EFFECT OF ADRENALIN ON HEMOGLOBIN METABOLISM

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The author's observations on hemoglobin metabolism in children with infectious diseases (scarlet fever, measles) showed that the autonomic nervous system plays an important role in the regulation of this type of metabolism. For instance, in the case of predominance of sympathetic function with an increase in the blood level of sympathins, the intensity of hemoglobin breakdown and synthesis is increased. Conversely, when excitation of the parasympathetic nervous system is predominant, accompanied by an increased blood level of acetylcholine, the intensity of hemoglobin breakdown and synthesis is lowered [7, 8, 10, 11, 18-20].

The object of the present investigation was to make an experimental study of the effect of adrenalin (as a sympathomimetic substance) on the breakdown of hemoglobin and the intensity of erythropoiesis.

EXPERIMENTAL METHOD

Ten chronic experiments were performed on 7 dogs. The intensity of hemoglobin breakdown was determined on the basis of the total 24-hour excretion of pigments (stercobilin, stercobilinogen, urobilin, urobilinogen) which are the end products of breakdown of the pigment moiety of hemoglobin. The excretion of pigment was determined in 24-hour samples of urine and feces by Tervain's method. The intensity of erythropoiesis was judged by the reticulocyte count and hemoglobin level of the blood. Adrenalin was injected intramuscularly as a 0.1% solution in a dose of between 0.13 and 0.6 ml/kg body weight for 2-3 days, and in one case for 16 days. During the experiment the dogs were kept in metabolic cages, and the feces and urine were collected daily from each animal separately. The excretion of pigments was investigated from 3 to 5 days before the experiment began, and for 10 to 40 days after administration of the adrenalin. Blood for testing was taken form the ear twice or three times in the initial state, during the injection of adrenalin, and several times after its administration had been discontinued.

EXPERIMENTAL RESULTS

The experiments showed that adrenalin has a marked effect on the indices of hemoglobin metabolism, and the nature of the effect is largely dependent on the dose of adrenalin given. With small doses of adrenalin (0.13-0.18 ml/kg body weight) the 24-hour excretion of pigment rose (by 50-100%) temporarily, indicating stimulation of hemoglobin breakdown. Usually this was followed by stimulation of erythropoiesis, indicated by an increase in the reticulocyte count (by 6-11.5%) and a tendency for the hemoglobin concentration to increase (by 2-16 Sahli scale divisions). The prolonged administration of small doses of adrenalin was accompanied by a fluctuating increase in the excretion of pigments, with an increase in the reticulocyte count and in the hemoglobin concentration.

With large doses of adrenalin (0.4-0.6 ml/kg body weight) the picture was different, and after injection of adrenalin the increase in hemoglobin breakdown was more marked and the excretion of pigment rose to 4-6 times its normal level. Soon after this increase in the intensity of hemoglobin breakdown, stimulation of erythropoiesis usually occurred, with the reticulocyte count rising by 6-22.5%. However, this stimulation of erythropoiesis and hemoglobin formation did not compensate for the loss of hemoglobin resulting from the massive breakdown, and its level fell by 8-16 Sahli scale divisions. Some dogs developed abcesses at the site of injection of the adrenalin. The addition of the abscess caused an even sharper increase in the intensity of hemoglobin breakdown (by 8-10 times) and a sharper fall in the hemoglobin level (by 24-28 Sahli divisions).

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Adrenalin thus stimulates the breakdown and synthesis of hemoglobin, but the hemoglobin level changes in different ways depending on the dose of adrenalin injected with small doses it rises but with large doses it falls. The experiment showed that the intensity of breakdown is modified first, and stimulation of erythropoiesis develops later. The close connection between the processes of hemoglobin breakdown and synthesis and the precedence of the changes in the intensity of breakdown, followed by the stimulation of erythropoiesis, suggest that certain breakdown products of hemoglobin itself may activate erythropoiesis. This hypothesis is not new, for it has been put forward by several writers [1, 2, 12, 16]. The possibility is not ruled out that under the influence of hemoglobin breakdown products the production of hemopoietins is increased. Reports have been published [5, 6, 17] that hypoxia, blood loss, and increased hemolysis are accompanied by increased production of hemopoietins, and the author himself has shown that after blood loss hemoglobin breakdown is temporarily stimulated, leading to activation of erythropoiesis. Consequently, the increase in the blood level of erythropoietins and the stimulation of erythropoiesis are preceded by a transient increase in the intensity of hemoglobin breakdown.

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