proceeds to threaten the use of peer review and the FDA to penalize physicians (of course, the word "educate" is used) if they should by chance prescribe one of these horrible preparations.

In summary, I feel that the author's statement that the patient suffers both medically and financially from the use of any product other than a single entity iron preparation has been totally unsubstantiated except by rhetoric.

HOWARD LUTZ, MD
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In Reply.—Dr Lutz's concluding remark is that my statement "the patient suffers both medically and financially" from the use of shotgun hematinic therapy is totally unsubstantiated, except by rhetoric. Yet, Dr Lutz's entire letter is unsubstantiated rhetoric, most of it erroneous and/or inaccurate.

Dr Lutz's assertion that "it seems to me, in my clinical practice, that [iron] preparations that do include folic acid, vitamin C, and possibly other vitamins, seem to work much better..." is highly unscientific and anecdotal. I find it hard to accept the notion espoused by Dr Lutz that "a certain amount of empiricism is important in finding therapies that seem to work for the majority of the patients." The physician is obligated to make the proper diagnosis by finding the cause of the patient's anemia and treating it appropriately. To grope in the dark and to use shotgun hematinic therapy as a substitute for a correct diagnosis, in an inadequately investigated patient with anemia, represents poor medical practice at best.

A major hematology textbook[1] states that "proprietary combination hematinics are promoted with great vigor and great success for the management of a surprising array of unrelated conditions. Yet there is little that can be said about these preparations, except to condemn them."

Why does Dr Lutz find it difficult to understand why occult bleeding from the gastrointestinal tract may be masked by the administration of iron? Anemia from a bleeding carcinoma may never develop and, in the absence of other signs or symptoms, the patient's neoplasm may go undetected until the patient is beyond help, especially if one is dealing with insidious bleeding from carcinoma of the stomach or the right side of the colon.[2]

How can Dr Lutz categorically state that a patient who is given folic acid and iron is unlikely to have pernicious anemia masked? Every medical student knows that the use of folic acid to treat a patient with megaloblastic anemia may precipitate a neurological crisis if the patient has pernicious anemia rather than folate deficiency.

I agree with Dr Lutz that no "ethical formulation" contains amounts of vitamins A or D or ascorbic acid or iron that approach a toxic level. However, although a few pills may not product toxicity, the prolonged or excessive use of such medications may, in fact, produce serious toxicity.

Although Dr Lutz admits to the possibility of vitamin A or D toxicity, he states that: "I do not know of any specific article or reference showing ascorbic acid toxicity...." Dr Lutz is obviously not an avid reader of the current medical literature, nor has he taken the time or effort to look at the *Index Medicus*. Otherwise, he would be familiar with the multitude of articles in numerous journals dealing with ascorbic acid toxicity.[3-13]

I find Dr Lutz's comments to be without merit and without foundation. Dr Lutz is in fact guilty of the very thing of which he accuses me, ie, unsubstantiated rhetoric.

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1. Fairbanks VF, Fahey JL, Beutler E: *Clinical Disorders of Iron Metabolism*, ed 2. New York, Grune & Stratton, 1971, p 320.
2. Holland JF, Frei E III: *Cancer Medicine*. Philadelphia, Lea & Febiger, 1973, p 1092.
3. Herbert V: Risk of oxalate stones from large doses of vitamin C. *N Engl J Med* 298:856, 1978.
4. Du Bruyn DB, De Klerk WA, Liebenberg NW: High dietary ascorbic acid levels and oxalate crystallization in soft tissues of baboons. *S Afr Med J* 52:861-862, 1977.
5. Herbert V, Jacob E, Wong K-TJ, et al: Low serum vitamin B_{12} levels in patients receiving ascorbic acid in megadoses: Studies concerning the effect of ascorbate on radioisotope vitamin B_{12} assay. *Am J Clin Nutr* 31:253-258, 1978.
6. Vitamin C toxicity. *Nutr Rev* 34:236-237, 1976.
7. Mengel CE, Green HL Jr: Ascorbic acid effects on erythrocytes. *Ann Intern Med* 84:490, 1976.
8. Barness LA: Safety considerations with high ascorbic acid dosage. *Ann NY Acad Sci* 258:523-528, 1975.
9. Schrauzer GN, Ishmael D, Kiefer GW: Some aspects of current vitamin C usage: Diminished high-altitude resistance following overdosage. *Ann NY Acad Sci* 258:377-381, 1975.
10. Tan SG, Cunliffe WJ: High doses of ascorbic acid. *Br J Dermatol* 93:731, 1975.
11. Hines JD: Ascorbic acid and vitamin B_{12} deficiency. *JAMA* 234:24, 1975.
12. Walta DC, Giddens JD, Johnson LF, et al: Localized proximal esophagitis secondary to ascorbic acid ingestion and esophageal motor disorder. *Gastroenterology* 70:766-769, 1976.
13. Campbell GD Jr, Steinberg MH, Bower JD: Ascorbic acid-induced hemolysis in G-6-PD deficiency. *Ann Intern Med* 82:810, 1975.

