

# PostScript

## LETTERS

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### Cyclooxygenase 2 selective inhibitor induced bowel stricture: a case report

Several reports have mentioned the role of non-steroidal anti-inflammatory drugs (NSAID) in inducing diaphragm disease and strictures in the small and large bowel.<sup>1-3</sup> To our knowledge, there is no such report in patients treated with cyclooxygenase 2 (COX-2) selective inhibitors.

We report the case of a 55 year old man with a past history of axial spondylarthropathy, successfully treated with NSAID from 1975 to 2001; from February 2001, he was treated with celecoxib 400 mg per day for three weeks and then 200 mg/day for two years. He had previous abdominal surgery (appendectomy) in 1965.

He presented with a 24 hour history of central abdominal pain with persistent vomiting. Clinical and radiological examination confirmed small bowel obstruction. At laparoscopy, a distal ileal obstruction was identified. Coelioscopic laparotomy was then performed, showing evidence of bowel wall stricture; 10 cm of the distal ileum was spared. Macroscopic and microscopic examination of the resected specimen was consistent with a diagnosis of stricture on submucosal ulceration of the small bowel.

This condition is known to be associated with long term use of NSAID. The COX-2 specific inhibitors have been developed in order to improve the gastrointestinal safety of therapy with NSAID. In various clinical trials, COX-2 selective inhibitors have been shown to have similar efficacy to NSAID, with a concomitant association with fewer endoscopic ulcers and serious lower gastrointestinal events.<sup>3</sup>

This case suggests that COX-2 selective inhibitors can induce bowel wall ulcerations, followed by submucosa fibrosis, which may cause strictures or diaphragm-like disease.

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### Should we screen adults with osteoporotic fractures for coeliac disease?

In the recently published debate in *Gut* regarding the utility of mass screening of European and North American populations for coeliac disease (CD), divergent conclusions were presented (*Gut* 2003;52:168-9 and 170-1). In this context, the increased utility of screening adults for CD in those presenting with concomitant morbidity (for example, metabolic bone disease and fracture) was raised. To support such an hypothesis, evidence of either an increased fracture rate in those with CD or, alternatively, an increased incidence of CD in those presenting with fracture would be required.

Thomason and colleagues,<sup>1</sup> in a study of 244 patients with CD and 161 age and sex matched controls, addressed the first of these possibilities. They found that patients with CD "as a whole do not represent a population of particularly high risk of osteoporotic fracture".

Available data regarding the prevalence of CD in older people with osteoporosis are limited and controversial. Several reports<sup>2-3</sup> have suggested an increased prevalence of CD among patients with idiopathic osteoporosis leading the authors to recommend screening for CD of all osteoporotic patients. However, these findings are not supported by other studies.<sup>4-5</sup> In a study aimed at determining the prevalence of previously undetected secondary contributors to osteoporosis in otherwise healthy older women (mean age 65.5 years), the incidence of CD was 1.7%.<sup>6</sup>

Consequently, it seems important to know whether in older adults screening for CD in those presenting with osteoporotic fractures would yield a significant number of unsuspected cases. Osteoporotic hip fracture, a dramatic consequence of osteoporosis and a leading cause of morbidity and mortality in older people, has been reported in association with clinically silent CD.<sup>7-8</sup> However, to our knowledge, serological screening tests for CD have not been systematically studied in older adults with hip fracture.

We screened the serum of 347 consecutive older patients (>60 years of age) with hip fracture (74% females; age range 60-101 years, mean age 81.5 (SD 7.3) years) for the

presence of IgA endomysial antibodies (EMA), IgA and IgG gliadin antibodies (IgA-AGA and IgG-AGA), and total IgA. In 13% of patients, the IgA-AGA test was positive (above 34 ELISA units) while in 11% of patients the titre of IgG-AGA was slightly elevated (above 46 ELISA units). However, none of the patients had a positive anti-EMA test which is known to have a high specificity (98-100%).<sup>9</sup> This negative finding is particularly noteworthy given that 86% of the screened population had a low body weight (<60 kg), 79.1% had low serum 25-hydroxyvitamin D concentrations (<50 nmol/l), 69% had secondary hyperparathyroidism (serum PTH >5.5 pmol/l), and 21.6% had anaemia (haemoglobin <110 g/l). Such abnormalities are often associated with CD and are believed to contribute to the development of osteoporosis in CD. Therefore, one might expect that investigation of a cohort of older adults with osteoporosis presenting with a hip fracture might yield a moderate number of people with subclinical CD. However, this was not the case in this analysis. Our findings indicate that CD appears not to be an important contributing pathogenic factor in an older hip fracture population with osteoporosis. It further suggests that routine screening for CD in a similar population, or even in those individuals with a hip fracture and accompanying hypovitaminosis D and/or secondary hyperparathyroidism, would have a low yield and not be cost effective.

