At the same time, Williams and his co-authors advocate ERCP only in severe, but not mild, acute pancreatitis, justifying this by the fact that the majority of stones pass spontaneously in the subgroup of patients with mild pancreatitis. I fully agree with that argument, but I believe that the same may also be applied to patients with severe ABP. It seems that a recent RCT from the USA,[1] comprised of patients with both mild and severe acute pancreatitis, supports this point of view, as it has revealed that in 53% and 71% of patients, respectively, the obstruction cleared spontaneously within 24 and 48 h after onset of symptoms. More importantly, that trial demonstrated significantly better clinical outcomes in patients in whom the obstruction cleared (spontaneously or by means of sphincterotomy) within 48 h of pain onset in comparison with those in whom the obstruction cleared beyond 48 h (spontaneously or by means of sphincterotomy). Collectively, the presented findings suggest that the duration of obstruction rather than the severity of acute pancreatitis may play a crucial role in the exacerbation of this disease.

Summarising the above, the use of early ERCP with sphincterotomy may be useless and, given a definitive percentage of procedure-related complications, even harmful in unselected patients with severe ABP. Only patients with co-existing acute cholangitis and/or prolonged common bile duct obstruction may benefit from early endoscopic intervention.

M S Petrov

Correspondence to: Dr Maxim Petrov, PO Box 568 Nizhny Novgorod 603000, Russia; max.petrov@gmail.com

Competing interests: None.

REFERENCES

1. **Acosta JM,** Katkhouda N, Debian KA, *et al*. Early ductal decompression versus conservative management for gallstone pancreatitis with ampullary obstruction: a prospective randomized clinical trial. *Ann Surg* 2006;**243**:33–40.
2. **Oría A,** Cimmino D, Ocampo C, *et al*. Early endoscopic intervention versus early conservative management in patients with acute gallstone pancreatitis and biliopancreatic obstruction: a randomized clinical trial. *Ann Surg* 2007;**245**:10–7.
3. **Petrov MS,** van Santvoort HC, Besselink MG, *et al*. Early endoscopic retrograde cholangiopancreatography versus conservative management in acute biliary pancreatitis without cholangitis: a meta-analysis of randomized trials. *Ann Surg* 2008;**247**:250–7.
4. Working Party of the British Society of Gastroenterology; Association of Surgeons of Great Britain and Ireland; Pancreatic Society of Great Britain and Ireland; Association of Upper GI Surgeons of Great Britain and Ireland. UK guidelines for the management of acute pancreatitis. *Gut* 2005;**54**(Suppl 3):iii1–9.
5. **Mofidi R,** Madhavan KK, Garden OJ, *et al*. An audit of the management of patients with acute pancreatitis against national standards of practice. *Br J Surg* 2007;**94**:844–8.
6. **Fölsch UR,** Nitsche R, Lüdtke R, *et al*. Early ERCP and papillotomy compared with conservative treatment for acute biliary pancreatitis. *N Engl J Med* 1997;**336**:37–42.
7. **Neoptolemos JP,** Carr-Locke DL, London NJ, *et al*. Controlled trial of urgent endoscopic retrograde cholangiopancreatography and endoscopic sphincterotomy versus conservative treatment for acute pancreatitis due to gallstones. *Lancet* 1988;**2**:979–983.

Authors' reply

We are grateful to Dr Petrov for his interest in the the guidelines on management of common bile duct stones. We agree that the evidence base in this area has been static for some time, and readers will understand that consensus guidelines can take a considerable time in their genesis and publication. It is unfortunate that we were unable, therefore, to consider very new and up-to-the-minute developments in this particular area prior to publication.

The historic evidence on early management of gallstone pancreatitis was limited and has generated considerable controversy over the years. We accept that there probably were confounding variables in some of the historic data, particularly when comparing the details and differing designs of the studies. The recent data cited by Dr Petrov will undoubtedly contribute to the debate. We agree in particular that patients with cholangits and pancreatitis are a particular subgroup within previously published data sets and that these are the patients most likely to benefit from early intervention. This is an area that needs further study in order to inform future practice and guidelines.

