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Diabetic Coma: Some Problems Not Solved By The Nobel Prize 1923

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ABSTRACT

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Introduction

At the beginning of the 20th century, two diseases had 100% lethality: pernicious anemia and diabetic coma. Between the two World wars substantial progress in their treatment has been honoured with Nobel prizes.Today, at the beginning of the 21th century, nobody remembers lethal outcome in a patient with pernicious anemia, whereas between 1990 and 2010 in USA died yearly 2000-3000 patients in „hyperglycemic crises" despite the availability of insulin therapy [1]. Was the Nobel prize 1923 to Banting and Macleod an error? It is very complicated to explain this unexpected reality. Lethal coma in diabetes has been first reported 1874 by Kussmaul [2]. 3 patients, 16-35 years old, with diabetes mellitus, died in coma; in one of them was aceton in his urine. Already 1881 and 1886 has Dreschfeld [3,4]. emphasized that coma in diabetic patients occurs not only with aceton and acetoacetic acid in their urine, but also without them in their urine. Unfortunately, these facta have not been noted until 1957 after a case-history reported from South Africa [5]. It is interesting that this report did not come from the USA or Europe, where many diabetologists were active and it is very difficult to understand why the world diabetology has not accepted coma without ketoacidosis according to the paper of Dreschfeld.

Very good exemple for existence of coma without aceton is the paper of Rolly [6] from 1913: at this time, it was already possible to measure pH of the blood; in 11 comatose diabetic patients only 3 patients had acidosis. After the paper of Sament and Schwartz [7]. a„new interpretation" of the two kinds of diabetic coma was necessary: „decrease" of insulin cocncentration in the blood is the cause of non-ketotic hyperglycemic coma and „absolute" deficiency of insulin in the blood is the cause of ketoacidotic coma. A definitve solution was possible only after the Nobel prize 1977 to Rosalyn S. Yalow for development of new methods of biochemical analysis that make possible to measure insulin concentration in human plasma. In patients with diabtic ketoacidosis have been observed sufficient amounts of plasmatic insulin, e g [7]. On the other hand, absolute deficiency of plasmatic insulin has been reported also in diabetic patients with hyperglycemic hyperosmolar non ketotic coma, e g [8]. and in diabetic patients on routine control, without subjective complaints, e g [9]. Thus, insulin deficiency is not the cause of ketoacidosis. As the cause of diabetic ketoacidosis and coma has been identified low blood-pH [10]. and this has been confirmed in both pediatric [11]. and adult [12]. patients. The validity of this observation is confirmed by zero lethality of patients in diabetic ketoacidotic coma after treatment with infusions of sodium bicarbonate, e g [13]. The decrease of blood-pH is caused by increased amouts of 36 organic acids [14]. In conclusion, insulin treatment is successfully life-saving in hyperosmolar hyperglycemic non-ketotic coma, whereas in ketoacidotic coma is life-saving treatment with alkalising solutions such as sodium bicarbonate.

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