LETTERS TO THE EDITOR

Management of leg ulcers

EDITOR,—I congratulate Sarkar and Ballantyne for their excellent review on the management of leg ulcers.[1] Additional comments regarding investigative methods of peripheral arteriopathy and the management of venous leg ulcers are, however, necessary.

In the United States, more than 1200 amputations are performed each week for a diabetic foot ulcer.[2] One of the main cause of non-healing ulcer in diabetics is peripheral arteriopathy, which is often underestimated. In these patients, the ankle/brachial pressure index (ABI) is not sensitive enough. Falsely raised values are frequent in the case of incompressible arteries (mediocalcinosis), especially in diabetics, but they are also found in elderly patients or in those with chronic renal failure. An ABI in the normal range is also observed in hypertensive or diabetic subcutaneous microangiopathy. Therefore, an arterial duplex ultrasound is recommended as well as recording the toe blood pressure or the skin perfusion pressure by plethysmography or laser Doppler.

Almost all venous leg ulcers can heal with adequate compression therapy. However, 30% to 40% of patients with venous leg ulcers have an isolated superficial venous incompetence that can be treated with surgery alone, avoiding long term use of compression bandages and reducing recurrence rate.[3 4] Some degree of superficial reflux in the ulcer area is also found in patients with deep venous incompetence. Treatment of these local haemodynamic abnormalities may be an important factor in the healing of the ulcers and in prevention of their recurrence. Consequently, venous surgery is a good alternative for many patients, especially the elderly who have difficulties putting on compression stockings and those with peripheral arteriopathy, which may be aggravated with it. Phlebectomies of saphenous veins and their branches with ligation of incompetent perforating veins can be done under local anaesthetic and are therefore particularly suitable for elderly patients.[5] Preoperative duplex ultrasound is, however, necessary to localise precisely the incompetent venous segments.

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1 Sarkar PK, Ballantyne S. Management of leg ulcers. *Postgrad Med J* 2000;**76**:674–82.
2 Centers for Disease Control and Prevention. *National diabetes facts sheet: national estimates and general information on diabetes in the United States*. Rev Ed. Atlanta, GA: US Department of Health and Human Services, Center for Disease Control and Prevention, 1998.
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4 Barwell JR, Taylor M, Deacon J, *et al*. Surgical correction of isolated superficial venous reflux reduces long-term recurrence rate in chronic venous leg ulcers. *Eur J Vasc Endovasc Surg* 2000;**20**:363–8.
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Amiodarone induced hyperthyroidism

EDITOR,—It is with great interest that we have read the correspondence between Findlay and Seymour[1] and Bhattacharya and Bhattacharya[2] relating to amiodarone induced hyperthyroidism. We recently had a case of a 55 year old man who has had atrioventricular nodal ablation and pacemaker implantation for refractory atrial fibrillation. He originally presented with atrial arrhythmias in March 1999 and his thyroid function and "atrial fibrillation screen" were all normal including a structurally normal heart on transthoracic echo. He had no symptoms related to thyroidal illness bar palpitations. Despite digoxin, flecainide, and amiodarone (he could not have β-blockers because of asthma) his atrial fibrillation was highly symptomatic and it was increasingly difficult to control his ventricular rate successfully. He was therefore referred for ablation, which took place in January 2000. From March 1999 to January 2000 he had thyroid function tests performed at least four times, all of which were well within normal limits. Amiodarone treatment was stopped in January after his ablation. He was reviewed in September complaining of feeling very hot and sweaty and was found to be biochemically hyperthyroid with a thyroid stimulating hormone concentration of 0.01 mU/l, free thyroxine 94.1 pmol/l, and free triiodothyronine 18.5 nmol/l. He has been started on carbimazole.

