well as that of others, does not support the validity of the criteria."[3] It is now clear that Rich's experimentally induced angiitis more closely resembled Zeek's hypersensitivity angiitis than PAN.

Born in 1899 in Ironton, Ohio, Dr. Zeek still maintains her interest in PAN. On June 4 she celebrated her 92nd birthday, and I invite all who benefit from her work to join me in sending her warmest wishes and many happy returns.

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1. Calabrese LH. Differential diagnosis of hypersensitivity vasculitis. Cleve Clin J Med 1990; **57**:506–507.
2. Zeek PM, Smith CC, Weeter JC. Studies on periarteritis nodosa: differentiation between vascular lesions of periarteritis nodosa and of hypersensitivity. Am J Pathol 1948; **24**:889–917.
3. Rich AR. Studies on hypersensitivity. Can Med Assoc J 1958; **78**:163–170.

DRESSINGS FOR STASIS ULCER

■ *To the Editor:* I read with interest the article by Drs. Young and Terwoord[1] on a compression dressing system for stasis ulcer treatment, in the September 1990 issue. This is a very appealing and, no doubt, effective way to treat stasis ulcers. However, in an era of cost containment, the cost of this type of treatment seems excessive. In our area, the therapeutic stocking would cost $44 or more if it were made to measure. *The Allevyn* 4 × 4 sterile dressings would cost $57.50 for a box of six, and the Intrasite 4 × 4 sterile dressings would cost $32 for a box of six.

It has been my habit for some time to use an alternative form of care which has been extremely effective. It was originally taught to me by Dr. Brownell Wheeler, Chief of Surgery at University of Massachusetts Medical School, and he learned it in England. The technique consists of a small amount of antibacterial ointment placed on the ulcer, covered with a Vaseline gauze or adaptic and, in turn, covered with a 3 × 3 gauze pad. Over this, a roll of Webril (cost, about $1) is smoothly wrapped, and over this an Elastikon bandage (cost, about $5.25). This dressing can be left on perfectly safely for a minimum of 2 weeks, even in the presence of a draining ulcer. I have had patients wearing this kind of dressing for as long as 3 to 4 months without changing it. This, also, has proven to be perfectly safe.

I think this cost information should be brought to the attention of your readers.

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1. Young JR, Terwoord BA. Stasis ulcer treatment with compression dressing. Cleve Clin J Med 1990; **57**:529–553.

■ *Reply:* Dr. Hill brings out a good point regarding the cost of the Jobst UlcerCare system when using Allevyn or Intrasite dressings.

Since the publication of our article in the *Cleveland Clinic Journal of Medicine*, we too have become concerned about the cost of the Allevyn and Intrasite dressings. In addition, a few patients have noticed some sensitivity reactions. Because of this, we rarely use these two dressings. Instead, we use normal saline dressings for infected ulcers. We still find the light white compression liner stocking very helpful in holding the dressings in place. It also enables patients to put on their heavy elastic stockings more easily.

The literature contains hundreds of ways to treat stasis ulcers. Dr. Hill's method is an interesting one, but I would be concerned about maceration of the skin when the drainage is excessive. In addition, the patient would also not be able to observe or bathe the leg while the dressing was on.

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ASPIRIN AND REYE'S SYNDROME

■ *To the Editor:* I write to protest at the misrepresentation of our study[1] in the article by Orlowski et al[2] and the editorial by Hurwitz and Mortimer[3] in the July 1990 issue of the Cleveland Clinic Journal of Medicine. It was particularly unfortunate that the authors of the accompanying editorial did this, as our study findings support their views rather than refute them as they implied. We accept that our methodology was not as rigorous as that in the most recent US

case-control studies; nevertheless, we did find a significant association between Reye's syndrome and prior aspirin ingestion in British patients.

Even though exposure was reported in only 59% of our Reye's syndrome patients, this represented a highly significant difference from the rate in our comparison group—a fact omitted by Dr. Orlowski. There was also no reference in either paper to our finding that *within* our cases there was a statistically significant trend between aspirin exposure and "Reye score"—an instrument which we devised to measure how closely our cases matched the "typical US" form of Reye's syndrome referred to by Hurwitz and Mortimer. In fact, almost all of our high-scoring cases had taken aspirin, compared to none at the lower end of the scale, and we concluded that the overall rate (59%) was low because our cases were diluted by "Reye mimickers."

Reye's syndrome in Britain has, as in the United States, declined dramatically in the last 4 years, and the age distribution has shifted towards the very young; we no longer see the "US type" cases and, for that reason, support the conclusion of Hurwitz and Mortimer's editorial that the warning about aspirin use in children should remain.

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1. Hall SM, Plaster PA, Glasgow JFT, Hancock P. Preadmission antipyretics in Reye's syndrome. Arch Dis Child 1988; **63**:857–866.
2. Orlowski JP, Campbell P, Goldstein S. Reye's syndrome: a case control study of medication use and associated viruses in Australia. Cleve Clin J Med 1990; **57**:323–329.
3. Hurwitz E, Mortimer EA. A catch in the Reye is awry. Cleve Clin J Med 1990; **57**:318–320.

■ *Reply:* We continue to find it untenable that Reye's syndrome is a different disease in the United States than anywhere else in the world. Therefore, the development of a Reye score to measure how closely cases matched the typical US type of Reye's syndrome is irrelevant.

The "gold standard" of Reye's syndrome diagnosis is a combination of the following: 1) standard case criteria, 2) liver biopsy demonstrating microvesicular steatosis, 3) histochemical examination of the liver showing absent succinic dehydrogenase activity, 4) electron microscopy demonstrating the typical ultrastructural findings, 5) biochemical investigations to rule out inborn errors of metabolism, and 6) an admission salicylate level. Our study comes the closest to fulfilling this gold standard and fails to show any relationship between Reye's syndrome and aspirin ingestion.

The most important point is that 86% of our Reye's syndrome cases[1] were confirmed pathologically, whereas fewer than 33% of the cases from the US Public Health Service and only 48% of the cases of Hall et al[2] were histologically confirmed as Reye's syndrome. Assumptions about the role of aspirin in Reye's syndrome are questionable when more than half and up to as many as two thirds of the cases are not even proven Reye's syndrome.

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1. Orlowski JP, Campbell P, Goldstein S. Reye's syndrome: a case control study of medication use and associated viruses in Australia. Cleve Clin J Med 1990; **57**:323–329.
2. Hall SM, Plaster PA, Glasgow JFT, Hancock P. Preadmission antipyretics in Reye's syndrome. Arch Dis Child 1988; **63**:857–866.

■ *Reply:* We concur with Dr. Hall that her study, conducted in Britain, provides evidence for an association between Reye's syndrome and aspirin. We did not intend to reference her study in this way and can only assume that inclusion of the reference number was an editorial error on our part.

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