Despite these findings, we would continue to encourage physicians evaluating older adults to consider, but not routinely screen for, CD when unexplained metabolic bone disease presents even in the absence of gastrointestinal complaints and/or dermatitis herpetiformis.

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## Mesalazine is safe for the treatment of IBD

The article by Ransford and Langman (*Gut* 2002;**51**:536–9) on suspected serious adverse drug reactions for sulphasalazine and mesalazine reported in the UK from 1991 to 1998 revealed significant differences between both drugs.

Pancreatitis and interstitial nephritis were reported more frequently for mesalazine in comparison with sulphasalazine. The authors' conclusion that mesalazine would not offer a safety benefit over sulphasalazine however appears unjustified for several reasons.

Sulphasalazine is an older compound used for the treatment of both rheumatoid arthritis and inflammatory bowel disease (IBD). For 30 years, the adverse event (AE) profile of sulphasalazine has been well known.<sup>1</sup> It often induces oligoteratozoospermia in male patients and frequently causes nausea, vomiting, headache, and folic acid deficiency. Although not "serious" AEs, these often lead to low compliance, incorrect use, and early discontinuation. In addition, it is more than likely that the many adverse reactions, identified in the 1970s, were not reported again to the medical authorities in the 1990s. The introduction of mesalazine in the 1980s enabled effective treatment (often at higher doses) without the numerous adverse effects attributed to the sulphapyridine moiety of sulphasalazine.<sup>2</sup> Focus on the risk of interstitial nephritis caused by mesalazine preparations in the mid 1990s undoubtedly led to a low threshold for reporting. However, the incidence of renal insufficiency was recently studied in a large cohort of 1449 European IBD patients (more than 70% on mesalazine/sulphasalazine) and did not exceed the expected incidence in the general population.<sup>3</sup>

Furthermore, pooling the data of all pure mesalazine products (*Gut* 2002;**51**:536–9) does not seem appropriate as the different release mechanisms of the various products could bring about different AE profiles. Pentasa has less frequently been associated with interstitial nephritis than other 5-ASAs.<sup>4</sup>

Unlike Pentasa, Asacol, Claversal, and Salofalk indeed have a relative dose dumping effect with higher peak serum concentrations, allegedly contributing to potential nephrotoxicity.<sup>5</sup>

In addition, reporting serious AEs in relation to the number of prescriptions is an unusual approach. Dosage and duration of therapy would have been more relevant as the risk of side effects is dose dependent with sulphasalazine but not with mesalazine. Physicians may prefer to prescribe mesalazine to patients who are susceptible to side effects caused by sulphasalazine. For some reason, adverse events with a fatal outcome were not mentioned separately in Ransford and

Langman's report (*Gut* 2002;**51**:536–9). Based on the British CSM database, 18 fatal events occurred in patients taking sulphasalazine versus 12 in the pooled pure mesalazine group during the same observation period. Moreover, the mortality rate for Pentasa was zero in an earlier French pharmacovigilance report, revealing an incidence of reported adverse events with this product (the most commonly used mesalazine preparation in France with a market share >70%) of 6–9 per million days of therapy.<sup>6</sup>

In conclusion, based on all the available data on mortality, serious irreversible adverse events, and tolerability of both drugs, mesalazine should be preferred to sulphasalazine in the treatment of IBD. Eighty per cent of patients intolerant to sulphasalazine will tolerate mesalazine without problems.<sup>7–9</sup>

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## Diet and colorectal cancer: fibre back on the menu?

