cian is instructed to use sodium citrate intravenous injections to reduce the amount of ionized calcium in the blood.

I take strong issue with the suggestion that sodium citrate has any beneficial effect in the hypercalcemia of vitamin D intoxication or any other hypercalcemic state.[1] Harrison and Harrison[2] have reported that changes in the citrate contents of the blood are not dependent on changes in serum calcium. I have seen no reports of serum calcium being dependent on serum citrate levels.

Moreover, the advertisement ignores the role of glucocorticoids in the treatment of vitamin D intoxication and states of vitamin D sensitivity (such as sarcoidosis and infantile hypercalcemia). The usefulness of glucocorticoids was appreciated almost from the initial trials.[3,4] Return to normocalcemia no longer required two to three months, but only seven to 14 days with cortisone.[5]

In fact, in an early report of the rapid response of the manifestations of vitamin D intoxication to cortisone, Verner[5] stated that cortisone was an important addition to the therapeutic regimen of critically ill patients with vitamin D intoxication.

These important clinical observations have been supported by in vitro studies by Harrison[6] and more recently by Kimberg[7] in which glucocorticoids have been shown to have an effect on the intestinal absorption of calcium opposite in direction to that of vitamin D but not dependent upon a direct interaction with the vitamin.

Clearly, then, the efficacy of glucocorticoids is established, and the contents of the advertisement is not only misleading but a disservice to physicians who may use the information given.

MURRAY J. FAVUS, M.D.
Fellow in Endocrinology
Beth Israel Hospital
Boston, Mass.

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Note: Philips Roxane Laboratories have informed the *Journal* that the deficiencies that were to be found in both the package insert and the advertisements for dihydrotachysterol have been recognized and that appropriate changes are being made. In the meantime, the advertisement in question has been withdrawn. — ED.

SCALP TOURNIQUET TO LESSEN ALOPECIA AFTER VINCRISTINE

To the Editor: Loss of scalp hair ranging from partial to total baldness is a frequent complication of cancer chemotherapy. This may have profound psychologic effects on children, particularly adolescents, and their families. The use of wigs only partially eases the emotional distress caused by alopecia.

A number of chemotherapeutic agents produce alopecia with varying degrees of regularity. The alkaloid, vincristine (Oncovin), is one of the greatest offenders, causing alopecia in 23 to 71 per cent of the patients receiving it,[1-4] and hair loss has been shown to be unrelated to dose.[4] Because this drug is useful in many cancers, including acute leukemia, Wilms's tumor, neuroblastoma, lymphoma and rhabdomyosarcoma, it is widely used in the management of children with cancer.

A brief report in the British Medical Journal described the use of an occlusive scalp tourniquet that markedly reduced the frequency of hair loss after vincristine therapy.[5] The rationale for the use of the scalp tourniquet was that the vinca alkaloids are rapidly cleared from the bloodstream after intravenous injection,[6] presumably because of tissue fixation of the drug. Since the scalp is supplied by superficial blood vessels that can be temporarily occluded by pressure, the contact of the drug with hair follicles can be minimized.

A specially constructed scalp tourniquet* is placed around the head just above the ears and inflated to 10 mm of mercury above systolic pressure immediately before the injection of the drug. The pressure is maintained continuously for five minutes after injection.

During the past two years at Yale-New Haven Hospital we have treated more than 30 children with vincristine (1.5 mg per square meter for five to seven weeks), using this tourniquet. Obvious alopecia has developed in only three patients. One of these was also receiving cyclophosphamide and another actinomycin D. In the third, the tourniquet slipped off before five minutes had elapsed.

We have used the scalp tourniquet concomitantly with vinblastine and actinomycin D therapy, but have not treated sufficient numbers of patients to state unequivocally that it is of benefit. It has not been effective in protecting against cyclophosphamide-induced alopecia, presumably because this drug is not rapidly tissue fixed. In children with acute leukemia a small number of leukemic cells in the scalp circulation may be protected from the action of drugs during the period of occlusion. However, we have not been impressed by any difference in remission rates.

Alopecia after vincristine can be markedly reduced by the scalp tourniquet. We wish to call attention to the effectiveness of this simple maneuver in decreasing the incidence of a disturbing side effect in an already distressing clinical situation.

RICHARD O'BRIEN, M.D.
JOSEPH H. ZELSON, M.D.
ALLAN D. SCHWARTZ, M.D.
HOWARD A. PEARSON, M.D.
Department of Pediatrics
Yale University School of Medicine
Yale-New Haven Hospital
New Haven, Conn.

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*Available on special order from W. A. Baum Co., Copiague, New York 11726

NO DICTATED PRACTICE

To the Editor: May I take issue with Dr. Shocket (New Eng J Med 283:1057, 1970) on three points?

First of all, Dr. Shocket implies that doctors have an obli-