problems: 1) The 4+ lesions with skin involvement should have been staged as $T_{1}N_{0}M_{0}$. Both have the same poor prognosis. 2) Many $T_{2}N_{0}M_{0}$ lesions were included as $T_{1}N_{0}M_{0}$. When this was corrected, the treatment failure rate fell from 50% to 33% among the high risk group and from 2% to 1% among the low risk group.

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May 7, 1978

Dear Editor:

Because you have allowed Dr. Condon (Ann Surg 1978; 187:438–439) to carry his argument *ad hominem* (mihi), I suppose I should be allowed to reply as a matter of personal privilege, though I have no wish to prolong this unpleasant discussion beyond the point of a fair hearing of both sides of the issue.

It was not correct of Drs. Condon and Best to say that I am opposed to the use of controlled clinical trials. I did not say that in my letter; moreover my position on this matter was made very clear in Ann Surg 73:161, wherein I took a stand almost identical to that of Dr. Best in stating that, "I do not consider it immoral or unethical for surgeons who espouse the null hypothesis to submit their patients to (a) prospective study, (b) it would be wrong for a surgeon to offer to half of his patients a treatment that he believed probably inferior, even if randomly selected."

Dr. Best and I, then, agree that physicians who "believed strongly that selected drugs were of value" should not have participated in a study involving a placebo group. According to the reference (10) cited by Dr. Condon, three such physicians were Nichols, R. L., Condon, R. E., and Gorbach, S. L.!

Conspicuously omitted from the list of authorities mentioned by Dr. Condon were references to the writings of J. F. Burke, I. Cohn, and W. A. Altemeier, the last of whom, in discussing the paper of Condon et al. said that he had long ago discontinued the use of a placebo group in such studies; Dr. Condon replied, "We share Dr. Altemeier's concern about withholding antibiotics and having a placebo group." Would Dr. Condon's concern have made him a little reluctant to participate in the study himself, as a subject?

One would like to know the extent to which Dr. Condon's "concern" was conveyed to the patients entering this study. Were they informed of the evidence and opinions favoring the systemic and/or intraluminal use of antibiotics? Or were they informed only that the need for the intraluminal antibiotics had not been scientifically established? It would be of interest to see a copy of the consent form.

I must confess that Dr. Condon is partially correct in one of his arguments against my person. In fact, the adjective, "grumpy", perhaps understates the emotion I experienced in reading about the 43% septic complications with seven deaths experienced by the 60 persons in the placebo group—disappointing statistics even for a preantibiotic series.

In a time when most thoughtful physicians would like to see the scientific method used to the optimum extent in order to establish the safety and efficacy of all our practices, it is necessary constantly to review the delicate ethical considerations that must be involved. I don't pretend to have the answers to these questions. But when a study is as close to the knife-edge as Dr. Condon's, it seems important to focus attention on it for a while, rather than for everyone to let it slip by out of our natural inclination not to become involved, to make waves, to appear rude, or to expose ourselves to public embarrassment.

Stephen E. (Grumpy) Hedberg, M.D.
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Massachusetts General Hospital
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January 4, 1978

Dear Editor:

In the article "Hematuria as a sign of aorto-caval fistula" by Brewster, Ottinger and Darling (Ann. Surg. 1977; 186:766), the authors emphasized hematuria as an important diagnostic sign, and repeated the previously described but unconfirmed hypothesis that the underlying regional venous hypertension involving the pelvis and lower extremities is due to the compression of the IVC central to the fistula by the aneurysm.

This hypothesis was tested in the laboratory when we reported recently on two similar cases. 1 Aortocaval fistulae were created in dogs and pull through IVC pressure tracings were obtained. In the absence of an aneurysm, and therefore without any cephalad IVC compression, there was a consistent and marked re-
gional venous hypertension in the IVC caudal to the fistula. We conclude that the pathogenesis of regional venous hypertension is rheological, related with the high velocity of blood flow in the aorta and the parallel anatomy of the aorta and IVC. In these experiments we also confirmed the source of hematuria to be the congested urinary bladder mucosa by the direct cannulation of both the renal pelvis and the bladder. However, our findings would suggest that one should not exclude the possibility of regional venous hypertension and hematuria in patients with large traumatic aortocaval fistulae, even though a compressing aneurysm may not be present.

C. J. Chiu, M.D., Ph.D.
Associate Professor of Surgery
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Reference


January 25, 1978

Dear Editor:

The observations reported by Dr. Chiu and associates are certainly of interest. In our manuscript, we wished to emphasize the concept of regional venous hypertension in patients with an aortic aneurysm and an aortocaval fistula. Such regional increases in venous pressure may lead to mainly peripheral manifestations involving the pelvic organs or lower extremities, rather than the “classic” central effects secondary to increased venous return to the heart.

In patients with an aneurysm, we proposed that compression of the vena cava above the fistula was a contributing factor to such regional increases. The laboratory evidence cited by the correspondents would also indicate an additional significant factor related to flow patterns which direct the high pressure arterial flow inferiorly.

Their laboratory observations regarding the source of hematuria would support our clinical impression that distended veins in the bladder, rather than the renal pelvis, are responsible. In several of our reported patients intravenous pyelograms were normal but cystoscopy revealed dilated fragile veins in the bladder mucosa. Their studies employing direct cannulation would seem to conclusively document the bladder as the source of the hematuria.

David C. Brewster, M.D.
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May 25, 1978

Dear Editor:

In commenting on the article “The Comparative Sur-
vivals of Alcoholics versus Nonalcoholics after Distal Splenorenal Shunt” by Dr. Zeppa (Annals of Surgery, May, 1978), Dr. McGuire made note of the significant problem cirrhotic patients have handling salt. In all of the articles about this operation, there does not seem to be enough emphasis on the use of Spironolactone preoperatively to counteract the very high level of Aldosterone all cirrhotic patients have and which is obviously made worse by the trauma of surgery. Since at least 90% of esophageal bleeding patients can be controlled with Pitressin and/or the Sengstaken-Blake-more tube, the necessary therapeutic levels of Spironolactone can be easily obtained. This level can easily be checked by measuring 24 hour urine electrolytes, which will show a reversal of the pre-Aldactone sodium-potassium ratio. To obtain this response, it is often necessary to give cirrhotic patients 600–1000 mg of Spironolactone per day in divided doses. It takes at least three days to obtain therapeutic levels and obviously should be reinstituted after surgery as soon as the patient is started on a diet.

Dr. McGuire also eluded to the problem of the development of hepatorenal syndrome in the postoperative shunted patient. This has not developed in any of our patients, possibly because of the use of Spironolactone preoperatively. In the patient who does develop hepatorenal syndrome, the physician finds that fluid loading with balanced salt solutions, plasma solutions, or salt-poor albumin in combination with Lasix in an attempt to increase urinary output invariably results in worsening of the patient’s ascites and continued deterioration of the patient’s condition. I wonder if the use of a Levine shunt under these circumstances would not effectively increase this type of patient’s intravascular volume and possibly reverse this lethal syndrome.

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