The Romans believed that illnesses stemmed from the "heart" but the Greeks from the gut. Were the Greeks right after all? Dietary influence on colorectal cancer (CRC) has been extensively studied but with very little agreed consensus. Pederson *et al* (*Gut* 2003;**52**:861–7) reawaken this idea by showing a causal relationship between alcohol and rectal cancer while Dray *et al* (*Gut* 2003;**52**:868–73) conclude that prolonged survival post curative surgery for CRC was

associated with high energy intake. What about fibre?

Burkitt was the first to attribute CRC to fibre deficiency<sup>1</sup> which was later challenged by Cleave as excess sugar or "the saccharine disease".<sup>2</sup> A wealth of epidemiological and interventional studies have presented conflicting views. Particularly damning were a clutch of papers in 2000 showing little or no benefit of fibre on adenoma formation.<sup>3,4</sup> Two recent papers<sup>5,6</sup> add significantly to the debate. Both have shown a protective role of fibre on distal colonic adenomas and on colorectal cancer, respectively. Hence the question arises, why the difference?

The earlier interventional studies<sup>3,4</sup> showing no benefit were of a much smaller size in a single population and had a shorter duration of follow up. Adenoma recurrence was used as an end point, presumably thereby skewing the data towards a population with as yet phenotypically silent premalignant mutations. This has implications as the time frame from exposure of nutritional factors which influence critical steps in the molecular and cellular development of CRC is quite long. Furthermore, the total quantity of fibre (g/day) consumed was low and the types of fibre studied were different. Non-starch polysaccharides (NSP) are fermented by gut microflora to produce three main short chain fatty acids (butyric acid, acetic acid, and propionic acid). These compounds have a range of properties and functions according to their "fermentability"—non-fermentable fibres have poor antitumour potential in *in vivo* models.<sup>7</sup> In contrast, poorly fermented fibres afford protection by yielding fermentation products along the entire length of the colon. Therefore, how can we put fibre confidently back on the menu?

What is needed is a large varied population study correlating molecular/cellular markers and CRC with dietary fibre. In addition, a distinction must be made between colon and rectal cancer. Apart from having different embryological derivations, right sided colonic cancer and left sided colonic cancer (distal to splenic flexure) exhibit differences in incidence according to geographic region, age, and sex.<sup>8</sup> Secondly, the problem, as in previous works, is studying NSP as a homogenous group. Butyric acid is the main short chain fatty acid (SCFA) produced in millimolar quantities in the colonic lumen. It has a number of functions in the colon: (i) as a fuel source for colonocytes; (ii) a survival factor for healthy cells; (iii) a stimulator of proliferation; and (iv) it suppresses carcinogenesis in a rat model. Butyrate therefore has a multifactorial role in the determination of bowel health. Examining specific SCFAs in stool or biomarkers of their utilisation therein, is likely to provide more consistent observations.

Finally, the study of dietary fibre using colorectal adenomas as an end point in interventional studies is questionable. This is based on the assumption that adenomas are an adequate surrogate marker for colorectal cancer. In light of studies showing highly different ratios of adenoma and carcinoma formation between populations,<sup>9,10</sup> implying distinct aetiology and triggering events, this is a particularly unsafe assumption. The EPIC study has justified renewal of interest in the protective role of fibre in the colon. More carefully designed intervention studies may put it back on the menu.

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**Obesity as a risk factor for colorectal polyps in Japanese patients**

Colorectal cancer (CRC) is one of the most common malignant diseases in developed countries. Recent epidemiological studies<sup>1</sup> suggest that CRC is associated with obesity (*Gut* 2002;**51**:191–4).

Although primary prevention of CRC via dietary measures is controversial, secondary prevention by interrupting the adenoma-carcinoma sequence is possible. One cross sectional study<sup>2</sup> and a case control study<sup>3</sup> have demonstrated the association between obesity and colorectal adenomas in men<sup>2</sup> and women,<sup>3</sup> respectively, whereas another study

failed to show any association.<sup>4</sup> Although cross sectional studies in Japan have demonstrated an association between obesity and colorectal adenomas, all subjects were males and total colonoscopy was performed in only some subjects.<sup>5</sup> Colonoscopy is proven to be superior to double contrast barium enema for detection of adenomatous lesions as well as early CRC. We therefore aimed to examine the association between obesity and colorectal polyp by total colonoscopy.

