Oxpentifylline treatment of venous leg ulcers

SIR,—We do not accept the conclusion of Dr Mary Paula Colgan and colleagues that "oxpentifylline used in conjunction with compression bandaging improves the healing of venous ulcers of the leg."[1] The criteria on which a diagnosis of venous ulceration was based were not stated and it seems that the presence of venous disease was not confirmed by phlebography, plethysmography, or duplex imaging. In a study set in four centres objective criteria for uniform selection of patients should be mandatory. Other diseases that might delay healing of venous ulcers can be overlooked if diagnosis is based only on clinical impression and arterial Doppler pressure measurements.

In our clinic over 20 months venous disease was thought to be the only aetiological factor in 137 patients with leg ulceration. This provisional diagnosis was based on clinical examination and arterial Doppler pressure measurement. After photoplethysmography, blood glucose measurement, and serological investigation, however, 24 patients were found to have diabetes or rheumatoid arthritis, or both, in addition to venous disease. In three patients investigation did not confirm the clinical impression of venous disease.

The healing rate of the control group (34% at six months) is lower than would be expected from other published work.[2,3] In our clinic, using graduated compression bandages, the healing rate of ulcerated legs in which venous disease has been confirmed by photoplethysmography and for which other causes of ulceration have been excluded is 73% at six months. The healing rate of ulcerated legs in which venous disease is not the only aetiological factor is lower (67% at six months).

The authors do not comment on the low healing rate of their control group. They did not exclude aetiological bias and we suspect that the causes of ulceration in the two groups may not be the same. Although it is difficult to compare different studies directly, we do not accept that the healing rate of leg ulcers treated with oxpentifylline and compression bandaging (64% at six months) is an improvement on current methods of treatment where a venous aetiology has been established firmly.

H GAJRAJ
ANDREA JOPP-McKAY
M C STACEY

University Department of Surgery,
Fremantle Hospital,
Fremantle 6160,
Western Australia

1 Colgan MP, Dormandy JA, Jones PW, Schraibman IG, Shanik DG, Young RAL. Oxpentifylline treatment of venous ulcers of the leg. *Br Med J* 1990;**300**:972-5. (21 April.)
2 Backhouse CM, Blair SD, Savage AP, Walton J, McCollum CN. Controlled trial of occlusive dressings in healing chronic venous ulcers. *Br J Surg* 1987;**74**:626-7.
3 Northeast ADR, Layer GT, Stacey MC, Wilson NM, Browse NL, Burnand KG. Effect of fibrinolytic enhancement of venous ulcer healing. *Br J Surg* 1989;**76**:1332.

AUTHOR'S REPLY,—The presence of venous disease was confirmed in all cases by non-invasive testing. Though diabetic patients were not excluded if their ulcers were considered to be venous in origin, only three patients in the entire group were diabetic and all three received oxpentifylline. Additionally, at the first visit plasma glucose concentrations were measured to ensure that occult diabetes had not been overlooked.

We agree that the overall healing rate may appear low, but this was not a normal population of patients with venous ulcers but a group of patients resistant to treatment. All patients had been attending a specialist clinic for at least two months with no improvement in their ulcers. These patients will, therefore, have a lower healing rate than those reported.

We feel that our conclusion that oxpentifylline used in conjunction with compression bandaging improves the healing of venous ulcers is indeed justified.

MARY PAULA COLGAN

St James's Hospital,
PO Box 580,
Dublin

Health checks in general practice

SIR,—Dr Deborah Waller and others invited men and women in the age group 35-64 to a health check for cardiovascular disease.[1] The only indication for this invitation was the age of the patient, and the group that attended were described as "the worried well."

This inverse care effect could have been reduced if case finding had been used in the target group before invitations were given. This can be done with a questionnaire about signs and symptoms of cardiovascular disease, history of cardiovascular disease or hypercholesterolaemia in relatives under 60 years, diabetes mellitus, hypertension, severe obesity, and smoking 20 or more cigarettes a day. Only those with one or more positive answers to these questions should be invited to a check on cardiovascular disease. People at high risk can then be invited, not just because they are the correct age.

A L C J VAN DEN HOGEN

4 Weerder, 1141 JE,
Monnickendam,
The Netherlands

1 Waller D, Agass M, Mant D, Coulter A, Fuller A, Jones L. Health checks in general practice: another example of inverse care? *Br Med J* 1990;**300**:1115-8. (28 April.)

