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Metabolism

PSEUDOCHOLINESTERASE LEVELS AFTER BODY BURN Cholinesterase and pseudocholinesterase levels in the erythrocytes of 69 persons who had suffered extensive body burns were determined at intervals. The normal levels in the authors' laboratory are 2.3–4.0 I.U. for cholinesterase and 2.5–5.0 I.U. for pseudocholinesterase. Thirty-seven patients with an average burn surface area of 23 per cent survived, while 22 who died had an average burn surface area of 56 per cent. Seven patients had been exposed to organic phosphorus compounds at the time of their burns; of these, only one survived. In the survivors, the average pseudocholinesterase level started at 3.6 I.U., fell below 2.0 by the fourth post-burn day, reached a low of 1.4 on the twelfth post-burn day, and returned to 2.0 I.U. by the sixteenth day. In the patients who died, the average pseudocholinesterase level started at 2.0 I.U., fell 1.0 I.U. on the fifth day, and continued to decline to 0.5 I.U. or less by the sixteenth day. Signs and symptoms observed with the low pseudocholinesterase levels (1.0 I.U. or less) included confusion, blurred vision, generalized muscle twitching and weakness, increased tracheobronchial secretions, bronchoconstriction, respiratory muscle weakness, dyspnea, and cyanosis. The mechanism of death in the burned patient with cholinesterase depletion appeared to be primarily one of respiratory failure, often associated with pleural effusions. Treatment of the low pseudocholinesterase states consisted of the administration of blood stored for less than 24 hours. (*Price, W. R., and others: Enzyme Depletion in Major Thermal Burns, Amer. J. Surg. 120: 671–675, 1970.*) EDITOR'S COMMENT: It is of interest to contemplate the possible relationship between these findings and the use of muscle relaxants in similar patients. For example, do we know what happens to pseudocholinesterase levels after massive transfusion with blood stored for more than 24 hours? Parenthetically, one can only express surprise at the largesse exhibited by these authors in concluding that patients died of respiratory failure without any indication whether an attempt was made to support lung function. It is relevant, however, to include acute hypopseudocholinesteresemia as a source of acute respiratory insufficiency! The more we look, the more we find.