A cross sectional study was conducted on a total of 541 consecutive adult subjects (361 males and 180 females) who attended the University Hospital outpatient clinic with gastrointestinal problems and underwent total colonoscopy, from December 2000 to December 2001. Patients with CRC, colonic obstruction, known inflammatory bowel disease, and a past history of gastrointestinal surgery were excluded. All colonoscopies were performed by experienced endoscopists. Body height and weight were measured, and body mass index (BMI) was calculated as weight (kg) per height (m<sup>2</sup>). In the present study, we defined BMI  $\geq 25.0$  kg/m<sup>2</sup> as “obese” and others as “non-obese”. Standard statistical methods were used and the results were given as mean (SEM). The significance of the difference between the two groups was examined using the  $\chi^2$  test. Differences with  $p < 0.05$  were considered significant. Table 1 shows the characteristics of the obese and non-obese groups.

Age, sex, and body height were not significantly different between the two groups while body weight and BMI were significantly higher in the obese group. Colorectal polyps were found in 57 of 112 obese patients (50.9%) but in only 124 of 429 non-obese patients (28.9%) ( $p < 0.001$ ). Biopsy or polypectomy was performed for all polyps, which were diagnosed as adenomatous polyps by histopathological examination. In common with previous studies,<sup>2,3,5</sup> we have clearly demonstrated an association between obesity and colorectal adenomas in Japanese adult patients, performing total colonoscopy in all subjects. In contrast, an inverse relationship between obesity and colorectal adenomas was reported in Western countries.<sup>4</sup> However, risk factors for colorectal polyps are not clear. This discrepancy may be due to racial and/or lifestyle differences. As this was a cross sectional study at a single university hospital, prospective multicentre case control studies are needed to demonstrate a close association between obesity and colorectal polyps in the Japanese.

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**BOOK REVIEWS****Colonic Diseases**

Edited by T R Koch. Totowa New Jersey: Humana Press, 2003, pp 553. ISBN 0-89603-961-7

Colorectal diseases are common, and interest in coloproctology continues to increase among surgeons, physicians, and scientists. A recent attendance at a surgical coloproctology meeting confirmed the very multidisciplinary nature of the specialty. The focus on coloproctology justified the publication of a well organised book edited by Dr Timothy Koch. The book has a distinguished panel of North American authors.

Part I deals with basic science in 11 excellent chapters and the book is worth having simply to marvel at the progress in our understanding of colonic physiology and immunology over the past decade. Colonic motility and its neural regulation, as well as colorectal sensation, are covered in depth in three chapters. The integration of colonic function is vividly described and understanding is then translated to relevance in continence and defecation. The exciting possibilities of pharmacological interventions to modulate colonic function are entering an era reminiscent of the understanding of vascular control that led to a plethora of

**Table 1** Characteristics of the obese and non-obese subjects

	Obese			Non-obese			p Value*
	Male	Female	Total	Male	Female	Total	
n	65	47	112	296	133	429	NS
Age (y)	57.6 (10.2)	62.3 (9.6)	59.6 (10.1)	60.1 (13.8)	59.1 (15.9)	59.8 (14.5)	NS
Body height (cm)	165.1 (14.9)	150.2 (5.6)	158.7 (8.2)	164.4 (6.9)	152.1 (6.9)	160.6 (8.9)	NS
Body weight (kg)	72.9 (6.3)	64.4 (6.2)	69.3 (6.3)	59.4 (7.8)	9.3 (6.8)	56.3 (8.8)	<0.01
Body mass index (kg/m <sup>2</sup> )	26.8 (1.2)	26.8 (1.2)	27.5 (1.7)	21.9 (2.4)	21.3 (2.3)	21.3 (2.3)	<0.01
Colon polyps (n)	36	21	57	69	55	124	<0.001

Data are mean (SEM).

\*Comparison between total obese and total non-obese subjects.

agonists and antagonists. Excellent chapters on mucin and goblet cell function, aging, micronutrients, and colonic endocrine cells follow, and these chapters integrate knowledge in an authoritative manner in areas not often appreciated by those not directly involved in relevant active research. The chapter on probiotics is more translational but nevertheless comprehensive. Even those with deep subspecialty interest in colorectal problems will come away with new information after reading this section of the book.