E J Williams, J Green, I Beckingham, R Parks, D Martin, M Lombard

Audit Steering Group, Department of Gastroenterology, Royal Liverpool University Hospital, Liverpool, UK

Correspondence to: Dr M Lombard, Audit Steering Group, Department of Gastroenterology, 5z Link, Royal Liverpool University Hospital, Prescot Street, Liverpool L7 8XP, UK; martin.lombard@rlbuht.nhs.uk

Competing interests: None.

Somatostatin analogues reduce liver volume in polycystic liver disease

Polycystic livers occur in the setting of two inherited conditions: (1) autosomal dominant polycystic kidney disease (ADPKD), also characterised by progressive development of renal cysts resulting in loss of renal function, and (2) polycystic liver disease (PCLD), with a polycystic liver as the sole manifestation. Symptoms, such as abdominal distension, result from hepatomegaly. Ascites is a rare complication and might be due to portal hypertension or caused by a ruptured cyst.

Treatment strategies for polycystic livers are aimed at reducing liver volume. So far, only surgical options are available, but these are associated by considerable morbidity and mortality.[1] Alternatively, ascites in the setting of polycystic livers might respond to endovascular stent placement in the narrowed caval vein,[2] but medical options are conspicuously lacking so far. We wish to report two cases with severe polycystic liver disease that were successfully treated with somatostatin analogues.

A 53-year-old woman with ADPKD complicated by a polycystic liver presented with abdominal pain and increased abdominal distension. Her renal function was normal. On physical examination she had hepatomegaly. An abdominal CT scan demonstrated ascites, a polycystic liver and a ruptured liver cyst. She was treated with diuretics (spironolactone 25–50 mg/day and furosemide 40 mg/day), but ascites progressed with concomitant increase of abdominal distension and onset of orthopnoea. Drainage of ascites (7 litres, serum ascites albumin gradient of 23.8 g/l) yielded only temporary relief. As no other therapeutic options were available, octreotide (50 μg/h intravenously) was started 9 months after the onset of ascites. Within days, the patient improved and we switched to octreotide 100 μg subcutaneously (three times a day). After 1 month, her abdominal distension had disappeared and diuretics could be stopped. Three months after start of octreotide treatment, the patient was free of abdominal pain. 3D-volumetry of the polycystic liver was performed using Pinnacle[3] (version 8.0d, Philips, Eindhoven, The Netherlands) according to previously published methods.[3] A CT scan 112 days after the start of octreotide demonstrated a decrease of liver volume from 4609 ml to 2843 ml (38.3%) and of kidney volume (left, from 246 ml to 240 ml (2.4%); right, from 554 ml to 455 ml (17.9%)) (fig 1).

The second patient, a 43-year-old woman with PCLD, developed ascites and oedema due to inferior caval vein syndrome, 11 days after laparoscopic fenestration of liver cysts. Her weight had increased from 79 to 94 kg. In

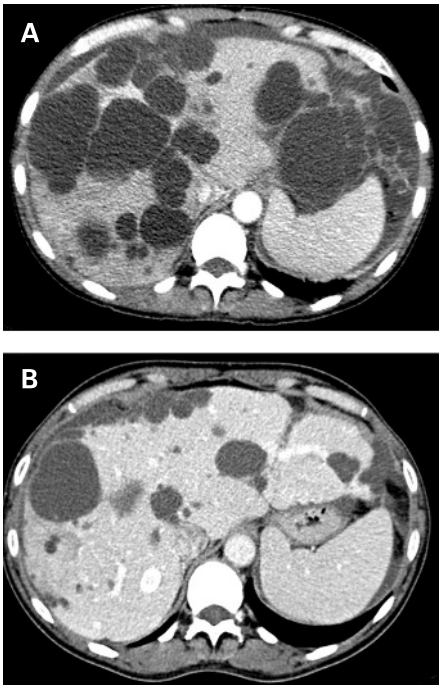

Figure 1 Somatostatin analogues decrease cyst volume in polycystic liver diseases. CT scan prior to (A) and after treatment (B). Volumetry showed a decrease of liver volume from 4609 ml to 2843 ml.

order to relieve the caval pressure, three cysts were aspirated and 14.5 litres of ascites was drained, without much benefit. One month after surgery, we started her on octreotide (100 μg three times a day subcutaneously) and 12 days later we switched her to lanreotide (120 mg once a month subcutaneously). 3D-volumetry of the polycystic liver 230 days after start of treatment demonstrated a volume decrease of 14.9% (from 8232 ml to 7004 ml). Renal function remained unchanged over this time period (creatinine previously 42 μmol/l, and after 46 μmol/l).

