Postgrad Med J 2000;**76**:189–190 © The Fellowship of Postgraduate Medicine, 2000

Letters to the Editor

Hypokalaemic paralysis

Sir,

Ahlawat *et al*,[1] in their excellent review, fail to mention the presence of hypophosphataemia and mild hypomagnesaemia in thyrotoxic periodic paralysis (TPP). In a retrospective review of 24 episodes of TPP in 19 patients, Manoukian *et al*,[2] found hypokalaemia in all patients. In addition, a mild to moderate hypophosphataemia (serum phosphorus 0.36–0.77 mmol/l; normal levels 1.0–1.4 mmol/l), was noted in 12 of 15 episodes when serum phosphorus was measured within 1.5 h of hospital admission or while the potassium level was still low. Neutra-phos was administered in only four episodes; nevertheless, serum phosphorus returned to normal, or was slightly elevated after paralysis was resolved in all episodes evaluated. Hypophosphataemia during paralysis in patients with TPP has been previously reported[3] and may contribute to muscle weakness along with hypokaelemia. Phosphorus is a major intracellular anion, and its influx into and efflux from the cell most probably occurs in conjunction with potassium transport. Hypophosphataemia and hypokalaemia are well known causes of rhabdomyolysis, and the mild elevation of creatine kinase concentration seen by Manoukian *et al*[2] in 12 of 18 episodes may be related to the low serum phosphorus and potassium levels.

Mild hypomagnesaemia was noted[2] in all 19 patients during paralysis, with an increase of 0.1 mmol/l or more after resolution of paralysis. This has also been observed by other workers.[4] Endogenous catecholamines may contribute to hypomagnesaemia during stress without depleting total body stores of magnesium by causing an intracellular shift in magnesium. *Yeo et al*[5] found that four of 10 patients with TPP had an increase in skeletal muscle magnesium content during paralysis.

A S KASHYAP
Department of Medicine

SUREKHA KASHYAP
Department of Hospital Administration, Armed Forces Medical College, Pune 411040, India

1 Ahlawat SK, Sachdev A. Hypokalaemic paralysis. *Postgrad Med J* 1999;**75**:193–7.
2 Manoukian MA, Foote JA, Crapo LM. Clinical and metabolic features of thyrotoxic periodic paralysis in 24 episodes. *Arch Intern Med* 1999; **159**:601–6.
3 Norris KC, Levine B, Ganesan K. Case report: thyrotoxic periodic paralysis associated with hypokalemia and hypophosphatemia. *Am J Kidney Dis* 1996;**28**:272–3.
4 Saeian K, Heckerling PS. Thyrotoxic periodic paralysis in a Hispanic man. *Arch Intern Med* 1988;**148**:708.
5 Yeo PPB, O'Neill WC. Thyrotoxicosis and periodic paralysis. *Med Grand Rounds* 1984;**3**:10–25.

This letter was shown to the authors who responded as follows:

Sir,

In response to the comments of Dr Kashyap *et al* on our article,[1] I would like to point out that although hypophosphataemia and hypomagnesaemia have been reported in patients with thyrotoxic periodic paralysis (TPP), this reporting has been inconsistent. On the other hand, hypokalaemia is considered to be the most consistent electrolyte abnormality in TPP and a hallmark of the syndrome, along with hyperthyroidism. The retrospective review by Manoukian *et al*[2] is the only study in which hypophosphataemia and hypomagnesaemia were seen in a large number of cases. This was a hospital-based study and out of 30 patients seen, only 19 could be included because metabolic data were not available for the remaining patients. There are only a few other case reports which mention these electrolyte abnormalities (hypophosphataemia, hypomagnesaemia) in TPP.[3–5] Because of inconsistent observations and the lack of prospective data, their therapeutic implication remains unclear.

