LETTERS TO THE EDITOR

June 24, 1990

Dear Editor:

Drs. Lohmuller, Pemberton, Dozois, Ilstrup, and Van Heerdon in their article "Pouchitis and Extraintestinal Manifestations of Inflammatory Bowel Disease After Ileal Pouch–Anal Anastomosis" discuss the relationship between pouchitis, extraintestinal manifestations, indeterminate colitis, and Crohn's disease.[1] They do not mention the autoimmune response mechanism and immunosupressive therapy that may be a common factor. Present[2] assumes a 5% to 10% failure with the present surgery and suggests a medical trial before extensive surgery. His indications for the use of immunosurpressive therapy include (1) failure to respond to steroids and sulfasalazine; (2) steroid toxicity and continuous steroids for Crohn's disease; (3) patients with proctosigmoiditis who have not responded to oral and topical therapy; and (4) patients with left-sided or universal disease who are continually active and have not had disease long enough to be at risk for carcinoma of the colon. He has found 6-MP and azathioprine to be effective in 60% to 70% of patients and toxicity infrequent and reversible and no definite increase in superinfections or neoplasms in long-term use.

Cyclosporin is under investigation and does seem to show promise for inflammatory bowel disease (IBD) as it does for organ transplant immunosupressive therapy.[3] According to Riskin et al.,[4] synthetic carbohydrates may represent an important new class of drugs for the treatment of inflamatory, autoimmune diseases. Effectiveness of these drugs should lend credence to the theory that the nature of IBD is autoimmune and add another dimension, besides surgery, to its treatment.

References

1. Lohmuller JL, Pemberton JH, Dozois RR, et al. Pouchitis and extraintestinal manifestations of inflammatory bowel disease after ileal pouch-anal anastomosis. Ann Surg 1990; 211:622–629.
2. Present DH. 6-mercaptopurine and other immunosuppressive agents in the treatment of Crohn's disease and ulcerative colitis. Gastroenterol Clin NA 1989; 18:57–71.
3. Lichtiger S, Present DH. Cyclosporin A in the treatment of severe, refractory ulcerative colitis. Gastroenterology 1989; 96:A301.
4. Riskin WG, Gillings DB, Scarlett JA. Amiprilose hydrochloride for rheumatoid arthritis. Am JI Int Med 1989; 111:455–464.

LAWRENCE BRASLOW, M.D.
Riverside, California

August 28, 1990

Dear Editor:

In response to Dr. Lawrence Braslow's comments regarding our article, we would point out that the purpose of our study was not to investigate the etiology of ulcerative colitis nor its medical management but rather to study the relationship we have seen between pouchitis and extraintestinal manifestations of ulcerative colitis after ileal pouch–anal anastomosis. Be that as it may, at the conclusion of our discussion we do, in fact, suggest that the pathophysiologic mechanisms involved in pouchitis may be similar to those involved in chronic ulcerative colitis. Of course it is highly speculative whether this is an autoimmune process or some other mechanism.

Although Dr. Braslow's comments are appreciated, we cannot speak to them because they involve a subject far broader than our study.

JOHN H. PEMBERTON, M.D.
JOSEPH L. LOHMULLER, M.D.
Rochester, Minnesota

September 10, 1990

Dear Editor:

I read with interest the paper by Dr. Klein and colleagues entitled "Current Management of the Budd-Chiari Syndrome" in the August 1990 issue of *Annals of Surgery*.

In their discussion they did not mention the nonsurgical treatment of Budd-Chiari syndrome. The authors listed several causes of hepatic venous outflow occlusion, including membranous obstruction of the suprahepatic inferior vena cava. The latter, which is more common in the Orient, is suited ideally for nonoperative treatment by percutaneous balloon dilation. My colleagues from China and I have now successfully treated six such patients, three of whom had previous surgical procedures. Improvement was dramatic and long lasting.

Therefore, in the current management of the Budd-Chiari syndrome, one form, specifically membranous obstruction of inferior vena cava, which can be accurately diagnosed by angiography, could and should be treated nonsurgically by percutaneous balloon dilation. The latter approach is an effective and safe alternative to surgery.

TSUNG O. CHENG, M.D.
Washington, D.C.

October 2, 1990

Dear Editor:

Obstruction of hepatic venous drainage can result from disease at several anatomic locations including (1) nonthrombotic veno-occlusive disease originating in the terminal hepatic venules, (2) thrombotic occlusion of the hepatic veins or suprahepatic vena cava, and (3) membranous obstruction of the suprahepatic inferior vena cava. It has been common practice to group this diverse array of disorders together as the Budd-Chiari syndrome. Much of the confusion and debate concerning the optimal treatment for these patients could be avoided if such practice was abandoned.

I would agree with Dr. Cheng that invasive radiologic techniques may be appropriate treatment for selected patients who develop isolated membranous obstruction of the suprahepatic vena cava, and the successful outcome of such strategy has been published previously.[1,2] Percutaneous laser-assisted angioplasty has also been used in similar cases.[3,4] Membranous vena caval obstruction, although a common etiology of the Budd-Chiari syndrome worldwide, is rare in the United States except in those areas with a large population of Oriental immigrants.[5] Our discussion concerning the surgical management of patients with the Budd-Chiari syndrome was directed toward patients with hepatic vein thrombosis that was either primary in nature or developed secondary to vena caval obstruction. Neither balloon angioplasty nor laser obliteration is likely to be safe or effective for such patients, who account for the majority of Budd-Chiari cases seen in this country. There may be, however, a role for invasive radiologic procedures in the postoperative management of these patients. In our experience both anastomotic and non-anastomotic venous stenoses have been managed successfully with a combination of percutaneous transluminal angioplasty and/or percutaneous transvenous stent placement.

