usual MET-EL-S rubber stopper (3.97 and 3.99 mg/litre), as compared to bottles capped with Parafilm (2.52 and 2.26 mg/litre). For the nitric acid washed MET-EL-S rubber stopper, a zinc value exceeding 30 mg/litre was found. The contents of a bottle of the MET-EL-S quality-control serum was mixed and left inverted on the rubber stopper for five days, when the zinc value was found to be 6.80 mg/litre. The zinc in MET-EL-S control serum is likely to be a function not only of the amount of zinc in the serum but also of how long the control serum remains in contact with the rubber cap. Therefore, no true range for zinc can be established for MET-EL-S quality control serum until zinc contamination from the rubber stopper is corrected.

References

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Alcoholic Myopathy and Changes in Serum Enzyme Activity

To the Editor:
In a recent Case Conference (1) the authors state that the high values for creatinine kinase (EC 2.7.3.2), lactate dehydrogenase (EC 1.1.1.27), and aspartate aminotransferase (EC 2.6.1.1) in an alcoholic patient with acute pancreatitis and seizures was probably related to cellular injury resulting from tissue damage incurred during the seizures and... when the patient had to be restrained in bed."

This explanation may well be correct for the patient presented. However, I would like to call attention to an alternative explanation for similar patterns of serum enzyme alterations in patients who are alcoholic, or who have recently consumed moderately large amounts of alcohol: the possibility of the presence of alcoholic myopathy.

The principal morphologic abnormality observed by light microscopy in alcoholic myopathy is segmental necrosis of muscle fibers. This change is not pathognomonic, being present also in cases of muscle trauma or severe anoxia (2). Alcoholic myopathy is not uncommon. Fifteen alcoholics were studied after having consumed no alcohol for two weeks, during which period they received vitamin-supplemented normal diets (3). Ten of 13 muscle biopsies were abnormal, with overt myopathy present in two patients. Of the 15, 14 had abnormalities of muscle function by electromyography, accompanied by loss of muscular strength. In another series (4), 109 of 191 patients admitted for detoxification showed electromyographic abnormalities related to muscle function.

The relationship of the condition to the toxic effects of alcohol rather than to accompanying nutritional problems seems convincing. Curran et al. (5) reported the case of a chronic alcoholic with cardiomyopathy and alcoholic gastritis, who presented with severe muscular weakness and tenderness, but no atrophy. On admission, creatine kinase activity was 40-fold normal, and lactate dehydrogenase and aspartate aminotransferase were 17-fold normal. After 21 days, the patient's first-degree heart block had disappeared. Muscular strength was nearly normal, as were activities of lactate dehydrogenase and aspartate aminotransferase, but creatine kinase activity was still three times normal. Ingestion of one pint of whiskey was followed by return of muscular weakness and elevation of creatine kinase activity to eightfold normal. Most convincingly, Rubin et al. (6) found ultrastructural changes in the muscles of chronic alcoholics following withdrawal, while on supplemented normal diets. The changes consisted of intracellular edema, enlargement and distortion of the sarcoplasmic reticulum, and increased fat and glycogen. The same authors also found that isolated actomyosin preparations from baboons and from human volunteers displayed reduced contractility in vitro.

I autopsied a middle-age woman, a chronic alcoholic, four weeks after her admission to hospital. During her terminal illness, she continued to have values for creatine kinase that were between three and five times the upper limit of normal. She died in hepatic coma due to cirrhosis. Section of her diaphragm, psoas, and intercostal muscles showed numerous areas of segmental necrosis.

Thus I believe that skeletal muscle damage due to acute or chronic alcohol intake must be considered in the differential diagnosis of patients with above-normal values for serum enzymes of possible skeletal muscle origin, especially creatine kinase.

References

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Kit Vendors: Make the Purchaser Aware of Compromises

To the Editor:
Recent letters by Alexander [Clin. Chem. 23, 1369 (1977)] and Tung et al. [Clin. Chem. 23, 1370 (1977)] concerning the Beckman Enzymatic Amylase reagent emphasize how important it is for manufacturers of enzymic reagents to supply the user with all of the technical information required for the meaningful application of these reagents in the clinical laboratory. The interdependence of user and manufacturer was clearly stated in this journal [Clin. Chem. 18, 1454 (1972)] by Moss:

... the clinical enzymologist should be prepared to specify whether and to what extent he is prepared to tolerate a relaxation from the strict requirements of kinetic analysis in the interest of convenience of automation and rapid rates of analysis. Manufacturers of automatic reaction-rate analyzers are entitled to receive this information from the user, and should actively seek it when developing this type of apparatus. In return, when the apparatus is marketed, the necessary compromise with strict kinetic principles, as well as its advantages, should be frankly stated.

Our own experience with kits and reagent packs for the determination of amylase activity parallels Alexander's. We have found that both the Beckman and DuPont amylase reagents measure enzyme activity in the lag phase. Differences between zero-order and lag-phase determinations are difficult to predict for any given specimen and the magnitude of change is such that any given specimen may produce an erroneous interpretation. The argument