SUSHIL K AHLAWAT
89-50 56th Ave, #2J, Elmhurst, NY11373, USA

1 Ahlawat SK, Sachdev A. Hypokalaemic paralysis. *Postgrad Med J* 1999;**75**:193–7.
2 Manoukian MA, Foote JA, Crapo LM. Clinical and metabolic features of thyrotoxic periodic paralysis in 24 episodes. *Arch Intern Med* 1999; **159**:601–6.
3 Norris KC, Levine B, Ganesan K. Case report: thyrotoxic periodic paralysis associated with hypokalemia and hypophosphatemia. *Am J Kidney Dis* 1996;**28**:272–3.
4 Nora NA, Berns AS. Hypokalemic, hypophosphatemic thyrotoxic periodic paralysis. *Am J Kidney Dis* 1989;**31**:247–9.
5 Guthrie G, Curtis J. Hypophosphatemia in thyrotoxic periodic paralysis. *Arch Intern Med* 1978;**138**:1284–5.

Temporary cardiac pacing

Sir,

I read the recent article by Petch[1] with much interest. As a specialist registrar training in general medicine, having completed two and a half years of general medical on-call, I can safely say that I now feel competent in the procedure of temporary pacing. I fully recognise that as a senior house officer (SHO) or first-year registrar my previous experience in this technique was inadequate, despite having worked in a large District General Hospital as a 'cardiology SHO'. However, I cannot agree with your statement that this procedure should no longer be essential to the Calman training requirements in general medicine. Admittedly, central line insertion can be a messy business in inexperienced hands, but once mastered, it is a straightforward and uncomplicated procedure providing that the internal jugular vein is the chosen route of access. Routinely calling upon an anaesthetist or cardiologist for assistance is both impractical and unrealistic. Moreover, the prime concern of the doctor performing the procedure is usually the potential provocation of ventricular arrythmias rather than failure to gain venous access.

The current situation is obviously not ideal but where do we draw the line between specialist and general medical training? Temporary pacing is no more hazardous or difficult than intercostal drain insertion and is probably performed with equal frequency within a District General Hospital. Perhaps the Joint Committee on Higher Medical Training should be concentrating their efforts more on discriminating between procedures which fall wholly within the realm of a particular specialty than on integrating them within the general medicine 'additional procedure' requirements (Section 6, *Record of specialty training*). For example, a gastroenterology specialist registrar seeking dual accreditation, should no more have the ability to perform a bone marrow aspiration than a specialist registrar in haematology an endoscopy.

Finally, in those hospitals lacking on-site facilities for permanent pacing, emergency transfer to a specialist centre often means waiting more than 2–3 days and occasionally up to or over a week (I have first hand experience of this). In such situations one obviously needs to weigh up the benefits of temporary wire insertion with the inherent risks of infection and replacement. More often than not this responsibility lies with a consultant physician with an interest other than cardiology supervising a specialist registrar seeking accreditation in general medicine.

J K LOEHRY
Specialist Registrar in General Medicine and Gastroenterology, Royal Bournemouth Hospital, Bournemouth BH7 7DW, UK

1 Petch MC. Temporary cardiac pacing (editorial). *Postgrad Med J* 1999;**75**:577–8.

A total eclipse of the sun

Sir,

Historically, a total solar eclipse has been regarded as a sign of divine discontent and a warning of imminent death and destruction. Fortunately, when on 11 August 1999 the solar eclipse was experienced by millions across the UK and elsewhere, the only major health sequel reported to date has been a handful of cases of solar retinopathy.[1] Specific environmental and climatic changes are well-recognised features during a solar eclipse and include a sharp fall in temperature, an 'eclipse gust front' despite a drop in overall wind speed, a rise in humidity and a variable change in atmospheric pressures.

We wish to report the case of a 45-year-old pregnant woman with late onset of brittle asthma who experienced an acute exacerbation of asthma during the eclipse. There had been no deterioration in the control of her asthma during the pregnancy, although she had been recently admitted with an early miscarriage of one foetus of a twin pregnancy. She gave a clear history of having experienced increased shortness of breath with wheeze at the time of the eclipse. This was associated with a fall in her peak flow readings and was managed with nebulised bronchodilators. The previously identified major precipitant of her asthma had been changes in environmental temperature.

Exacerbations of asthma have been reported in association with a variety of environmental changes including lightening strikes,[2] a high pollen count,[2 3] and environmental pollution.[4] An absolute fall in ambient temperature[3] and the velocity of temperature change[5] have also been described as precipitating an exacerbation of asthma. A rapid fall in temperature from 17.2 to 14.5° C and rise in humidity (60–88%) was recorded at the time of the eclipse (personal communication, meteorological office). The pollen count on that day was low. Although we acknowledge that psychological factors related to the eclipse may have played a part, we postulate that it was the rapid change in temperature that was the precipitant in this case. As far as we are aware this is the first case reported of eclipse-associated exacerbation of asthma. As a change in the ambient temperature is linked

to exacerbations of asthma, we would expect to hear of similar cases.