Eosinophil Counts in Bacteremia

To the Editor.—The role of eosinophils in health and disease is still matter for conjecture, yet the tendency for eosinophil counts to fluctuate predictably in certain infectious diseases is well known. Eosinophilia has been described in worm infections, especially trichinosis.[1] Eosinopenia has been described in pneumococcal pneumonia, tuberculosis, typhoid fever, scarlatina and other bacterial infections.[2-4] Bass has induced eosinopenia experimentally in mice first rendered eosinophilic with trichinosis, by effecting *Escherichia coli* pyelonephritis and *Streptococcus pneumoniae* subcutaneous abscesses.[3] During an infectious disease clerkship at Johns Hopkins Hospital in August 1977 I became interested in determining whether eosinopenia or eosinophilia could be used as clinical markers for evaluating ostensibly bacteremic patients.

I obtained the records of 75 adult inpatients who had had positive blood cultures at Johns Hopkins Hospital between May and September of 1977. Cultures positive for probable skin contaminants (diphtheroids, *Propionobacterium*, *Streptococcus viridans*, and *Staphylococcus epidermidis*) were excluded, as were patients with known oncologic disease, renal insufficiency, or history of recent steroid therapy. The total number of positive blood cultures per patient was determined by including only those cultures drawn within one week of another culture positive for the same organism. Complete blood cell counts with differentials drawn within 24 hours of a positive blood culture were used to evaluate eosinophil counts. Absolute eosinophil counts would certainly have been preferable, but, as this study was retrospective, they were not available.

The Table plots the number of positive blood cultures per patient against the percentage of eosinophils in the peripheral smear. Note that none of the patients with more than 3% eosinophils had more than one positive

Eosinophil Counts vs Number of Positive Blood Cultures

No. Positive Blood Cultures	No. of Patients	Eosinophils, % Greater Than				
		0	1	2	3	3
1	32	26	3	0	1	2*
2	23	19	3	1	0	0
3	8	7	1	0	0	0
>3	12	10	2	0	0	0

*One patient with 6%, one patient with 9%.

blood culture and that no patient with more than two positive blood cultures had more than 1% eosinophils.

Although eosinophil counts are subject to variation with the standard differential technique, the data indicate that there may well be a correlation between eosinopenia and bacteremia. Controlled, prospective studies with serial absolute eosinophil counts would seem warranted to determine whether eosinophil counts can be of diagnostic value to the clinician caring for the potentially bacteremic patient.

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1. Mahmoud AAF, Stone MK, Kellermeyer RW: Eosinophilopoietin. *J Clin Invest* 60:675-682, 1977.
Kay AB: The eosinophil in infectious diseases. *J Infect Dis* 129:606-613, 1974.
2. Bass DA: Behavior of eosinophil leukocytes in acute inflammation. *J Clin Invest* 55:1229-1236, 1975.
3. Cohen SG: The eosinophil and eosinophilia. *N Engl J Med* 290:457-459, 1974.

Computerized Tomography-Induced Acute Renal Failure

To the Editor.—Recent reports such as that of Alexander et al[1] emphasize that contrast-media-induced acute renal failure is more common than is generally believed. We are aware of only two brief reports of this entity caused by computerized tomography (CT).[2,3] We recently observed six cases of contrast-induced acute renal failure following seven CT procedures (head, five; body, two). Other known causes of acute renal failure were not present. There were three men and six women, with a mean age of 68 years (range, 28 to 84 years). The underlying illness was multiple myeloma in one patient, diabetes mellitus in two patients, and renal cell carcinoma in three patients. Five cases were characterized by oliguria (<400 mL/24 hr). Mean serum creatinine level (before contrast) was 1.9 mg/dL (range, 1.3 to 3.1 mg/dL) and mean rise in serum creatinine level was 3.7 mg/dL (range, 2.0 to 7.0 mg/dL). No patient required dialysis, and serum creatinine levels returned to baseline in five cases. All patients received iothalamate meglumine injection in doses ranging from 0.59 to 2.11 g of iodine per kilogram (mean, 1.08 g). Possible risk factors included preexisting renal insufficiency (creatinine level >1.5 mg/dL) in five of six patients, dehydration in four patients, hyperuricemia in three patients, diabetes mellitus in two patients, multiple myeloma in one patient, advanced age (>65 years) in five patients, and multiple contrast exposures in three patients. One nondiabetic patient had suffered a previous episode two months earlier, suggesting a susceptibility to this form of nephrotoxicity in some patients.

