ABSTRACTS

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COBALT

STUDIES ON THE NATURE OF OXYGEN-CARRIER COBALT-HISTIDINE COMPLEX BY RADIOACTIVE COBALT (Co⁶⁰). Toshio Tanizuchi and Iwao Yamada. From the Faculty of Medicine, Department of Physiology, Hokkaido University, Sapporo. Medicine and Biology (Japan) 28: 42-44, 1953.

Cobaltous chloride and cobalt-histidine complex, both containing Co⁶⁰, were injected into the femoral artery of mice placed under low pressure at 200 mm. Hg. CoCl₂ was given to ten of the mice and cobalt-histidine complex to the other ten. The group injected with cobalt-histidine complex survived by some days longer than the other group. The distribution of Co⁶⁰ in the dead bodies was studied. In the case of the mice injected with the complex, different from the other case, the plasma contained the greatest amount of the complex, and the remainder was almost evenly distributed through the various organs. The amount of the complex was measured by tracing Co⁶⁰. The complex was identified by inspecting the extract of each organ with paper chromatography. The following may be concluded from the above experiment: the reason why the mice injected with the complex survived longer than the others was that more of the complex was distributed in plasma and carried oxygen throughout the bodies as hemoglobin might do.—K.M.


Co⁶⁰ was subcutaneously injected to mice, 0.2 μe per Gm. of body weight. Its distribution in the body was studied, and further the change of the distribution in the case of anemia by splenectomy or after the injection of vitamin B₁₂ was looked into.

(1) Within 24 hours Co⁶⁰ was rapidly excreted from the body. As for the distribution of Co⁶⁰ in the various organs, the liver contained the greatest amount, and the kidney, small intestines, adrenal glands, heart muscle and blood less in order. Almost the same amount was found in the blood, spleen, lungs, thymus, thyroid gland, bone-marrow. The brain contained the least.

(2) There was no tendency observed in Co⁶⁰ itself that it would go into hematopoietic organs. Co⁶⁰ in the blood did not enter the blood cells either.

(3) The above distribution remained the same when B₁₂ was mixed with the Co⁶⁰ and subcutaneously injected.

(4) When the mice had been fed only with corn, the experiments with it indicated that the percentage of Co⁶⁰ in the blood was a little greater than that in the normal animals.

(5) When splenectomized mice were used for the experiment, excretion of Co⁶⁰ by the liver and kidney was much slower than usual. The greater amount of Co⁶⁰ was in the adrenal gland and tended to stay there longer when B₁₂ was injected together with Co⁶⁰.—K.M.
ABSTRACTS

THE RELATIONSHIP OF COPPER, COBALT AND OTHER TRACE ELEMENTS TO HEMOPOIESIS.

George E. Cartwright. From the Department of Medicine, College of Medicine, University of Utah, Salt Lake City, Utah. Am. J. Clin. Nutrition 8: 11, 1955.

In this paper, which was presented as a part of a symposium entitled, "Nutritional Aspects of Blood Formation," is a concise presentation of experimental data obtained during the last eight years regarding the roles of certain trace elements in hemopoiesis.

Copper. In swine, the anemia of copper deficiency morphologically resembles that of iron deficiency. It is characterized by hypochromia and microcytosis and there is normoblastic hyperplasia of the bone marrow. Absorption of iron from the intestine is decreased, there is hypoferrremia and the level of plasma iron-binding protein is increased. In copper deficiency the anemia is characterized by a decreased red cell life span as well as a decreased rate of production of erythrocytes. The various copper-containing proteins and enzymes are listed, and their functions are discussed.

Cobalt. The normal adult consumes about 5 to 8 µg. of cobalt per day. About 77 per cent is absorbed and about 67 per cent is excreted in the urine each day. Thus, it is possible to maintain a positive balance on as little as 5 µg. per day and the actual requirements may be less. A deficiency of cobalt results in anemia, but the administration of moderate amounts of cobalt induces a true polycythemia and the administration of massive quantities of cobalt depresses erythropoiesis. The administration of appropriate amounts of cobalt is effective in the correction of the anemia associated with such varied abnormalities as a low protein diet, hypophysectomy, benzene administration and inflammation. The mechanism whereby cobalt induces these changes is unknown. One well known function of cobalt is that it is a constituent of vitamin B₁₂. It would appear that the prolonged administration of cobalt may have undesirable and serious side effects, such as nausea, anorexia, tinnitus, substernal pain as well as thyroid hyperplasia associated with hypothyroidism and even sudden death.

