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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]