GAVIN D PERKINS
HARMESH MOUDGIL
Princess Royal Hospital, Telford TF6 6TF, UK

1 Dobson R. UK hospitals assess eye damage after solar eclipse. *BMJ* 1999;**319**:469.
2 Newson R, Strachan D, Archibald E, *et al*. Acute asthma epidemics, weather and pollen in England, 1987–1994. *Eur Respir J* 1998;**11**:694–701.
3 Celenza. Fothergill J, Kupek E, Shaw RJ. Thunderstorm associated asthma: a detailed analysis of environmental factors. *BMJ* 1996;**312**:604–7.
4 Damia D, Fabregas L, Tordera M, Torrero L. Effects of air pollution and weather conditions on asthma exacerbations. *Respiration* 1999;**66**: 52–8.
5 Silber JH. Forecasting asthmatic wheezing using temperature velocity. *Pediatr Emerg Care* 1987;**3**: 31–7.

Self-assessment questions

Sir,
I am concerned about some of the case presentations which have appeared in your self-assessment section recently, as they seem to me to exemplify a very old-fashioned attitude to differential diagnosis and one which, frankly, we should have abandoned many years ago. I refer specifically to the papers by Balcombe[1] and by Usalan and Özarslan.[2] In the first paper we are asked: "What is the cause of thrombocytopaenia?". The answer given on the next page begins by providing a box with a long list of potential causes of a low platelet count, most of which are irrelevant to the case in question. Surely the question that should be asked and discussed is: "What are the most likely causes of thrombocytopaenia in this patient?".

In the second case the differential diagnosis places adrenal crisis as the third potential cause of the patient's illness; given the clinical features described it should surely have been placed first, or at least second. The authors then report that they performed a rapid ACTH test before starting treatment. If a patient is as ill as this lady was and adrenal crisis is suspected there is no need to do a rapid ACTH test in the early phase of the illness: all that is required is to take a blood sample for later analysis for cortisol. Treatment should then be started immediately. Provocative ACTH testing can be done at a later date if necessary. Usalan and Özarslan go on to give a description of the potential causes of an adrenal crisis (box 2), but do not include the most likely cause in their patient, ie, that the patient had subacute or chronic, mild adrenal insufficiency which became critical as a result of her pregnancy and urinary infection.

It seems to me that both reports are examples of 'doing medicine by the book'. Over the years we have all met junior doctors whose book knowledge is impressive but who cannot apply that knowledge effectively in clinical practice, cannot prioritise and cannot act decisively and effectively when faced with a serious medical emergency. Should we not be laying much more emphasis on clinical problem solving in a 'real-world' fashion and less on the learning of long and often irrelevant lists?

ROGER A FISKEN
Consultant Physician, Friarage Hospital, Northallerton, North Yorks, UK

1 Balcombe NR. The chilling tale of a patient with thrombocytopaenia. *Postgrad Med J* 1999;**75**: 621–3.
2 Usalan C, Özarslan E. Hypotension and intractable vomiting in the first trimester of pregnancy. *Postgrad Med J* 1999;**75**:623–5.

Response from the Editor:

The Editor and Editorial Board have shared similar anxieties. For this reason we plan to develop a more 'commissioned' approach to self-assessment questions. In future, authors will be requested to only submit cases with a clear educational value, and to discuss the background to the case more fully. It is hoped that such cases will slowly replace the current type of self-assessment questions, and that they may also open up a forum for other clinicians to give a commentary on the case.

