vere shock, encephalopathy, pyrexia, watery or bloody diarrhea, and disseminated intravascular coagulation, with a failure to identify a causative infective organism.

The marked increase in CK (maximum activity, 4201 U/L; reference interval 24–135 U/L) was largely of skeletal muscle origin (CK-MB <1%). We decreased the potential of adenylate kinase to falsely enhance the measured CK activity by using an assay procedure incorporating, per liter, 5 mmol of adenosine monophosphate and 10 μmol of diadenosine 5'-pentaphosphate, components shown (5) to inhibit 75% of CK activity in serum that originates from liver and over 97% of the activity of the muscle and erythrocyte isoenzymes. Thus it is difficult to attribute the massive increase in CK activity to interference from this source. This, coupled with a rapid return of CK, AST, and LDH activities to normal values (see Figure 1) without residual liver damage either by clinical or biochemical means, suggests that the increased AST described in this condition is largely derived from damage to skeletal muscle rather than liver. We therefore consider it important to measure CK in cases of sudden onset coagulopathy without an obvious cause and not merely to assume that increased transaminase activity results exclusively from liver damage. Measurement of CK may also prove valuable in differentiating HSES from similar conditions such as Reye's syndrome, in which increases in AST and ALT are not accompanied by an increased CK activity.

References

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Does the Effective Diminution of Nonenzymatic Glycation by D-Lysine Really Highlight Its Potential Use in Vivo?

To the Editor:

Sensi et al. (1) proposed the use of D-lysine for in-vivo studies in healthy and diabetic animals. We cannot agree with this postulate, and we present our major arguments against its usefulness:

D-Amino acids are constituents of normal food, but many of them are highly toxic for the mammalian system (2, 3). Thus the digestive tracts of higher animals contain high activities of D-amino oxidases, to degrade (or) to convert the D form to the L form (4). This well-known biochemical and physiological fact can be found in most textbooks.

If D-lysine could be resorbed by the intact intestinal wall, the potential toxicity of the D-lysine acid is obvious. Even when D-lysine is converted to its naturally occurring L isomer—which is practically the fact—it still is toxic when given in high concentrations. Racusen et al. (5) found that lysine produces acute renal failure in rats. The sequence of elevated intratubular pressure and tubular dilatation, followed by decreased clearance of inulin and then by decreased renal blood flow, suggests that lysine produces acute renal failure primarily through tubular obstruction. This tubular damage later leads to increased renal vascular resistance. This is not due to nonspecific effects of amino acids, because lysine is the only essential amino acid that depresses renal function. Furthermore, it is well known and documented that lysine blocks tubular reabsorption (6), and it must be suspected that lysine is the component in hyperammonemia solutions that is responsible for their adverse effects in experimental animals (7).

References

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Dr. Sensi responds:

To the Editor:

Lubec et al. have not questioned here the value of our experimental in vitro work on the effect of D-lysine on protein nonenzymatic glycation, but rather our hypothesis of its possible therapeutic use in vivo. We believe that the principle of utilization of D-lysine in diabetes is correct and could be of help in contributing to control the pathogenesis of late diabetic complications. However, in attempting to offer answers to their speculative criticisms we would only introduce more speculations rather than solid facts. Studies are now in progress with experimental animals to determine all physiological and toxicity variables associated with D-lysine utilization. It will then be possible to have a proper debate on the subject.

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Serum Creatine Kinase in Primary Hypothesis

To the Editor:

We observed a case of increased creatine kinase (CK; EC 2.7.3.2) activity after primary hypothermia. The patient, a 72-year-old woman, was noticed to have "sick sinus" syndrome and a pacemaker was implanted 16 years ago, and she had had Parkinson's disease for 15 years. She was well and active until one winter afternoon when her family noticed that she could not walk well and she had be