Part II covers investigations relevant to colonic diseases. Some of the chapters in this section probably are more relevant as research methodology tools, such as inflammation, oxidative stress, and epidemiological/outcome research. The chapter on inflammation could have contained some references to imaging in inflammatory diseases, especially with radionuclides, in order to justify sitting comfortably in this part of the book. The rest are more clinically inclined and comprehensively cover the entire spectrum of investigations in colonic diseases, including colonic physiology and function, radiology, colonoscopy, and histology.

Part III details specific diseases in a further 11 chapters. This is certainly not a book to have for its coverage of colon cancer, and given the importance of this disease, more information on the basic science of colorectal neoplasia as well as clinical aspects could have been provided, preferably in additional chapters. A number of more unusual conditions are not covered, such as pneumatosis cystoides intestinalis and melanosis coli, and there is little on colonic vascular disorders, including angiodysplasia. Radiation colopathy is mentioned only in the section on colonoscopy. Microscopic colitis and infectious colitis surely deserved full chapters, rather than passing mentions. The vast majority of references are from year 2000 or before.

The colour plates are superb, but lack of a full caption prevents their enjoyment in isolation without referring to the text. Overall, this is a superb volume with a wealth of information, especially in basic science and translational aspects. I would recommend this book to anyone interested in colorectal diseases, but perhaps not to those interested in colorectal cancer alone. All gastroenterologists, most colorectal surgeons, and some colorectal nurse specialists would benefit from having access to this book, which is compact enough to slip into a briefcase.

S Ghosh

### Inflammatory Bowel Disease: Diagnosis and Therapeutics

Edited by R D Cohen. Totowa, New Jersey: Humana Press, 2003, pp 364. ISBN 0-89603-909-9. E-ISBN 1-59259-311-9

*"Knowledge is of two kinds. We know a subject ourselves, or we know where we can find information on it", Samuel Johnson (1709–1784).*

In this era of IT explosion, a concise source of information is always welcome. Inflammatory bowel disease (IBD) can now boast of several large reference books with international contributors. Smaller texts tend to bring either a personal dimension, often entertaining, or a focus on controversial

areas. The rather concise 350 page book edited by Dr Cohen however attempts to provide an overview of IBD within 17 chapters set out in a fairly conventional manner. It covers areas well beyond diagnosis and therapeutics. The contributors are all North American and the book therefore attempts to cater to a predominantly North American readership. When I received this lightweight but hardbound edition I immediately decided to put it to the "transatlantic flight test"—that is, whether the book would entertain and inform during a return flight to the USA, in between the inflight entertainment and an occasional champagne.

The book is organised in a conventional manner with historical facts, epidemiology, aetiopathogenesis, and genetics followed by presentation and diagnostics, medical and surgical management, paediatric issues, ostomy care, nutritional and extraintestinal manifestations, and cancer. Pathological features, for an obscure reason, come in as the last chapter. Two chapters towards the end cover very important gender issues and economic aspects. The chapter on economic aspects gives a wealth of information on medical care costs in IBD and almost anyone involved in caring for IBD patients will benefit from reading it. It is an ideal chapter on which to base a few slides whenever one is called upon to deliver a talk on some aspect of the management of IBD, which these days is incomplete unless a few economic issues are also addressed. Gender specific issues deals mainly with fertility and pregnancy in IBD patients, but does not address the issue of female fertility after ileal-pouch anal anastomosis, an increasingly important issue when counselling young women prior to a colectomy. In a book focusing on therapeutics, covering the entire medical management of both Crohn's disease and ulcerative colitis within 22 pages was clearly challenging, but the chapter does provide some interesting management algorithms. Probiotics are referred to very briefly in the first chapter. The chapter on surgical management is longer and well illustrated. Indeed some of the illustrations can well be adopted for the purpose of counselling patients. The chapter on stoma care has a disproportionately short list of references compared with the other chapters, which are all well referenced. Radiological findings and imaging attract a whole chapter but not endoscopic findings, which is given a passing reference in the chapter on pathological features.

The main weakness of the book is that it tries to have something for everybody; but this is probably also its main strength. If I wanted a volume that I could give to my new research fellow about to start a career in research into IBD I could use this book as an introduction to IBD for at least the next three years. Best of all, I could give this book to such a research fellow, irrespective of whether he is a clinician, nurse, or a basic scientist. Patients, their friends, and families will however find most of the chapters rather dense and technical, even by North American "expert patient" standard and I do not think that it seriously tried to fulfil the role of educating patients, despite the claim in the preface. The book as a whole has a number of useful illustrations although some chapters are completely free of illustrations and hence somewhat less attractive in layout.