J F MAYBERRY
Editor

Phaeochromocytomas in VHL disease

Sir,
Varghese *et al*,[1] reported an interesting case of von Hippel-Lindau (VHL) disease in a patient who also had left adrenal tumour. This is likely to be a phaeochromocytoma.[2] The authors had excluded phaeochromocytoma, as urinary normetadrenaline secretion was normal. However, plasma normetanephrine and metanephrine estimation has been found to be a more sensitive and specific test for detection of phaeochromocytomas in VHL disease and multiple endocrine neoplasia type 2 (MEN-2), compared to plasma concentrations of catecholamines (norepinephrine and epinephrine), urinary excretion of norepinephrine, epinephrine, metanephrines (normetanephrine and metanephrine combined), and vanillylmandelic acid.[3] Eisenhofer *et al*[3] found that in 35 patients with histologically confirmed phaeochromocytomas (26 patients with VHL and nine patients with MEN-2), sensitivity of measurements of plasma normetanephrine and metanephrine for the diagnosis of phaeochromocytoma was 97%, a sensitivity significantly higher than that of 65% for urinary metanephrines ($p<0.001$). This high sensitivity was accompanied by high specificity (96%), while urinary metanephrines had a specificity of 95%. In all patients with VHL disease or MEN-2 who also had phaeochromocytomas, the plasma concentrations of normetanephrine were increased by an average of 348% above the upper reference limit, compared to 55% for urinary metanephrines ($p<0.001$). This high sensitivity for plasma normetanephrine and metanephrine has also been reported for sporadic phaeochromocytomas.[4]

The large amount of catechol-O-methyltransferase in chromaffin cells is the reason for the high sensitivity of plasma normetanephrine and metanephrine assay in detecting phaeochromocytomas. The membrane-bound enzyme has much higher affinity for catecholamines than does the soluble enzyme present in other tissues; thus, the adrenal gland constitutes the single largest source of normetanephrine and metanephrine in plasma.[5]

Inadequate sensitivity of plasma and urinary catecholamines or their metabolites is a problem in periodic screening for phaeochromocytomas, particularly in patients with VHL disease. In these patients, small suspicious masses may be identified by imaging studies, but phaeochromocytomas may not secrete catecholamines in sufficient amounts to cause an abnormal result on a biochemical test. This should be overcome by the superior sensitivity of plasma normetanephrine measurement.

A S KASHYAP
Department of Medicine

SUREKHA KASHYAP
Department of Hospital Administration
Armed Forces Medical College, Pune 411040, India

1 Varghese B, Stephens WP. A new diabetic patient with an abdominal mass. *Postgrad Med J* 1999;**75**:249–50.
2 Aprill BS, Drake AJ III, Lasseter DH, Shakir KM. Silent adrenal nodules in von Hippel-Lindau disease suggest pheochromocytoma. *Ann Intern Med* 1994;**120**:485–7.
3 Eisenhofer G, Lenders JWM, Linehan WM, *et al*. Plasma normetanephrine and metanephrine for detecting pheochromocytoma in von Hippel-Lindau disease and multiple endocrine neoplasia type 2. *N Engl J Med* 1999;**340**:1872–9.
4 Lenders JW, Keiser HR, Goldstein DS, *et al*. Plasma metanephrines in the diagnosis of pheochromocytoma. *Ann Intern Med* 1995;**123**:101–9.
5 Eisenhofer G, Rundquist B, Aneman A, *et al*. Regional release and removal of catecholamines and extraneuronal metabolism to metanephrines. *J Clin Endocrinol Metab* 1995;**80**:3009–17.

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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

A colour handbook of dermatology, RJG Rycroft, SJ Robertson. pp 220, illus. Manson Publishing Ltd, 1999. £29.95, paperback. ISBN 1-874545-25-1 ****

This almost pocket-sized soft-cover book is a real treasure. The 480 colour photographs are of excellent quality throughout. The accompanying text is concise and very helpful. It deals with topics under a limited number of headings, ie, definition and clinical features, epidemiology, differential diagnosis, investigations, and special points; no attempt is made to cover treatment.

In the introduction the various terms used in the description of skin lesions are not only explained in words, but are clearly illustrated with diagrams.

With such a well produced informative book it seems almost churlish to point out that on p 10 a vesicle is defined as being 'a blister less than 5 mm in diameter', whereas on p 22 pompholyx is defined as having vesicles up to 10 mm in diameter. Also, on p 112 there is an unusual spelling of 'Gardner's syndrome'.

This books is bound to appeal to a wide range of people, including medical students, dermatologists, general physicians and general practitioners.