We are not sure of the aetiology of this man's thyroid problem and there was no history of pain in the neck area suggesting thyroiditis or a flu-like illness. He had been off amiodarone for nine months but there is a possibility, albeit unlikely, that his original presentation was with refractory AF and subbiochemical thyroid disease. The most likely explanation, however, is that this is amiodarone induced hyperthyroidism as this has been reported to occur up to several months after stopping amiodarone treatment.[3]

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1 Findlay PF, Seymour DG. Hyperthyroidism in an elderly patient. *Postgrad Med J* 2000;**76**:173–5.
2 Bhattacharya S, Bhattacharya A. Hyperthyroidism in an elderly patient. *Postgrad Med J* 2000;**76**:597–8.
3 Trip MD, Wiersinga W, Plomp TA. Incidence, predictability, and pathogenesis of amiodarone induced thryrotoxicosis and hypothyroidism. *Am J Med* 1991;**91**:507–11.

Dr Findlay responds:

I was very interested to read of the case presented by Sinha *et al* of a patient who had been treated with amiodarone and who developed hyperthyroidism several months after stopping the drug. Before the development of the hyperthyroid state the thyroid function was normal on four occasions while on amiodarone. The diagnosis of amiodarone induced hyperthyroidism is, as the authors state, very likely, although it would be important to check thyroid autoantibodies to exclude thyroid dysfunction unrelated to treatment with amiodarone.

Giant cell (temporal) arteritis and temporal artery biopsy

EDITOR,—I have read with interest the article by Dr Karseras, entitled "Ophthalmology and general medicine".[1] The author states that "Prepresentation constitutional malaise, tenderness of temporal arteries, raised erythrocyte sedimentation rate (ESR) and rarely, biopsy will usually exclude this potentially blinding and lethal condition". I would like to emphasise some salient points particularly regarding the ESR and biopsy. A more specific and accepted description of the disease is "giant cell (temporal) arteritis" to distinguish it from Takayasu's arteritis. It is important to maintain a high index of suspicion of the disease when seeing patients aged 50 years or older. The condition is an ophthalmic emergency. The condition can cause permanent total blindness unless urgent treatment with corticosteroids is provided. Treatment should not be delayed while awaiting results of the ESR or biopsy. Blindness can result from ischaemic optic neuropathy or retinal artery occlusion.[2] Death from myocardial infarction or stroke can occur. The ESR is raised in about 90% of cases.[3] Hence a normal ESR does not exclude the disease.

Most authorities recommend that temporal artery biopsy should be performed in all cases regardless of the presence of a firm clinical diagnosis and raised ESR. Treatment involves the long term use of oral prednisolone. The establishment of a biopsy proved diagnosis can be helpful particularly in cases where significant side effects from prednisolone occur.

The biopsy should be ipsilateral to the side of the visual symptoms or headache. Contralateral biopsy can be considered in cases where the histology of the ipsilateral biopsy is negative. The biopsy should preferably be performed within one week of the start of steroid treatment.

The biopsy is positive in about 70% of cases of giant cell (temporal) arteritis.[4]

Skip lesions may be present along the course of large and medium sized arteries. In addition, vasculitis may affect the ophthalmic artery but not necessarily the temporal artery. Hence negative histology does not necessarily exclude the diagnosis.

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1 Karseras A. Ophthalmology and general medicine. *Postgrad Med J* 2000;**76**:551–4.
2 Aiello PD, Trautmann JC, McPhee TJ, *et al*. Visual prognosis in giant cell arteritis. *Ophthalmology* 1993;**100**:550–5
3 Ellis JD, Munro P, McGettrick P. Blindness with a normal erythrocyte sedimentation rate in giant cell arteritis. *Br J Hosp Med* 1994;**52**:358–9.
4 Glaser JS. *Duane's ophthalmology*. Clinical volume 2. Hagerstown, MD: Lippincott-Raven, 1998 (chapter 5).