Zinc. Zinc deficiency has been difficult to induce in experimental animals. It is part of the enzyme carbonic anhydrase which is present in human erythrocytes and leukocytes.

Molybdenum. Administration of this substance to sheep and cattle produces a picture not unlike that of copper deficiency. Further evidence is necessary to evaluate the claim that the combination of molybdenum and iron is more effective in the therapy of iron deficiency anemia than is iron alone.

It is appropriately and emphatically pointed out that there is a great difference between the physiologic importance of a trace element and the importance of the same element as a dietary supplement. Although all of the above-mentioned elements have extremely important roles in hemopoiesis, this does not dictate a necessity for the administration of these elements in the form of dietary supplements.—W.N.J.


The pharmacologic and biologic actions of cobalt are discussed. There is a review of the literature and personal investigations are referred to. Emphasis is placed on the antianemic action, the vasodilator effect and the activity against tuberculous infection.—M.A.J.


This is an extensive review about cobalt and its distribution in animal and vegetable tissues. Emphasis is placed on the metallo complexes, such as the cobalt-insulin complex and the cobalt-protein tissue complexes studied by Benedicenti and the author. In 1928 the author presented the hypothesis that cobalt could substitute for iron in the hemoglobin molecule, an idea developed by Weisbecker (1949) who demonstrated in erythrocytes by spectrophotometric analysis the "existence of a Co-hemoglobin." The complexes of cobalt with penicillin and other antibiotics seem to have increased antibacterial activity. The diuretic action of cobalt is discussed, with Mascherpa referring to his first studies in 1929.—M.A.J.

Four children with sickle cell anemia and one adult with uremia due to renal amyloidosis were given enteric coated cobaltous chloride in doses of from 2.8 to 3.9 mg/Kg/day for from 3 to 7½ months. During therapy all of these patients developed definite thyroid enlargement. One case was not adequately followed, but in the other four this disappeared when cobalt was withdrawn. It is interesting that 3 of the cases of sickle cell anemia were aged 6, 3 and 4, which is quite young for the development of severe difficulty due to this disease. Two of them were not very anemic to begin with. Changes in PBI, cholesterol and 131 uptake were followed and tended to support the thesis of depressed thyroid function.

It is concluded that cobalt is a goitrogenic agent. The evidence seems fairly good that this is true, although what appears to this reviewer as the atypical nature of some of the cases is a matter of some concern.—T.R.T.


Four children with sickle cell anemia were given cobaltous chloride for several months. The initial satisfactory hemopoietic response could not be sustained with prolonged therapy and hemoglobin concentration dropped below pre-treatment values. All three children in whom thyroid function was evaluated developed enlargement of the thyroid gland. One developed severe myxedema and two showed laboratory evidence of depressed thyroid function. Thyroid biopsies in two patients showed extreme hyperplasia. Reversal to normal of these clinical and laboratory findings was accomplished when cobalt was withdrawn. Reversible thyroid enlargement with cobalt therapy in two premature infants and in one child with renal disease had been communicated to the authors by two other physicians. Experiments are cited showing inhibition by cobalt of the tyrosine iodinase system in tissue homogenates and in surviving rat thyroid slices. In view of these findings, authors condemn the indiscriminate use of cobalt as a hematinic.—A.G.M.


Histopathologic examinations were made of the heart, lungs, liver, kidney, spleen, pancreas, salivary glands, testicle, epididymis, bone and bone marrow of rats that had received cobaltous chloride in doses of 0.5 to 2.0 mg. per day over a 32-week period. The thyroid was not examined.

There were no significant degenerative changes noted in the parenchymal organs. Marked erythropoietic hyperplasia was observed in the bone marrow, but vascular changes in bone marrow which would provide a morphologic basis for marrow hypoxia were not observed.