Overall, the book passed its "transatlantic flight test" and I felt I had read a robust overview of IBD. I will have my copy ready to

pass on to my oncoming research fellows, but I will also continue to keep my larger reference books for more detailed information on specific therapeutic aspects.

S Ghosh

## NOTICES

### British Society of Gastroenterology Paul Brown Travel Fellowships

The Paul Brown Travel Fellowships are awarded by the Endoscopy Committee of the BSG. They are intended to assist trainee gastroenterologists and established consultants in visits to units outside the United Kingdom for specialist experience and training in endoscopy.

Specialist registrars who have not achieved their CCST are expected to have the approval of their Postgraduate Dean and their Regional Training Director when they apply for a Travel Fellowship. Applicants are expected to provide confirmation that they have been accepted for training in the unit that they wish to visit.

Successful applicants will be expected to provide a brief written report to the Endoscopy Committee of the outcome of their visit.

Application forms are available from the British Society of Gastroenterology Office, 3 St Andrew's Place, London NW1 4LB. Email: bsg@mailbox.ulcc.ac.uk

### Hong Kong-Shanghai International Liver Congress 2004

This conference will be held on 14–17 February 2004 in Hong Kong. The topic of the conference is "Liver Diseases in the Post-Genomic Era". Further details: Ms Kristie Leung, Room 102–105 School of General Nursing, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong. Tel: +852 2818 4300/8101 2442; fax: +852 2818 4030; email: kristieleung@hepa2004.org; website: www.hepa2004.org

### PET/CT and SPECT/CT Imaging in Medical, Radiation, Surgical and Nuclear Oncology

This continuing medical education programme will take place on 19–20 March 2004 at Johns Hopkins University School of Medicine, Baltimore, Maryland, USA. Further details: Office of Continuing Medical Education, Johns Hopkins University School of Medicine, Turner 20, 720 Rutland Avenue, Baltimore, Maryland 21205-2195. Tel: +1 410 955 2959; fax: +1 410 955 0807; email: cmenet@jhmi.edu; website: www.hopkinscme.org

### 39<sup>th</sup> Annual Meeting of the European Association for the Study of the Liver

This meeting will be held on 15–19 April 2004 in Berlin, Germany. Further details: Secretariat, c/o Kenes International, 17 rue du Cendrier, PO Box 1726, CH-1211 Geneva, Switzerland. Tel: +41 22 908 0488; fax: +41 22 732 2850; email: info@easl.ch; website: www.easl.ch/easl2004

- Deadline for receipt of abstracts: 16 November 2003

- Deadline for early registration 10 February 2004

### Second Sheffield Multi-Disciplinary Colorectal Meeting

There will be a multi-disciplinary symposium for surgeons, physicians, radiologists and specialist nurses on 9 January 2004. The faculty includes: Wendy Atkin —St Mark's (London), Professor Jonathan Rhodes — University of Liverpool, Professor John

Scholefield —Nottingham, Dr S Taylor—St Mark's Hospital, Mr Andrew Shorthouse—Sheffield, Dr Stewart Riley—Sheffield, and Karen Smith—Nurse Endoscopist at Sheffield. The Second Sheffield Multi-Disciplinary Colorectal Meeting takes place between 10am and 5pm at the Postgraduate Centre, Northern General Hospital, Sheffield. The registration fee is £25. For further details, please contact: Anne Smedley, Secretary to Mr AJ Shorthouse, Royal Hallamshire Hospital, Glossop Road, Sheffield, S19 2JF.

### 14<sup>th</sup> International Workshop of Digestive Endoscopy, Ultrasonography and Radiology

The 14<sup>th</sup> International Workshop of Digestive Endoscopy, Ultrasonography and Radiology will be held in Marseille on 27—28 May 2004. For further information, please contact: Nathalie Fontant, Atelier Phenix, 41 rue Docteur Morruci, 13006 — Marseille (tel: (33) 04-91-37-50-83; fax: (33) 04-91-57-15-28; e-mail: nfontant@aphenix.com).

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