We are not aware of any evidence to suggest that renal cell carcinoma could potentially be a risk factor. We suspect that the occurrence of acute renal failure in such patients was coincidental. Recognition and appropriate management of CT-induced acute renal failure are important because the use of CT scanning has increased dramatically and nearly all such procedures are performed with the aid of contrast enhancement.

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1. Alexander RD, Berkes SL, Abuelo JG: Contrast media-induced oliguric renal failure. *Arch Intern Med* 138:381-384, 1978.
2. Berezin AF: Acute renal failure, diabetes mellitus, and scanning. *Ann Intern Med* 86:829-830, 1977.
3. Hanaway J, Black J: Renal failure following contrast injection for computerized tomography. *JAMA* 238:2056, 1977.

Urethritis and Pure Urinary Tract Infection (UTI)

To the Editor.—Komaroff et al should be commended on their interesting and informative article in the ARCHIVES regarding the evaluation of dysuria in women with and without vaginitis (138:1069-1073, 1978). However, several problems in their analysis of their results should be noted. In Table 2, "urethritis" is listed as a subcategory of "pure UTI," whereas the authors imply that this diagnostic entity refers to patients with lower urinary tract symptoms, normal findings on pelvic examination, and insignificant bacterial growth on urine culture, a clinical situation that is often termed the "urethral syndrome." As noted in other studies,[1] the urethral syndrome accounts for approximately one third to one half (in this case 40 of 98, or 41%) of all cases of dysuria in the absence of marked vaginitis or gonorrhea. As patients with the urethral syndrome generally do not respond to antibiotics,[2] grouping them with bacteriuric "pure UTIs" for purposes of outpatient diagnosis and management is misleading.

The authors attempt to deal with their patients who have "pure" urethritis in Table 3, and, in agreement with other authors,[3] they find that symptoms of dysuria, urinary frequency, and vaginal discharge are similar in frequency in patients with substantial bacteriuria and in patients with the urethral syndrome. However, I fail to understand how they can attribute symptoms to pure urethritis if 12% of the patients also complain of vaginal discharge.

Finally, we are told that the study period was extended five months to include 55 additional cases of pure UTI. Examination of Table 3 shows 13 additional cases of Gram-negative UTI with heavy bacteria growth, and nine additional cases of urethritis. It would be interesting to know the findings on the remaining 33 patients.

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1. Dans PE, Klaus B: Dysuria in women. *Johns Hopkins Med J* 138:13-18, 1976.
2. Charlton CAC, Cattell WR, Canti G, et al: The non-urethral syndrome, in Brumfitt W, Asscher AW (eds): *Urinary Tract Infection.* London, Oxford University Press, 1973, pp 173-177.
3. Gallagher DJA, Montgomerie JZ, North JDK: Acute infections of the urinary tract and the urethral syndrome in general practice. *Br Med J* 1:622-626, 1965.

To the Editor.—I read with considerable interest the article by Komaroff and associates in the July issue of the ARCHIVES (138:1069-1073, 1978) concerning vaginal infection.

I was puzzled by the avoidance of examining patients with vaginitis for the organism *Corynebacterium vaginale* (referred to by some as *Haemophilus vaginalis*). Although I realize that some controversy still remains concerning the degree of pathogenicity of this organism, it is considered by many workers to be a cause, if not the most important cause, of so-called nonspecific vaginitis.

It is unfortunate that a rather major aspect of vaginal infections is not included in an otherwise interesting approach to common problems.

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In Reply.—We thank Drs Diehl and Lewis for their kind remarks about our article. Dr Diehl's definition of the "urethral syndrome" is identical with ours (urethritis). To clarify one point, a patient with the recent symptom of vaginal discharge, but without any abnormal discharge evident on pelvic examination, could be labeled as