In the spleen, the borders of the malpighian corpuscles were poorly defined and the "hematopoietic perifollicular envelope" which is normally present in the rat spleen was absent. The authors cite investigations demonstrating inhibition of enzyme systems involved in oxygen transport such as succinic dehydrogenase, choline oxidase, catalase and cytochrome oxidase to explain the action of cobalt.—W.N.J.

METHEMOGLOBIN FORMATION IN HUMAN BLOOD BY COBALT IN VITRO. Shu Chu Shen and A. B. Ley. J. Clin. Investigation 35: 1560-1566, 1954. From the Cancer Research and Cancer Control Unit of the Department of Surgery (Holy Ghost Division Cambridge) and the Department of Medicine, Tufts College Medical School, and the Thorndike Memorial Laboratory, Second and Fourth Medical Services (Harvard), Boston City Hospital, and the Department of Medicine, Harvard Medical School.
Authors studied the effect of cobalt on enzyme systems concerned with methemoglobin reduction. When normal blood was incubated at 37°C for 24 hours in the presence of 0.2 percent cobaltous chloride, formation of methemoglobin was observed. P-aminophenol and methylene blue formed methemoglobin readily only in the presence of oxygen while cobalt allowed methemoglobin formation in the absence of oxygen. In contrast to its suppressive effect on methemoglobin formation in blood containing p-aminophenol, methylene blue did not inhibit methemoglobin production in blood incubated with cobalt. Ascorbic acid suppressed methemoglobin formation by either cobalt or p-aminophenol. Cobalt failed to enhance methemoglobin formation when a pure hemoglobin solution was incubated. The results suggest that cobalt inhibits an intracellular reduction system which maintains hemoglobin in its ferrous state. Cobalt polycythemia is not associated with methemoglobinemia nor with diminished oxygen capacity of the blood. The physiologic relevance of these experiments, therefore, is not direct. It is suggested by analogy that cobalt may interfere with other enzymatic reduction systems and thus induce tissue anoxia which would stimulate erythropoiesis.—A.G.M.

**VITAMIN B12, FOLIC ACID, COBALT AND REDUCING SYSTEMS IN HUMAN ERYTHROCYTES.**
_E. Ambs, W. Künzer and D. Schneider._ From Universitäts Kinderklinik, Würzburg, Germany.

The influence of vitamin B12, folic acid and cobalt on the reducing enzyme systems of human red blood cells has been investigated. The reduction of methemoglobin of erythrocytes poisoned with nitrite was used as an indicator.

In the concentrations used—gammas of B12 and cobalt, mg. of folic acid—these substances showed a slightly stimulating influence upon the reducing systems.—M.H.H.


The experimental polycythemia produced by chronic cobalt administration is the result of an increase in the rate of red cell formation, or an increase in the average life span of the erythrocytes, or both. A number of studies seem to indicate that cobalt acts as a direct stimulant to erythropoieses but information as to whether or not cobalt has an effect on the life span of the red cells has been lacking. The authors administered labeled glycine to groups of normal rats and to rats made polycythemic by chronic cobalt injections. In both groups the average life span was found to be approximately 53 days. It is of interest that the rate of glycine incorporation into hemoglobin was significantly greater in polycythemic animals than it was in the normal controls.—R.S.


Red cell survival was determined by the C14 glycine technic in four Peruvian natives living at 12,000 feet altitude, with polycythemia of altitude. A normal erythrocyte life span ranging between 109-117 days was found. This rules out prolongation of red cell life as an explanation for secondary polycythemia.——A.G.M.


A cobalt-chlorophyllin compound was tested in a series of 74 patients with tumor and infection anemias refractory to iron. The daily application of 0.2-0.4 mg. over weeks resulted in an increase of the red cell count (more than 1 million) in 36 cases, with equal distribution in both groups.—M.H.H.

A 49 year old female patient had a hemoglobin level of 4.5 Gm. per 100 ml., red cells 1,250,000, reticulocytes 0.1 per cent, white cells 7,600 (69 per cent polymorphs), platelets 350,000. The sternal marrow was cellular, red cell precursors representing only 4 per cent of the total marrow cells. These were proerythroblasts or early normoblasts. Granulocytepoiesis and platelet development appeared normal. A diagnosis of pure red cell aplasia was made, and for a year treatment was by blood transfusion, supplemented by various hematines and by corticotrophin and cortisone. Splenectomy caused improvement and a return of the marrow to normal. A fall in the hemoglobin and red cell levels occurred however. Treatment with cobalt chloride in a dosage of 50 mg. twice daily was followed by a hematologic remission and for seven months this has been maintained without further treatment.—R.H.G.