C N A MATHEWS
Consultant Dermatologist, Royal Gwent Hospital, Newport, South Wales NP20 2UB, UK

Sigmundoscopy. Medical-psychiatric consultation-liaison. The bases, David J Robinson. pp 220. Rapid Psychler Press, 1999. $21.95, paperback. ISBN 0-9680324-5-1 ***

The cover of this book shows a colour cartoon of a white-coated cigar-smoking Freud 'look-alike', auscultating his own heart. The associated title led me to think that I might be reviewing an anthology of psychiatric humour! I was wrong. The blurb on the back cover advised: "do not let the title, 'Sigmundoscopy' fool you". I had been fooled and thereafter found it hard to be charitable! The author's Foreword states that the book was written "to provide readers with background information on many aspects of C-L Psychiatry, as well as an in-depth review of the consultation process". The foreword ends with the meaningless injunction to 'Keep Psychling!' and the author turns out to also be the publisher under the imprint of Rapid Psychler Press.

Contrary to appearances, this is a serious book, for the most part well-written, well-set-out, readable and clearly referenced. The Author's aim is to use humour to "enhance the educational aspects being presented ....." Cartoons appear intermittently through the text; they add nothing to it, they are distracting and intrusive, rather than complementary. The humour is of a quality which I had assumed most doctors had left behind in medical school!

I found myself irritated by the use of the words 'consult' and 'consultation' which were used interchangeably, although clearly meant the same thing. The statement that counter-transference is the therapist's conscious emotional reaction to the patient would find little support among psychotherapists. These are minor criticisms in the context of the general impact of the book, which left me in a state of profound cognitive dissonance.

I suggest that this book should be rapidly 'repsychled'! The cartoons and the forced mnemonics should be removed and the book needs a title which bears some relation to its contents. I think we might then be left with a useful little handbook on the development and practice of Consultation-Liaison Psychiatry from a trans-Atlantic perspective. It is unlikely to find a market in the UK.

RICHARD SCORER
Consultant Psychiatrist, Landough Hospital, Penarth, South Glamorgan CF64 2XX, UK

Guidelines in clinical practice, Allen Hutchinson, Richard Baker, eds. pp 207. Radcliffe Medical Press, 1999. £18.95, paperback. ISBN 1-85775-088-8. ****

Variations in practice, difficulty in keeping up with the medical literature and the slow introduction of new ideas into clinical practice are some of the factors that have led to the development of guidelines over the last decade. A veritable industry of guideline production has appeared, with many hours spent in creating them and trying to put them into practice. Many of them, however, have ended up in drawers or on shelves and have had little impact on the practice of clinical medicine.

This valuable book tells us how we could do better. Several of the authors remind us that the production of guidelines is complex, time-consuming and expensive. It is therefore unlikely that local groups have the ability to create high quality guidelines and they should use their time more effectively by making minor changes to national guidelines so that they are appropriate for local use. Two chapters in this book are particularly useful as they provide details culled from practical examples in the field. Other chapters include descriptions of how to implement guidelines and urge a managed strategy which should include mechanisms for monitoring how effective the guidelines are in practice. This is a timely and instructive book as NICE is starting to produce national guidelines which we must hope will abide by the advice given here. The book should be read by all those who are going to be involved in the production of national guidelines and who will be responsible for moulding and implementing them locally.

PETER WILKINSON
Consultant Cardiologist, Ashford Hospital, Ashford, Middlesex TW15 3AA, UK

International Postgraduate Diary

Royal Society of Medicine conferences
1/2 March 2000: Men's health: the 'working' years
9 March 2000: Bench to bedside: colorectal cancer
20 March 2000: Key advances in atopic eczema
23 March 2000: Medical education and the new media
30/31 March 2000: Pain: nature and management in man and animals
13–15 April 2000: Marathon medicine
17 May 2000: The role of cloning in healthcare
25/26 May 2000: Healthcare workers: their health risks and how to minimise them
23 June 2000: The model emergency service provision for London
24 June 2000: Medicine and Shakespeare
29 June 2000: Bench to bedside: coronary artery disease
13/14 July 2000: Management of GU infections in women
Details: Rosamund Snow, External Relations Department, The Royal Society of Medicine, 1 Wimpole St, London W1M 8AE, UK. Tel +44 (0)20 7290 2904; fax +44 (0)20 7290 2992; email: rosamund.snow@roysocmed.ac.uk