Both patients developed intractable ascites secondary to polycystic liver disease. While liver transplantation was considered, we decided to start somatostatin analogues first. In both patients, this resulted in a dramatic clinical improvement, disappearance of ascites and a decrease of liver volume.

Cyst growth is modulated by cAMP, which stimulates cholangiocyte proliferation and cyst fluid secretion, a process which is inhibited by somatostatin.[4] In an animal model for polycystic liver disease, octreotide reduced hepatic cyst volume in a dose-dependent manner.[5] In ADPKD, octreotide inhibited renal cyst growth by ~55%, but hepatic cyst volume was not evaluated in this trial.[6]

How does this effect compare with other therapeutic modalities for polycystic livers? We evaluated aspiration sclerotherapy and laparoscopic fenestration for this indication and found that these achieve a volume reduction of 19.2% and 12.5%, respectively.[3 7]

Although somatostatin reduces the hepatic venous pressure gradient, this alone would be insufficient to ameliorate the intractable ascites.[8] Therefore, we surmise that the therapeutic effect of octreotide results from direct reduction of hepatic cyst volume. In addition, this report implies that cystic fluid accumulation is a dynamic process which can be reversed by somatostatin analogues.

L van Keimpema,[1] R A de Man,[2] J P H Drenth[1]

[1] Department of Gastroenterology and Hepatology, Radboud University Nijmegen Medical Center, The Netherlands; [2] Department of Gastroenterology and Hepatology, Erasmus Medical Center, Rotterdam, The Netherlands

Correspondence to: Miss L van Keimpema, Department of Gastroenterology and Hepatology, Radboud University Nijmegen Medical Center, PO. Box 9101, 6500 HB Nijmegen, The Netherlands; L.vanKeimpema@mdl.umcn.nl

Competing interests: None.

Patient consent: Obtained.

Gut 2008;**57**:1338–1339. doi:10.1136/gut.2008.155721

REFERENCES

1. **Everson GT,** Taylor MR, Doctor RB. Polycystic disease of the liver. *Hepatology* 2004;**40**:774–82.
2. **Di Costanzo GG,** Ragozzino A, Niola R, *et al*. A rare cause of diuretic refractory ascites. *Gut* 2006;**55**:53, 73.
3. **van Keimpema L,** Ruurda JP, Ernst MF, *et al*. Laparoscopic fenestration of liver cysts in polycystic liver disease results in a median volume reduction of 12.5%. *J Gastrointest Surg* 2008;**12**:477–82.
4. **Alvaro D,** Gigliozzi A, Attili AF. Regulation and deregulation of cholangiocyte proliferation. *J Hepatol* 2000;**33**:333–40.
5. **Masyuk TV,** Masyuk AI, Torres VE, *et al*. Octreotide inhibits hepatic cystogenesis in a rodent model of polycystic liver disease by reducing cholangiocyte adenosine 3′,5′-cyclic monophosphate. *Gastroenterology* 2007;**132**:1104–16.
6. **Ruggenenti P,** Remuzzi A, Ondei P, *et al*. Safety and efficacy of long-acting somatostatin treatment in autosomal-dominant polycystic kidney disease. *Kidney Int* 2005;**68**:206–16.
7. **van Keimpema L,** de Koning DB, Strijk SP, *et al*. Aspiration-sclerotherapy results in effective control of liver volume in patients with liver cysts. *Dig Dis Sci* 2008; [Epub ahead of print].
8. **Kalambokis G,** Economou M, Kosta P, *et al*. The effects of treatment with octreotide, diuretics, or both on portal hemodynamics in nonazotemic cirrhotic patients with ascites. *J Clin Gastroenterol* 2006;**40**:342–6.

