

TYROSINE METABOLISM IN THE PROLONGED CRUSH SYNDROME

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The content of free tyrosine in the blood serum and excretion of tyrosine metabolites, p-hydroxyphenylpyruvic and homogentestic acids, in the urine were studied in 40 rabbits with a syndrome of prolonged crushing of the soft tissues. No changes in the tyrosine concentration were found. Excretion of metabolites of tyrosine in the acute form of this syndrome showed a considerable change which correlated with changes in diuresis.

Tyrosine metabolism during a prolonged crush syndrome, characterized by the development of toxemia, was studied in experiments on 40 rabbits [1, 3].

The concentration of free tyrosine in the blood serum was determined by the method of Udenfriend and Cooper [4]. In addition, the diuresis was measured, and the excretion of p-hydroxyphenylpyruvic acid (p-HPA) in the urine was estimated by a modified Millon's reaction, and the excretion of homogentestic acid (HGA) was estimated by the writers' modification of Briggs' method [2].

Crushing of the soft tissues of both thighs caused the development of a severe crush syndrome in the animals with a total mortality of 85%. Half of the rabbits died from shock, while the rest developed severe uremia. Depending on the time of death of the animals, three forms of crushed syndrome were distinguished: fulminating (death within the first 36 h), acute (death during 2-7 days), and chronic (animals survived). In

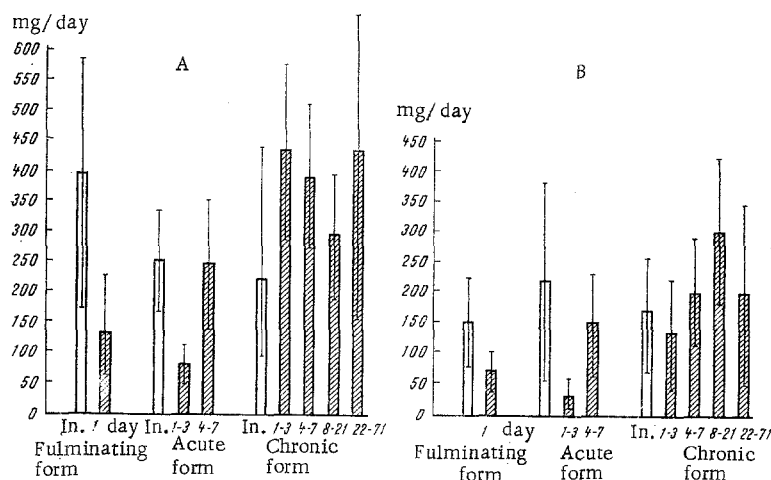


Fig. 1. Excretion of p-HPA (A) and HGA (B) in prolonged crush syndrome. Numbers near columns show time in days. In) Initially.

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the fulminating form and in the initial period of the acute form of crush syndrome, the excretion of p-HPA and HGA with the urine was sharply reduced (Fig. 1). This was evidently associated with the decrease in diuresis, which amounted to more than 50%. At the time of death of the animals with the acute form of crush syndrome, the diuresis and excretion of p-HPA and HGA had returned to their initial level. The serum tyrosine concentration in the fulminating form of crush syndrome was not significantly affected, while in the acute form it was slightly increased. In the chronic form of crush syndrome the urinary excretion of neither p-HPA nor HGA was significantly changed, although the serum tyrosine concentration by the end of the first week showed a tendency to increase, and in the subsequent two months it fell considerably.

In rabbits with the crush syndrome the blood tyrosine concentration is thus considerably reduced during the recovery period. The decrease in excretion of p-HPA and of HGA in the period of development of shock during the crush syndrome is evidently attributable mainly to the decrease in diuresis.

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