article by Jenny Simpson tempts questions about how to develop beyond Clinical Directorates.

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Navigating the NHS: core issues for clinicians, P Lees, ed. pp ix+150, illustrated. Radcliffe Medical Press, Oxon, 1996. £15.00, paperback.

My first pessimistic thought on being asked to review this book was that it sounds thick and uninteresting. It is neither. Firstly, it is concise, being both brief and informative, and secondly it is written by people 'doing the job' and largely avoids academic discourse. Thirdly, it is a comprehensive introduction to the major issues impacting upon the NHS.

Arranged in a series of mercifully short chapters, it deals admirably with longer standing issues, eg, case mix, coding and contracting and introduces the new and some would say black arts of marketing and being a Clinical Director!

If there is a criticism to be levied it is that a short book of 150 pages can only begin to explore how the issues interact and often conflict in an increasingly busy and some-

times overstretched NHS, although Tim Scott's final chapter highlights some of this dissonance. Some things need to be experienced to be properly savoured.

Although *Navigating the NHS* is presented as briefing notes for hard pressed medical trainees, it will be a very useful introduction for anyone new to the NHS, be they clinicians, managers, non-executive Directors, or lay readers, and will be of particular interest to senior registrars going for their first Consultant interview!

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The house officer

The hands-on guide for house officers, A Donald, M Stein, eds. pp xviii+236, illustrated. Blackwell Science, Oxford, 1996. £12.95, paperback.

It is amazing how much you can forget in that short time between the end of finals and the beginning of house jobs. As you are handed your bleep it becomes a real struggle to remember the numerous causes of finger clubbing or the characteristics of Von Hippel-Lindau syndrome and you wonder if you'll survive. This

book reassures you that these things aren't what being a house officer is about. Its concise and relevant information is easily accessible and the range of topics is wide enough to enable you to cope with most problems, from anaemia through to skin rashes.

The chapters on practical procedures and approaches are particularly useful. They accurately explain *how* different procedures and investigations are performed. The practicalities of medicine are often glossed over in undergraduate teaching, so unless you've seen, for example, pleural fluid being aspirated or a tuberculin test being performed as a student then encountering these things for the first time can be daunting.

The book has some welcome additions, like diagrams of anatomical systems to help explain procedures to patients, and chapters on self-care and coping with death and dying. The layout of the book is user friendly and being pocket-sized it can be easily carried around. The use of colour would no doubt have enhanced its appeal but it manages well without it. Overall, this is an excellent book for those starting out on the wards and the authors are to be congratulated for recognising the important topics.

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Letters to the Editor

Post-prandial syncope due to nitrates in food

Sir,

A case is reported of a 58-year-old man who suffered a syncopal episode after eating some sausages. The high nitrate content of these may have been responsible, causing hypotension via production of nitric oxide. Although nitrate-induced methaemoglobinaemia is rare, this potentially fatal condition should also be suspected.

At a physician's summer party, an otherwise healthy 58-year-old man consumed 10 sausages over a 20-minute period. He had no significant medical history and was taking no medication. Shortly afterwards, he fell to the ground without loss of consciousness and remained there for approximately 10 minutes during which time a doctor recorded his vital signs. The patient appeared flushed and had a blood pressure of 80/60 mmHg. He was alert and had no neurological deficit. Recovery was rapid and two minutes later his blood pressure was 125/60 mmHg. His progress was uneventful thereafter and no abnormalities were found at follow-up.

The preserving and colour-stabilising properties of nitrates and nitrites in cured meats dates back through the aeons of time to the saline deserts of Hither Asia. The upper legal limit of nitrate concentration in meat is 200 ppm. Each sausage eaten here had a nitrate concentration of 150 ppm and the 10 sausages consumed were calculated to be equivalent to 68 mg of ingested nitrate.¹ A review of the literature suggests that the patient may have been fortunate to avoid more serious complications. Nitrate is converted to nitrite in the gut and in turn can form methaemoglobin, displacing the oxygen

dissociation curve to the left. Symptoms are due to hypoxia and occur at concentrations of methaemoglobin above 10%.² Treatment includes oxygen, gastric lavage, intravenous methylene blue (a coenzyme for a reductase involved in methaemoglobin breakdown) and, in very serious cases, exchange transfusion. Although nitrate-induced methaemoglobinaemia is rare, it should be considered in cyanosed patients in the absence of other cardiac or respiratory problems.

Work by Cederqvist *et al*³ has demonstrated the *in vivo* formation of nitric oxide from organic nitrates and nitrites with a significant correlation between nitric oxide formation and hypotension. The authors concluded that nitrates exert their effects via nitric oxide production. The affects in man of ingested nitrate do require further investigation⁴ but *N*-methylnicotinamide, a metabolite excreted in the urine, may provide a marker for exposure.⁵

While the aetiologic role of the sausages in the syncopal episode described cannot be firmly established, it is possible that the high nitrate-nitrite level in the meat resulted in a nitric-oxide-induced hypotensive episode causing the syncope. This is distinct from reports of acquired methaemoglobinaemia causing cardiopulmonary catastrophe.