Dr Karseras responds:

I thank David Infeld for his interest in my article. There is not much in his letter of commentary that I would disagree with. Certainly where there is a high index of suspicion and symptoms fit the clinical picture I would personally start treatment on systemic steroids before the results of the ESR or biopsy. I am not too sure about undertaking a temporal artery biopsy in all cases, regardless of the presence of firm clinical diagnosis. Possible side effects from prednisolone do not seem to

me to be a good reason to subject patients to this less than easy procedure in all circumstances.

I usually have not needed to resort to temporal artery biopsy in cases that have come clinically to the ophthalmic department. A balanced clinical view would be to assess the likelihood of temporal arteritis being present in a patient who has possible impending blindness. The decision for biopsy is based on clinical acumen and wisdom. Some patients may be treated unnecessarily but others may well be saved from the blindness that can come on so quickly and bilaterally.

I am pleased to see that Dr Infeld recognises the presence of difficult cases, which can still have drastic consequences despite a normal ESR and biopsy.

I suppose 40 years of personal clinical experience of the occasional case of temporal arteritis gives one the confidence to advocate "better safe than sorry" and accept that it is better to have some side effects, which may be unnecessary, rather than some irreversible loss of sight. But I agree with most of Dr Infeld's comments.

Foreign bodies in the nasal cavities

EDITOR,—We read with great interest the review of foreign bodies in the nasal cavities compiled by Kalan and Tariq in a recent issue.[1] The authors state that there are no comprehensive discussions on the subject of foreign bodies in the nose, but appear to have overlooked previous reviews by Werman[2] and Baker,[3] perhaps because their methodology lacks a systematic approach. Furthermore, we feel that the article affords undue emphasis to the role of the ear, nose, and throat (ENT) surgeon, and the requirement for their early involvement in this condition.

Emergency physicians frequently encounter foreign bodies in the nose, often in children brought to the hospital by their parents soon after insertion. In this group the presentation is not an offensive unilateral nasal discharge, as stated by Kalan and Tariq, but a history of recent insertion with local irritation. We have published a systematic review of foreign bodies in the nose and ear, including techniques for their removal in the emergency department.[4] Current evidence indicates that the vast majority can be removed without complications by suitably trained emergency department staff, and without the need for ENT involvement. Locally we have been able to demonstrate considerable success with an appropriate teaching programme and a specific set of instruments for foreign body extraction.

It seems likely that Kalan and Tariq's article is biased by the fact that only difficult, complex, or late presenting cases tend to be referred to an ENT surgeon. We wish to emphasise the important role that emergency physicians have in this common condition.

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1 Kalan A, Tariq M. Foreign bodies in the nasal cavities: a comprehensive review of the aetiology, diagnostic pointers, and therapeutic measures. *Postgrad Med J* 2000;**76**:484–7.
2 Werman H. Removal of foreign bodies of the nose. *Emerg Med Clin North Am* 1987;**5**:253–63.
3 Baker MD. Foreign bodies of the ears and nose in childhood. *Pediatr Emerg Care* 1987;**3**:67–70.
4 Davies PH, Benger JR. Foreign bodies in the nose and ear: a review of techniques for their removal in the emergency department. *J Accid Emerg Med* 2000;**17**:91–4.

BOOK RECEIVED

The receipt of these books is acknowledged and this listing must be regarded as sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed, space permitting. The journal does not publish unsolicited reviews.

Schizophrenia & Mood Disorders. Edited by Peter F Buckley and John L Waddington. (Pp 353; £35.) Butterworth Heinemann, 2000. ISBN 0-7506-4096-0.

Atlas of Ophthalmology. Edited by Richard K Parrish II. (Pp 678; £150.) Butterworth Heinemann, 2000. ISBN 0-7506-7075-4.

Epidemiology of Work Related Diseases. 2nd Edition. Edited by Corbett McDonald. (Pp 556; £62.) BMJ Books, 2000. ISBN 0-7279-1432-4.

BOOK REVIEWS

The reviewers have been asked to rate these books in terms of four items: readability, how up to date they are, accuracy and reliability, and value for money, using simple four point scales. From their opinions we have derived an overall "star" rating: * = poor, ** = reasonable, *** = good, **** = excellent.

