# Editorial

## *Diabetic Acidosis**

Optimal therapy of diabetic coma is in a sense largely a public health problem! The fact of the matter is that the major stumbling block in most of our hospitals and practices is the failure to apply properly and accurately even *one* of the many reasonable systems of management. That the proper and accurate application of *one* of these systems is more important for successful therapy than controversies concerning differences among these systems is the contention of this editorial.

Can we plead innocent to the charge that our patients with diabetic acidosis are frequently if inadvertently neglected? I think not. How often is treatment of this medical emergency undertaken, by default perhaps, by an inexperienced interne or resident? How often do practitioners who see diabetic coma once or twice a year, and who in any case are quite insecure in their therapy, assume full responsibility? In my opinion, the incidence of these indiscretions is far greater than many of us might care to admit.

We need not think that the distressingly casual approach to the problem of diabetic acidosis characterizes only certain private hospitals and certain private medical services. Slipshod therapy of this disorder is also a hallmark of some of our major teaching hospitals.

For example, suppose the full-time staff of a given University Department of Medicine is primarily interested in, say, infectious diseases and in diseases of the liver. The latter patients might then receive meticulous attention. The patient with diabetic acidosis, on the other hand, might be uncomfortably regarded as a black sheep who happens along before or after regular working hours. Since such a patient presents no unusual infection or exotic liver disease, therapy can be conveniently directed by a house officer, perhaps one who has just begun his tour of the medical service!

It appears that optimal therapy of diabetic acidosis depends in great part upon attention to and knowledge of detail, upon planning, and upon co-ordination of many services. I believe that the basis for success in this disorder rests upon a scheme in which each patient is carefully followed by experienced and devoted groups of physicians and allied personnel.

For example, in every hospital a team interested in and responsible for the detailed care of *all* patients with diabetic acidosis could be organized. Such a group, depending on hospital size, might consist of at least one physician, several nurses, and a competent laboratory technologist. A conscientious team of this type would soon achieve remarkable facility in providing the best care for the patient with diabetic acidosis. It is clear that planned, centralized, assiduous care of the patient with diabetic acidosis is fundamental to successful therapy.

Many controversies are raging. Should glucose be given early or late; should fructose be used instead of glucose; should potassium be administered routinely? I believe that such controversies sometimes become smoke screens for common, glaring, and sometimes fatal errors: For example, patients with diabetic coma may not receive insulin for several hours after arrival in the emergency room; or

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patients who report obvious symptoms of severe acidosis over the telephone are advised to come in the next morning because of the lateness of the hour; or a responsible physician is not personally checking upon the patient at least hourly until ketosis has largely subsided.

It would seem that our batting average in diabetic coma could be greatly improved if our program included these two vital factors:

(1) Organization of a competent team responsible for all such patients in any given institution.

(2) Following one of the many reasonable systems of management of the disorder.

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## *Man's Requirement for Vitamin C*

The British, who have done such outstanding work in nutrition since the days of John Lind,[1] have carried on this tradition with the publication of a significant study by a subcommittee of the Medical Research Council.[2] The Chairman was Sir Rudolph Peters, and among the committee members was Professor H. A. Krebs of the University of Sheffield.

Twenty conscientious objectors were studied. They received a diet containing less than 1 mg. of vitamin C daily. Three received a 70 mg. supplement of the vitamin daily; seven received 10 mg. daily; and 10 remained unsupplemented.

Clinical signs of scurvy developed in all 10 unsupplemented subjects. The first changes noted were enlargement and keratosis of hair follicles after about 17 weeks of deprivation. Some follicles later became hemorrhagic and developed into characteristic scorbutic spots. Gum changes were seen after 26 weeks of vitamin C deficiency.

Among the other signs noted were pains in the back and limbs, exacerbation of acne, ecchymosis, and knee joint effusion. However, considerable variation in degree was observed. Conventional tests of "capillary strength" failed to show a correlation with the states of vitamin C depletion.

Perhaps the most interesting finding was that 10 mg. of vitamin C daily given to six of the scorbutic subjects removed the clinical signs in all. The skin lesions disappeared in about two months and the gum lesions in three months on this small supplement.

It is significant that the concentration of vitamin C in the white cells appeared to be a useful guide to the state of vitamin C nutrition. The lowest values were reached 3 to 6 weeks before clinical scurvy developed, whereas the *plasma* vitamin C level was practically zero as long as 100 days before clinical scurvy developed.

In the scorbutic subjects, no change in hemoglobin, red or white cell count, or bleeding time was noted.

Experimental wounds were made in the skin of these volunteers and the rate of healing was studied by many techniques, including histologic examination. Delayed healing developed in the deprived group after a long period of time, but not in the early stages. It never developed in the two supplemented groups.

As the report indicates, the fact that a supplement of 10 mg. daily cured clinical scurvy and the fact that 10 mg. of vitamin C daily protected these subjects for periods up to 424 days suggest that the minimum protective dose (as indicated by the signs of scurvy) was in the range of 10 mg. daily. However, certain tests of physical fatigue suggest that the group receiving 70 mg. daily had a better overall performance record than did the 10 mg. group. It is to be expected that the prevention or cure of scurvy is possible on doses less than those necessary for "optimum health."

To satisfy the ill-defined additional needs associated with various human activities and

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