University of Warwick Short Courses
17–20 July 2000: Techniques and applications of molecular biology: a course for medical practitioners
27/28 September 2000: An introduction to immunology
Details: Dr Charlotte West, Department of Biological Sciences, University of Warwick, Coventry, CV4 7AL, UK. Tel +44 (0)247652 3540; fax +44 (0)247652 3701; email: Charlotte.West@warwick.ac.uk

Third International Symposium on Angiotensin II Antagonism
28 February—2 March 2000: London UK
Details: Hampton Medical Conferences Ltd, 127 High Street, Teddington, Middlesex TW11 8HH, UK. Tel + 44 (0) 181 977 0011; fax + 44 (0) 181 977 0055; email: hmc@hamptonmedical.com

Cardiovascular Disease Prevention V
4–7 April 2000: London UK
Details: Hampton Medical Conferences Ltd, 127 High Street, Teddington, Middlesex TW11 8HH, UK. Tel + 44 (0) 181 977 0011; fax + 44 (0) 181 977 0055; email: hmc@hamptonmedical.com

23rd European Conference on Psychosomatic Research
17–21 June 2000: Oslo, Norway
Details: Congress-Conference AS - CONGREX, Thomas Heftyes gt. 2, PO Box 2694 Solli, N-0204 Oslo, Norway. Tel + 47 (0)2256 1930; fax + 47 (0)2256 0541; email: ecpr2000@congrex.no

Falk Symposia
4–6 May 2000: Hepatology 2000 (Munich, Germany)
9/10 June 2000: Cholestasis and gallstones (Cluj Napoca, Romania)
1/2 October 2000: Non-neoplastic diseases of the anorectum—an interdisciplinary approach (Freiburg, Germany)
3/4 October 2000: Immunosuppression in inflammatory bowel diseases—standards, news, and future trends (Freiburg, Germany)
12/13 October 2000: Biology of bile acids in health and disease (Den Haag, The Netherlands)
4 November 2000: Chronic inflammatory bowel diseases—progress and controversies at the turn of the century (Bucharest, Romania)
Details: Falk Foundation eV—Congress Division, Leinenweberstr 5, PO Box 6529, D-79041 Freiburg, Germany. Tel +49 (0)761 130340; fax +49 (0)761 1303459; email: symposia@falkfoundation.de

Columbia University College of Physicians and Surgeons, New York
26 April 2000: 15th Annual schizophrenia conference
5/6 May 2000: 12th Annual orthopaedic trauma course. Current techniques in upper & lower extremity trauma
22–25 May 2000: 4th Annual conference. Botanical medicine in modern clinical practice
28–31 July 2000: 10th Annual corse. A comprehensive review of movement disorders for the clinical practitioner
30 July–5 August 2000: 5th Annual course. Update and intensive review in internal medicine
Details: Center for Continuing Education, Columbia University College of Physicians and Surgeons, 630 West 168th Street, Unit 39, New York, NY10032, USA. Tel + 1 212 781 5990; fax + 1 212 781 6047; email: cme@columbia.edu

Barrow Neurological Institute, Phoenix, AZ, USA
2–4 March 2000: 26th Annual symposium: recent advances in neurosurgery
5–7 March 2000: 26th Annual symposium: What's important? What's new? Neurology/neuroimaging
Details: Neuroscience Conference Coordinator, Barrow Neurological Institute, 350 West Thomas Road, Phoenix, AZ 85013, USA. Tel +1 602 406 3067; fax +1 602 406 4104; email: deskildson@theBNI.com

University of California, San Francisco
6/7 April 2000: 8th Symposium on clinical trials: design, methods and controversies
13–15 April 2000: 33rd Annual Advances and controversies in clinical pediatrics
Details: University of California, Office of Continuing Medical Education, 1855 Folsom St, MCB Room 630, San Francisco, CA 94143-0742, USA. Tel +1 415 476 4251; fax +1 415 476 0318; email: inquire@ocme.ucsf.edu

Ninth International Symposium on celiac disease
10–13 August 2000: Hunt Valley, MD, USA
Details: Althea Pusateri, Program Coordinator, University of Maryland School of Medicine, 655 W Baltimore Street, Baltomore, MD 21201, USA Tel +1 410 706 3957; fax +1 410 706 3103; http://www.celiaccenter.org