BOOK REVIEWS

GI epidemiology

Edited by N J Talley, G R Locke. Published by Blackwell, pp 288, £54.99 (hardcover). ISBN 9781405149495

GI epidemiology is the first book devoted to the subject since Michael Langman's slim but provocative volume *The epidemiology of chronic digestive disease* was published about 30 years ago. As the editors state in the preface, it has grown out of a postgraduate GI population sciences Masters module run at the Mayo Clinic, and almost half the 46 contributors are from there, with the remainder from North America, and a handful only from elsewhere. Not surprisingly, therefore, it has a strong US flavour both in content and in its use of epidemiological statistics, to the extent that the overview of the burden of GI disease in Chapter 2 is solely concerned with the US.

Just under half of the remaining chapters are devoted to methodologies in GI research and the rest to traditional disease specific epidemiology. Summarising methodologies such as health economics, and genetic epidemiology in five to six pages is a tough challenge but some such as those on questionnaires and decision analysis stand out for their clarity. Two chapters on careers in GI epidemiology and obtaining NIH funding I suspect will be of little interest outside the US.

Similarly, the chapters on individual diseases are a mixed bag. I particularly enjoyed those on gallstones and coeliac disease, but for me the outstanding contribution was Nyren's on *Helicobacter pylori*, peptic ulcer and gastric cancer which managed to be both authoritative and provocative in highlighting gaps in knowledge. Other GI cancers are covered only briefly and readers looking for reviews of their epidemiology would do better consulting a text on cancer epidemiology. There were also some remarkable omissions such as nothing on appendicitis or diverticular disease – listed in Chapter 2 as being responsible for 180 000 admissions and 3400 deaths in the US. Epidemiology lends itself to the use of maps, figures and tables and the lack of these in several chapters was frustrating.

Despite these criticisms I celebrate the publication of *GI epidemiology* as recognition that there is now a sufficient body of expertise and knowledge to identify the area as a distinct sub-speciality analogous to cardiovascular epidemiology and pharmacoepidemiology. While it is not, and was not, intended to be a heavyweight reference tome it will serve as a sound introduction for undergraduates and clinical trainees interested in gastrointestinal diseases.

Richard F A Logan

Correspondence to: Professor Richard F A Logan, Division of Epidemiology and Public Health Sciences, Queen's Medical Centre, Nottingham NG7 2UH, UK; richard.logan@nottingham.ac.uk

Intestinal failure. Diagnosis, management and transplantation

Edited by Langnas A, Goulet O, Quigley EMM, Tappenden KA. Blackwell Publishing, Massachusetts & Oxford, 2008, pp 390, £75.00 (Hard). ISBN 9781 4051 46371

In his complimentary foreword the intestinal failure guru Professor Stanley Dudrick describes this book as unique. While I agree with many of his positive comments, I must take him to task on this. Naturally all medical texts have a quality of uniqueness, but I think the general reader will assume uniqueness to mean that it is the only book in the field, or at least the only one of its type. This is not true given the existence of a book with the same title edited by Dr Jeremy Nightingale. This was published in California and in the UK so we cannot excuse Professor Dudrick on geographical grounds!

The two books cover similar ground, have a similar number of authors, are drawn from a similarly wide range of disciplines and are of very similar size. Admittedly one leans towards the east of the Atlantic and the other to the west (and the covers are different colours). By now you will have concluded that the reviewer must be biased as well as pedantic, and to some extent you would be right, as more of the authors in the Nightingale book are personal friends, and I wrote two of its chapters. However, I hope I can introduce some objectivity in my comments.

There is a great deal that is good here. The editors have selected wise authors, and have been creative in their choices of chapter titles and the overall coverage, which is beautifully illustrated. The result is relevant to those working with intestinal failure patients and those engaged in related research. I liked the simple definition(s) of intestinal failure offered, even if I was somewhat puzzled that this keystone upon which the whole book rests could be found only in Chapter 6! The chapter on complications of bariatric surgery will be especially useful to those who currently see these patients infrequently (ie, Europeans), but who have the sure expectation that there will be more to come. The editors have evidently instructed many of their authors