ANDREW S. KLEIN
Baltimore, Maryland

References

1. Loya YS, Sharma S, Amrapurkar DN, Desai HG. Complete membranous obstruction of inferior vena cava: case treated by balloon dilation. Cathet Cardiovasc Diagn 1989; 17(3):164–167.
2. Sparano J, Chang J, Trasl S, Bonanno C. Treatment of the Budd-Chiari syndrome with percutaneous transluminal angioplasty. Am J Med 1987; 82:821–828.
3. Deckelbaum LI. Laser-assisted angioplasty of inferior vena caval obstructions: what's good for the artery is good for the vein. Hepatology 1989; Feb 9(2):338–339.
4. Furui S, Yamauchi T, Ohtono K, et al. Hepatic inferior vena cava obstructions: clinical results of treatment with percutaneous transluminal laser-assisted angioplasty. Radiology 1988; 166:673–677.
5. Rector WG, Yuhui X, Goldstein L, et al. Membranous obstruction of the inferior vena cava in the United States. Medicine 1985; 64(2):134–143.

August 9, 1990

Dear Editor:

It seems to me that Drs. Crowley and Seigler, the authors of "Late Recurrence of Malignant Melanoma" (August 1990), missed an excellent opportunity to make a contribution to the debate concerning the current concept of local excision of thin melanomas of that amount of tissue that can be closed primarily is sufficient in a 10-year follow-up study. They indicated in their article, as I read it, that tumors less than 1 mm in depth represented approximately 25% of their cases (this was illustrated in Table 2.) In Table 4 they indicated that local skin recurrence occurred in approximately 34% of their cases.

The additional factor that would have been helpful would be a correlation of those thin melanoma excisions with the local skin recurrences (if any) to determine if there was any relationship between the extent of the surgical excision and the recurrence of the tumors.

GEORGE F. BALE, M.D.
Memphis, Tennessee

October 14, 1990

Dear Editor:

Dr. Seigler and I thank Dr. Bale for his letter regarding our article "Late Recurrence of Malignant Melanoma."

Approximately 25% of patients with complete histologic records had melanomas measuring less than 1 mm. These thin melanomas included 6 patients with extremity primaries, 10 patients with trunk primaries, and 5 patients with head/neck primary lesions. Interestingly only three of these patients experienced local recurrence. Most of these patients (11 of 21) had recurrence in the regional nodes and a large percentage (7 of 21) at distant sites, including lung, bone, and gastointestinal tract. The small number of patients (three) with thin melanomas who experienced local recurrence make analysis of the influence of margins of excision extremely difficult.

As indicated in Table 4, there were 17 patients with cutaneous melanomas who had local recurrence. These included 7 patients with extremity primaries, 7 patients trunk primaries, and 3 with head/neck primary lesions. These 17 local relapses occurred in the total population of 155 patients with cutaneous melanomas, for a total of 11% (rather than 34%). The Breslow thickness in this group of patients ranged from 0.45 mm to 3.35 mm. Again, only three of these patients had melanomas measuring less than 1 mm.

The issue of recurrent disease and survival in patients with thin melanomas (less than 0.76 mm) was evaluated for a group of 681 patients seen at the Duke University Melanoma Clinic. I would refer Dr. Bale to this paper by Slingluff et al. in a previous issue of the *Annals of Surgery* (1988; 208:150–161). In this analysis two clinical risk factors (axial primary site and male sex) and two histologic risk factors (Clark's level IV and severe histologic regression) were associated with an increased incidence of recurrence. Local skin recurrence was seen in 5% of patients and margins of excision were evaluated for this group of patients. Of those patients who experienced local recurrence, patients with margins of excision less than 1 cm had recurrence no sooner than those with wider margins, suggesting that narrow margins of excision (less than 1 cm) did not play a role in the risk of subsequent recurrence.

NANCY J. CROWLEY, M.D.
Durham, North Carolina

March 9, 1990

Dear Editor:

We read with interest the article entitled "Is Preoperative Angiography Useful in Patients with Periampullary Tumors?" by Dooley and associates (Ann Surg 1990; 211:649–655).

The authors evaluated the role of preoperative visceral angiography as a staging test adjunctive to computed tomography scan in patients with periampullary tumors. Thirteen of twenty-eight patients with positive angiographic evidence of vascular involvement were not explored and 6 of the remaining 15 who were explored had successful resection of the tumor. This false-positive rate of angiography (6 of 28) is quite high and disconcerting. The conclusion that all 17 patients with vascular occlusion as shown on angiography were unresectable is erroneous because only four of these were confirmed to be unresectable on exploration. But even if one believes that all of the 11 patients with total vascular occlusion were unresectable, the advantage of identifying this group of patients in 11 of 90 patients (12%) is offset by unwanted laparotomy in 14 of 90 patients (15%) in the angiogram-normal but unresectable group.

The data regarding peritoneal and liver spread is not available in 11 of 13 patients who were not explored (two had liver secondaries). Fifteen of the remaining seventy-nine patients had evidence of liver and peritoneal spread, which would have been amenable to detection by laparoscopy, a safe, cost-effective and accurate method.[1]

The question mark posed at the end of the title is very valid, and it should have been followed by an emphatic 'No.'

S. S. SIKORA, M.S.
V. K. KAPOOR, M.S.
Luchnow, India