Research Approaches in Primary Care. Edited by Andrew Wilson, Martin Williams, and Beverley Hancock. (Pp 176; £15.95.) Radcliffe Medical Press, 2000. ISBN 1-85775-392-5.****

This text is one of a series of books supported by "Trent Focus for the Promotion of Research and Development in Primary Health Care". Its objectives are to provide the beginner in primary care research with an overview of appropriate methodology and an awareness of when and how to obtain specialist advice.

It is a delightful book to read as the text is laid out with clearly labelled, short, succinct, and highly relevant sections. It contains chapters on an introduction to research methodology, experimental designs, qualitative research, surveys and questionnaires, the use of interviews in a research project, and data collection by observation as well as a glossary and index. As there are five contributors, there is inevitably a degree of duplication. However, good cross referencing compensates.

Basic research terminology is defined and important information on topics such as sampling, calculation of sample size required, confidence intervals, and types of questionnaires is clearly set out in language which uses the minimum of jargon. Boxes and bullet points emphasise important facts and the text is interspersed with exercises which maintain reader's interest and give feedback on understanding. The chapters conclude with a summary, answers to the exercises and references, further reading, and sometimes suggestions for other resource information.

At £15.95, this book is certainly good value for money and should find a place on the bookshelves of aspiring primary care researchers.

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Helping People with Eating Disorders. A Clinical Guide to Assessment and Treatment. By Bob Palmer. (Pp £17.99; 273.) John Wiley & Sons, 2000. ISBN 0-471-98647-X.****

A multitude of books on eating disorders have been written in the last two decades. Bob Palmer's vast experience in this field easily sets this book apart. Although an overview and not a detailed study of eating disorders, it adopts a pragmatic approach and contains down-to-earth knowledge, thus becoming an invaluable tool for clinicians.

The text is free from dubious research and theoretical frameworks and focuses mainly on the daily clinical work with sufferers. It is divided in two parts. In part I, an account of several pathogenetic factors is given, but the multifactorial nature of the disorders is emphasised. Part II outlines their management. Some of the strongest points of this part are the focus on specific management issues, such as hindrances in progress or making help meaningful and accessible to patients, as well as on general aspects of running a comprehensive eating disorders' service. The use of case scenarios, metaphors, and tables enhance clarity. Although the author points out that the focus of this book is on adult patients, a great deal of the knowledge provided is also applicable to adolescents.

This is an all inclusive, clear, and practical book. It would be useful not only to those who work closely with patients but also to other health professionals, as a means of understanding sufferers and the services available to them. Its concise volume and affordable price makes it accessible to all.

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MRCGP Preparation and Passing. Edited by John J Ferguson. (Pp 194; £17.50.) Royal Society of Medicine Press Ltd, 2000. ISBN 1-85315-468-7.****

This book is based on a highly successful pre-MRCGP course held at the Royal Society of Medicine. Although it contains papers from 21 eminent contributors, the book as a whole presents a comprehensive and up to date guide. The discipline of general practice has few fixed boundaries and therefore a guide through this diverse field of medicine is essential, particularly when one is preparing to become a member of its Royal College.

A candidate will ask "What does the examination test?" The answer is anything relevant to British general practice, a large and complex area which frequently changes. This book assists by presenting current information about issues relevant to general practice, including vocational training, medicopolitical issues in relation to primary care, practice management, audit, and revalidation. There is some repetition of certain topics resulting from the fact that a number of contributors are discussing current issues, however this did not detract from the value of the book.

The structure of the MRCGP examination changes almost as frequently as the medicopolitical scene and therefore the detailed explanation of the elements of the college exam is invaluable. The insights into what examiners are looking for together with some marking schedules are extremely useful.

The book is concise and contains invaluable references to all the current papers with which candidates should be familiar.

I can thoroughly recommend it, not only to candidates working towards their membership examination, but also GP trainers and GP principals alike.

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DIARY

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