AUTHORS' REPLY,—Dr van den Hogen suggests that initial screening for cardiovascular risk is done by questionnaire so that only those patients at relatively high risk need be invited for appropriate counselling and advice. This would certainly allow more time to be spent dealing with risk factors rather than simply identifying them. Of course, if questionnaires were sent by post this would not necessarily avoid the problem of inverse care because of the possibility of poor response from those at greatest risk. But the new contract requires that risk factors are sought when patients first register with the practice, and this can be done by the doctor, practice nurse, or reception staff when the patient first visits the surgery. In Berinsfield the intention is to use such records to target intervention. As the proportion of general practitioners that uses computerised records grows, data on which selective screening could be based will be increasingly accessible to general practitioners without special questionnaires.

But there are difficulties with this approach. Firstly, the proportion of patients with at least one cardiovascular risk factor is high. We analysed recently data collected from 1047 health checks and found that 451 patients (43·1%) had either a personal history of coronary heart disease, diabetes, hypertension, or long term smoking (more than 10 years) or a family history of premature coronary heart disease (under 50 years). If obesity is added to this list of selective criteria the proportion of patients included is even higher.

Secondly, 85 of the 1047 patients had a total plasma cholesterol over 7·8 mmol/l but only 50 (58·8%) had one or more of the selective criteria cited. Thirdly, self reports of smoking elicited by questionnaire may be unreliable.

None the less, the separation of screening and intervention, with more emphasis on intensive intervention in selected patients, may be a way forward. In the near future we will be able to address Dr van den Hogen's suggestion directly because we will be analysing interim data from the "Ox-check" trial. This is a randomised controlled trial of the effectiveness of health checks in reducing cardiovascular risk, and completion of a questionnaire, which includes the specific risk factors mentioned by Dr van den Hogen, is a precondition of recruitment to the study and the offer of a health check.

M AGASS L ROE
D WALLER J MUIR
D MANT A COULTER

Radcliffe Infirmary,
Oxford OX2 6HE

Meningococcal meningitis

SIR,—The editorial by Mr Philip D Welsby and Dr Clayton L Golledge on meningococcal meningitis[1] merits further comment on indications for hospital admission.

In Israel recently experience has been accumulating regarding patients with a genetic predisposition to meningococcal infection due to a deficiency of the terminal complement components (C5-C9). A recent review of 111 survivors of sporadic meningococcal disease in 10 Israeli hospitals found an incidence of C7 and C8 deficiency of 18% in patients of Sephardic origin (and an incidence of 40% in patients of Moroccan origin), and cases among Arabs have also been identified.[2] The clinical characteristics of the illness were different in this group, including a higher age at first presentation and a high incidence of recurrence (40%), and a high proportion of the patients had a family history of meningococcal infection.[2] Some patients may present with fever and no relevant clinical signs (Malnick and others, unpublished observations), as may also occur in children with meningoccocal infection.[3] Inherited deficiencies of the complement system that are associated with increased susceptibility to meningococcal infections have also been reported in white and black people[4] and may be more prevalent than currently realised. One American study of 20 consecutive patients presenting with a first known episode of meningococcal meningitis disclosed three cases of "idiopathic" terminal pathway protein deficiency.[5]

Thus a history of meningococcal infection or a positive family history should be added to the criteria for hospital admission suggested by Mr Welsby and Dr Golledge.

S D H MALNICK
A SCHATTNER

Division of Internal Medicine,
Kaplan Hospital,
Rehovot and Hadassah Medical School,
Jerusalem,
Israel

1 Welsby PD, Golledge CL. Meningococcal meningitis. *Br Med J* 1990;**300**:1150-1. (5 May.)
2 Zimran A, Rudensky B, Kramer MR, *et al*. Hereditary complement deficiency in survivors of meningococcal disease: high prevalence of C7/C8 deficiency in Sephardic (Moroccan) Jews. *Q J Med* 1987;**240**:349-58.
3 Dashefsky B, Teek DW, Klein JO. Unsuspected meningococcemia. *J Pediatr* 1983;**103**:69-73.
4 Ross SC, Densen P. Complement deficiency states and infection: epidemiology, pathogenesis and consequences of neisserial and other infections in an immune deficiency. *Medicine* 1984;**863**:243-73.
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Coronary artery bypass surgery

SIR,—In their editorial Mr E W J Cameron and Mr W S Walker draw attention to the important issue of selection of the conduit in coronary artery bypass surgery.[1] Graft failure is more common with saphenous vein conduits than with internal mammary artery grafts. Consequently we were