LETTERS TO THE EDITOR

September 23, 1991

Dear Editor:

We recently reported a series of clinical variables associated with high risk of associated abdominal injury.[1] In that study, assault or abuse as the mechanism of injury was associated with a significant risk of abdominal injury (odds likelihood ratio = 5.08, 95% confidence intervals = 1.07, 24.2, $p < 0.05$), and bicycle accidents were not associated with such risk (odds likelihood ratio, 0.702; 95% confidence interval, 0.21, 2.35; p = not significant).

While performing subsequent analyses on these data, we discovered that a computer error had caused the reversal of the above variables. The corrected analysis shows that children injured as a result of bicycle accidents are at significantly higher risk for having associated abdominal injury. Assault or abuse was not found to be a significant variable in predicting associated abdominal injury based on a multivariate analysis. This is probably due to the relatively small number of children with this variable (n = 26; 4%). Nevertheless, we believe that these children probably should continue to be viewed as "high risk" for abdominal injury. In a prior series, children scanned after suspected abuse had a high frequency of thoracic or abdominal abnormalities (67%), as well as a high mortality rate (50%).[2]

These changes do not alter the conclusions of the original paper. Nonetheless, we regret this error and hope that it has not been the cause of serious inconvenience to readers of *Annals of Surgery*.

References

1. Taylor GA, Eichelberger MR, O'Donnell R, Bowman L. Indications for computed tomography in children with blunt abdominal trauma. Ann Surg 1991; 213:212–218.
2. Sivit CJ, Taylor GA, Eichelberger MR. Visceral injury in battered children: a changing perspective. Radiology 1989; 173:659–661.

GEORGE A. TAYLOR, M.D.
MARTIN R. EICHELBERGER, M.D.
Washington, D.C.

September 19, 1991

Dear Editor:

I wish to raise a criticism of Dr. Klar's conclusion.[1] The conclusion states: "These results lead us to suggest that vasoconstrictor agents may worsen pre-existing pancreatitis in humans and therefore should be avoided if possible." The design of this study and choice of animal model automatically predetermine this result. Perfusion is the issue in septic shock, but perfusion is dependent on adequate flow (cardiac output) and an adequate perfusion pressure (mean arterial pressure). When vasoconstrictors are used to improve perfusion, one is obligated to assure that it is not at the expense of blood flow. In Dr. Klar's study, no attempt was made to document flow stability during the phenylephrine infusion. It is also known that in rats, to maintain cardiac output during septic shock, high-volume infusions must be given or low-flow states result. Therefore, rats are not a true hyperdynamic septic model. Use of phenylephrine to maintain blood pressure alone in the presence of a low-flow state would, by necessity, decrease perfusion to organ systems, and is contraindicated in the treatment of any shock state. Dr. Breslow's study in swine, which more closely approximate human cardiovascular physiology, showed no flow decrease to multiple organ systems with the use of high-dose pressors in the presence of septic shock when filling pressures and cardiac output are maintained at pre-existing levels.[2] Human studies using vasopressors in a systematic approach to septic shock, which aim to augment total perfusion as indexed by oxygen consumption and delivery parameters, have shown beneficial effect in overall survival and organ perfusion, specifically in kidneys.[3–5]

I applaud the authors' attempt to answer the question of regional flow in septic shock; however, I seriously question their use of this model and the subsequent extension of their conclusion to the human condition.

References

1. Klar E, Rattner DW, Compton C, et al. Adverse effect of therapeutic vasoconstrictors in experimental acute pancreatitis. Ann Surg 1991; 214(2):168–174.
2. Breslow MJ, Miller CF, Parker SD, et al. Effect of vasopressors on organ blood flow during endotoxin shock in pigs. Am J Physiol 1987; H291–H300.
3. Desjars P, Pinaud M, Bugnon D, Tasseau F, Norepinephrine therapy has no deleterious renal effects in human septic shock. Crit Care Med 1989; 426.
4. Bonfiglio MF, Dasta JF, Gregory JS, et al. High-dose phenylephrine infusion in the hemodynamic support of septic shock. DICP Ann Pharmacother 1990; 24:936–939.
5. Gregory JS, Bonfiglio MF, Dasta JF, et al. Experience with phenylephrine as a component of the pharmacologic support of septic shock. Crit Care Med 1992; 19:1395–1400.

JAMES S. GREGORY, M.D.
Chicago, Illinois

October 15, 1991

Dear Editor:

We wish to reply to the letter of James S. Gregory, M.D., who has written a critique of our article entitled "Adverse Effect of Therapeutic Vasoconstrictors in Experimental Acute Pancreatitis."

Dr. Gregory points out that studies of the use of vasoconstrictors in septic shock in some animal models do not show that there is any adverse effect, and in fact there is some augmentation of total perfusion in some studies. He points out that particularly in rats high-volume infusions must be given to maintain cardiac output lest vasoconstrictors indeed have an adverse effect.

Although we do not disagree with the material cited by Dr. Gregory, we wish to point out quite clearly that his comments are directed at models of septic shock. Our study is not concerned with septic shock but with pancreatitis. In addition, one should note that the degree of pancreatitis induced by cerulein is quite