central disturbances of visual cortex. In a similar case, Grimes et al.3 demonstrated bilateral cerebral lesions in the occipital cortex which resolved completely by 7 months after delivery.

The second case involved a 30-year-old, Sudanese woman who was seen during the 30th week of gestation for deteriorating vision of 48 h duration. She had eclampsia in her previous pregnancy. She weighed 112 kg, with a blood pressure of 220/140 mmHg. Urine examination showed no proteinuria. Neurological examination was non-contributory except that vision was limited to finger counting at 6 inches, with bilateral gross papilloedema. Her MRI of brain was normal. Hence a lumbar puncture was done after control of the blood pressure was established and the opening pressure of the CSF was 430 mm of water. She was treated with hypotenensives and acetazolamide and made a good recovery.

Benign intracranial hypertension is aggravated during pregnancy, occurs in obese patients, and is a totally reversible condition when it is diagnosed early and managed properly.

Partial or complete detachment of the retina has been reported during pregnancy. Of 132 cases of eclampsia, seven had retinal detachment.2 In our third case a 17-year-old primagravida presented with acute loss of vision in both eyes of 48 h duration. She had no past history of visual or neurological complaints. Ophthalmoscopic examination showed bilateral total retinal detachment, which was successfully treated with laser therapy. She went on to term and delivered a healthy male baby.

These three cases are examples of acute emergency eye conditions during pregnancy and they need to be recognized early for total reversal.

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References