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Hyperhomocysteinaemia

Sir,

It was with great interest that I read the article by van den Berg and Boers¹ on hyperhomocysteinaemia. Although the title was somewhat provocative, it was appropriate and also timely. An association between hyperhomocysteinaemia and premature coronary artery disease was first reported two decades ago,² but homocysteine has emerged as the new player in the field of coronary risk factor only recently.^{3,4}

The causes of hyperhomocysteinaemia are multifactorial and the authors gave a comprehensive review of the various risk factors. They noted that possible relationships between plasma homocysteine and conventional risk factors for vascular disease have been studied but no such relation was established for tobacco smoking. However, a group of investigators from Hong Kong⁵ have recently reported that cigarette smoking was a risk factor. They noted a strong correlation between hyperhomocysteinaemia, smoking and coronary artery disease. Furthermore, according to these authors, only smoking

was found to be an independent risk factor, suggesting that the association between hyperhomocysteinaemia and coronary artery disease was probably the result of a higher prevalence of smokers in the hyperhomocysteinaemic group in their population or an interaction between the two risk factors. Although the authors did not postulate the mechanism of cigarette smoking in causing hyperhomocysteinaemia, cessation of smoking should be included in the treatment plan for hyperhomocysteinaemia.

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Treatment of 'warfarin intolerance'

Sir,

Hypersensitivity to food substances may cause symptoms pertaining to the central nervous system (headaches, hyperactivity, 'mental fuzziness', etc) or musculoskeletal system (body aches and pains, arthralgia, etc),^{1,2} mimicking other pathological conditions such as migraine, psychiatric illnesses, chronic fatigue syndrome, rheumatism, etc. A variety of food substances, including maize protein and lactose, are used in the formulations of drugs as bulking agents. Hence, hypersensitivity to these food components may cause 'drug intolerance' that may clinically present as nonspecific neurological, musculoskeletal or gastrointestinal disorders. Maize protein is used as a bulking agent in several widely used drugs, including warfarin, prednisolone, atenolol, isosorbide dinitrate, ibuprofen and piroxicam. Therefore, symptoms due to maize allergy may be incorrectly attributed to the drug and may even result in the withdrawal of the drug from clinical use in these cases.

Sodium cromoglycate has been used to treat various food allergies^{3–5} with beneficial effects in some patients. We report a case of 'warfarin intolerance' in a patient with apparent maize protein allergy successfully treated with sodium cromoglycate.

A 59-year-old man, presented to the Anticoagulant Clinic in November 1995, having commenced warfarin for atrial fibrillation. He had a history of ischaemic heart disease requiring surgical treatment with a coronary

artery bypass graft earlier in the year. Since starting anticoagulant therapy he had experienced generalised muscular aches and joint pains. He had apparently suffered from a 45-year history of allergy to maize-containing foods which provoked symptoms identical to his current complaints. However, no precipitating antibodies to maize were detected on countercurrent electrophoresis (dilutions of maize from 1:4 to 1:8000 were run against patient's undiluted serum). Total serum IgE was 134 ku/l. Test for specific IgE (Elisa technique) to maize was negative but that for specific IgG (Pharmacia Cap technique) to maize gave a borderline result. In view of the troublesome nature of his symptoms, warfarin was discontinued and he was commenced on enoxaparin 20 mg subcutaneously once a day. His symptoms resolved within a few days. A week later, warfarin was re-introduced with concurrent oral sodium cromoglycate (200 mg bid) treatment. The patient has remained asymptomatic over a follow-up period of six months and is currently on maintenance dose warfarin and sodium cromoglycate.

Since food substances are widely used in drug formulations, hypersensitivity to a food substance may develop 'drug intolerance' and may preclude the use of the drug. Clinicians should be aware of these possibilities and the true nature of the 'intolerance/adverse reaction' should be investigated and treated appropriately. Furthermore, the response of this patient to sodium cromoglycate clearly indicates that such 'intolerance' may be amenable to simple treatment enabling the continued use of the drug.

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Presentation of *Plasmodium vivax* malaria

Sir,

Malaria can present in unusual forms. *Plasmodium falciparum* malaria has been known to be associated with urticarial reaction,^{1,2} but *P. vivax* malaria presenting with urticaria is a rare association.^{3,4} We report here a case where *P. vivax* presented as recurrent anaphylaxis leading to syncope.

A 44-year-old man presented to the emergency room with complaints of generalised itching and flushing, and mild puffiness of the

face for which he had been to see his general practitioner. While waiting for this medication, he felt dizzy and collapsed. Being recumbent, he regained consciousness and was prescribed antihistamines. He gave a history of feeling feverish a day before. The patient was observed for 24 hours and then discharged. The blood smear was negative for malarial parasite. Two days later, he again presented to clinic with fever up to 103°F and chills. This time his blood smear was positive for *P. vivax* so he was treated with chloroquine.

One year later, he again presented to the emergency room with generalised itching and flushing of one-hour duration, and while entering the emergency room, fainted. On examination, his pulse was 52 beats/min regular, blood pressure 60/40 mmHg, and temperature 36.6°C. He was treated with antihistamines and hydrocortisone. He was fine after 24 hours and was discharged. Three days later, he again presented with fever and chills, generalised erythematous rash and itching. His temperature was 38.4°C, pulse 74 beats/min and blood pressure 100/50 mmHg. His blood smear was positive for *P. vivax* and he was again treated with chloroquine. Later, he was put on weekly chloroquine prophylaxis for a year and has not had any recurrence.

The repeated episodes of anaphylaxis and urticarial rash indicate that these were related to malaria infection. Orthostatic hypotension has been reported as a prominent clinical feature of malaria⁵ and has been attributed to a combination of relative bradycardia and peripheral vasodilatation. This to our knowledge is the first case of vivax malaria presenting as urticarial flushing leading to syncope, which on one occasion even preceded the development of fever. The exact mechanism of anaphylactic reaction in malaria is not clear; a detailed study is needed to identify the immune mechanism involved.

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