mild and does not produce shock or significant hypovolemia, as evidenced by the data given. It is specifically our point, furthermore, that there is impairment of microcirculatory blood flow by phenylephrine in this setting, and our data suggest both increased intravascular fluid loss producing hemoconcentration and increased tissue injury, which we presume to be resultant from impairment of the microcirculation. In addition, we have now accumulated considerable additional evidence, using both intravital microscopy to measure capillary flow and reflectance spectroscopy to measure tissue oxygenation, that blood flow increases in cerulein pancreatitis but that phenylephrine markedly reduces the perfusion of the pancreas at the microcirculatory level both in normal animals with normal hemodynamics and intravascular volume and in animals with cerulein pancreatitis.[1-3]

We do agree that it is important in the treatment of pancreatitis, as well as that of septic shock, to maintain the circulating blood volume. Although this is not protective in and of itself, certainly inadequate volume support most probably adds to the degree of injury and to the susceptibility to the adverse effects of vasocontrictors. Whether the conditions in septic shock, as addressed by Dr. Gregory, are relevant to those in pancreatitis is not the subject of this study, nor do Dr. Gregory's comments indicate any less need to beware of the possible adverse effects of vasoconstrictors in patients with pancreatitis.

References

1. Knoefel WT, Rattner DW, Waldner H, Warshaw AL. Direct visualization of pancreatic microcirculatory changes caused by phenylephrine and isoproterenol. Gastroenterology 1990; 98:A222.
2. Knoefel WT, Rattner DW, Kollias N, et al. Evaluation of pancreatic tissue perfusion by intravital microscopy and reflectance spectroscopy. Digestion 1990; 46:150.
3. Knoefel WT, Kollias N, Warshaw AL, et al. Pancreatic microperfusion in experimental pancreatitis of increasing severity. Digestion 1991; 49:31.

ANDREW L. WARSHAW, M.D.
DAVID W. RATTNER, M.D.
ERNST KLAR, M.D.
Boston, Massachusetts

September 24, 1991

Dear Editor:

I read with interest the recent article by Dr. Hennington et al., entitled "Acute Appendicitis Following Blunt Abdominal Trauma."[1] I would like to report of my recent experience of a case of acute appendicitis after blunt abdominal trauma, diagnosed by peritoneal lavage.

A 20-year-old motorcyclist was involved in a motor vehicle accident and was admitted to the intensive care unit after a closed head injury with decerebrate posturing. His abdomen initially was cleared with an abdominal computed tomography scan that showed no evidence of bleeding or obvious visceral injury. He was observed in the intensive care unit, and throughout his first day of hospitalization his abdomen was soft and he was afebrile. On the morning of the second day of hospitalization, the patient was still unresponsive because of his closed head injuries, but his abdomen was firm and he had a temperature of 100.6 F. A percutaneous diagnostic peritoneal lavage (DPL) was performed, and the lavage fluid was very cloudy and the cell count demonstrated 3250 white blood cells and 250 red blood cells per milliliter. The patient was taken to the operating room and the abdomen was explored, with the finding of acute appendicitis. Postoperatively, the patient did well. He regained bowel function shortly and was able to be started on tube feedings until his mental status improved sufficiently for him to take an oral diet.

The use of DPL in the management of peritonitis of nontraumatic origin has been investigated by several authors.[2-4] False-positive and false-negative DPL in acute appendicitis was reported. Positive DPL may occur in mesenteric adenitis,[2] and negative DPL has been reported in mild appendicitis and appendiceal abscess.[3] Clearly, clinical suspicion remains the hallmark of diagnosis in acute appendicitis.

References

1. Hennington MH, Tinsley EA, Proctor HJ, Baker CC. Acute appendicitis following blunt abdominal trauma: incidence or coincidence? Ann Surg 1991; 214(1):61-63.
2. Hoftman J. Peritoneal lavage as an aid in the diagnosis of acute peritonitis of non-traumatic origin. Dig Dis 1988; 6:185-193.
3. Barbee CL, Gilsdorf RB. Diagnostic peritoneal lavage in evaluating acute abdominal pain. Ann Surg 1975; 181(6):853-856.
4. Veith FJ, Webber WB, Karl RC, Deysine M. Diagnostic peritoneal lavage in acute abdominal disease: normal findings and evaluation in 100 patients. Ann Surg 1967; 166(2):290-295.

ROGER G. BANGS, M.D.
Lafayette, Indiana

October 3, 1991

Dear Editor:

We read with interest the letter from Dr. Bangs in which he shared the case of the young motorcyclist with us. We appreciate his thoughtful comments, and have a couple of comments to make in addition. We are pleased with the interest that has been sparked by our article. Since the article, we have had two letters describing cases similar to the ones we reported. In one of these cases, the patient was a surgeon who injured himself while water skiing and a day or two later had acute appendicitis. It has also been brought to our attention that the great magician, Harry Houdini, probably died of appendicitis of post-traumatic origin. We found Dr. Bangs' use of peritoneal lavage to be interesting, but would agree with his comments that this can often be misleading in this setting. Clearly, traumatic injury to the appendix is extremely rare; it did not merit any comment in a recent comprehensive textbook on trauma.[1] Therefore, we would concur entirely with Dr. Bangs, who reached the same conclusion that we did—namely clinical suspicion is probably the most important aspect of making the diagnosis in these patients who developed appendicitis after trauma. We thank Dr. Bangs for his interest in our report, and for sharing his case with us.

References

1. Moore EE, Mattox KL, Feliciano DV. Trauma, 2nd Edition. Norwalk, CT: Appleton and Lange, 1991.

CHRISTOPHER C. BAKER, M.D
MARK HENNINGTON, M.D.
ELLIS TINSLEY, M.D
Chapel Hill, North Carolina