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 nonanastomotic venous stenoses have been managed successfully with a combination of percutaneous transluminal angioplasty and/or percutaneous transvenous stent placement.

> ANDREW S. KLEIN Baltimore, Maryland

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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 twentyeight 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 falsepositive 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

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October 30, 1990

Dear Editor:

We thank Drs. Sikora and Kapoor for their interest in our study. We started with 90 patients who on the basis of computed tomography (CT) scan appeared to have resectable periampullary tumors. Visceral angiography demonstrated vascular occlusion in 11 of these 90 patients. Major vascular occlusion secondary to tumor is widely accepted as indicative of unresectability in periampullary tumors. Angiography is considered the definitive test for demonstrating vascular occlusion. After exploring 4 of these 11 patients and confirming that they were unresectable, we thought it was inappropriate and unwarranted thereafter to continue to explore such patients with major vascular occlusions. Seventeen patients had vascular encasement without occlusion demonstrated on angiography. At the time of laparotomy, encasement was confirmed in 13 patients. In two of these patients, however, the involved portal vein was resected and reconstructed. The remaining four patients at surgery were found to have no major vessel encasement and were resected. Thus the false-positive rate for the angiographic findings of both encasement and occlusion was 4 of 28 patients, or 14%. Although this rate is high, it does not obviate the value of angiography in preoperative staging of patients with periampullary tumors. We think the benefits of angiography are as follows: (1) avoiding needless laparotomy in patients with vessel occlusion; (2) alerting the surgeon to the possible need for major vascular resection and reconstruction in those patients with encasement; and (3) providing useful anatomic information to the surgeon concerning the hepatic arterial anatomy.

Other means of staging, such as laparoscopy, have been evaluated for patients with periampullary tumors. Although laparoscopy can identify superficial liver metastases and peritoneal implants, it is our belief that laparoscopy adds little to our current routine of staging by CT and angiography. In our series of 90 patients, six had peritoneal implants that would have been detectable at laparoscopy. Three of these patients had resectability excluded on the basis of visceral angiography. In addition at the time of exploration seven patients had resectability excluded by unsuspected liver metastases. Only four of these patients had superficial liver metastases that would have been detected by laparoscopy. The other three were deep-seated lesions. Therefore only 7 of our 90 patients would have benefited from laparoscopy in their staging evaluation before laparotomy. Using CT scan and viseral angiography, our overall resectability rate was 68%. This is the highest rate reported, and we think it justifies our approach.

> WILLIAM C. DOOLEY, M.D. JOHN L. CAMERON, M.D. HENRY A. PITT, M.D. KEITH D. LILLEMOE, M.D. NANCY C. YUE, M.D. ANTHONY C. VENBRUX, M.D. Baltimore, Maryland

Dear Editor:

The paper by Dr. Souba et al.¹ published in *Annals of Surgery* is both interesting and valuable. The authors studied the effects

of severe infection on gut glutamine metabolism in laboratory rats and in hospitalized patients. This study suggests that the gastrointestinal response to severe infection clearly is different from the response to surgical stress.

Intestinal glutamine consumption is increased in surgical stress despite a decrease in the circulating glutamine concentration. In sepsis, on the other hand, a marked reduction in gut glutamine use occurred with normal or increased arterial glutamine levels. The uptake of circulating glutamine by the gut occurs almost exclusively in the mucosal cells, where the content of the enzyme glutaminase is very high. In this study gut glutamine extraction decreased in septic patients and in endotoxin-treated rats. The authors attribute this phenomenom to an important compromise of the integrity of the gastrointestinal mucosa barrier by repeated exposure to systemic endotoxins.

Our group studied enterocyte amino acid concentrations in rats and we observed no changes in glutamine levels in starved or stressed animals compared to control.² In septic patients, however, we found significantly high plasma levels of glutamine with respect to reference values.³ Freeysz et al.⁴ also observed high plasma values of glutamine in a similar clinical situation.

The septic patients in our study received total parenteral nutrition with only 10% branched-chain amino acids. Furthermore it is accepted that in sepsis muscle glutamine values are low. The fact that our results showed high plasma glutamine levels may be explained by the decreased gut glutamine extraction described by Souba et al.

> M. PLANAS, M.D. M. FARRIOL, M.D. S. SCHWARTZ, M.D. J. B. PADRO, M.D. Barcelona, Spain

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October 16, 1990

Dear Editor:

We thank Dr. Planas and colleagues for their interest in our recent paper "The Effects of Sepsis and Endotoxemia on Gut Glutamine Metabolism."¹ We appreciate the observation by this group that the intestinal metabolic response to severe infection clearly is different from the response to pure surgical stress.

We would like to emphasize that the relationship between the decreased ability of the bowel to use circulating glutamine during sepsis and endotoxemia and the apparent breakdown in the gut mucosal barrier is, at this time, only an association. However the relationship between gut metabolism, structure, and function appears to be important because several studies showed that supplemental glutamine can influence gut morphology and function.²⁻⁴