This article is a detailed presentation of two cases and a brief review from the literature of 10 other cases. The authors feel that the term, "acquired erythrocytic hypoplasia," is a clinical entity characterized by (1) chronic anemia and reticulocytopenia, (2) normal differential and total leukocyte counts, (3) normal platelets and no hemorrhagic phenomena, (4) cellular marrow with normally active leukopoiesis and thrombocytopoiesis but marked hypoplasia to virtual absence of nucleated erythrocytes, (5) no extramedullary hematopoiesis. One patient of this sort was thought to have recovered due to cobalt therapy. The authors admit that a single case might be a coincidence.—T.R.T.


It had previously been found by Ingbar and co-workers that a sharp increase in reduced glutathione (GSH) of erythrocytes from sickle cell anemia patients occurred as the O2 tension was decreased, which also corresponds to the development of sickling. Assuming that cobalt would bind sulfhydryl groups, especially GSH, the authors undertook the treatment of ten patients who had sickle cell anemia, with cobaltous chloride. The dose varied from 40 to 180 mg. per day over periods of from 2 to 4½ months. The authors felt that no benefit derived from this therapy and had some evidence to suggest that the patients might have been made worse by it.—T.R.T.

IRON


Iron kinetics studies on 5 patients with polycythemia and 2 normal subjects are presented. Analysis of the plasma iron turnover rates shows that at least two exponential functions are present. Consequently a two-pool model is postulated as a means for the analysis of experimental data. Comparisons of the results obtained from the one-pool and the two-pool models are presented. In normal individuals similar results are obtained, but in patients with polycythemia vera, the two-pool model indicates a plasma iron turnover and a renewal of hemoglobin iron of much less magnitude than does the one-pool model.—W. N. J.
INFLUENCE OF THE GASTRIC JUICE ON TISSUE METABOLISM. G. Dominici, G. Oliva and C. Trarrontana. From the Medical Clinic of the University of Perugia, Italy. Lancet, 2: 1105-1106, 1954.

Iron takes part in complex biological reactions closely connected with the function of the digestive tract. Gastric juice was collected from an unspecified number of persons; some were healthy; some had hypochromic achlorhydric anemia, pernicious anemia, achlorhydria without anemia, hyperchromic hypersideremic anemia of pregnancy, hypoplastic anemia or granulocytopenia. The gastric juice in 5 ml. volume was injected intramuscularly into healthy persons and into others with hypochromic achlorhydric anemia, pernicious anemia or achlorhydria without anemia. This resulted in a lowering of the levels of blood sugar, serum iron and serum amino acids except when the gastric juice had been obtained from patients with hypochromic anemia and achlorhydria.

It is considered that a factor is present normally in the gastric juice that plays some part in body metabolism; so far it has been shown to be absent only from those with idiopathic hypochromic anemia and achlorhydria. The various clinical features usually considered to be due to iron deficiency are claimed to be due to deficiency of this gastric-juice factor. The term “dysenostomia” is suggested for the basic disturbance.—R.H.G.


Fe59Cl2 was administered to normal male albino rats by gavage, subcutaneously, intravenously, and intraperitoneally. The content of radioiron was then measured in lymph obtained by thoracic duct cannulation. It was determined that the small amount of iron which reaches the lymph after gavage is derived from the blood stream. Subcutaneously administered Fe59Cl2 is absorbed via the blood vessels, whereas subcutaneous protein-bound Fe59Cl2 passes almost exclusively into the lymphatics. Intravenously administered radioiron appears rapidly in the lymph. Leukocytes and lymph have a negligible role in absorption of radioiron regardless of the method of iron administration.—W.N.J.


Two series of observations were made on the hemoglobin concentration in the blood of male and of female University students. In both series there was a highly significant difference between the mean hemoglobin levels in the two series. The mean value for 24 men was 15.92 ± 0.23 Gm. Hb./100 ml. blood and for 41 women, 14.20 ± 0.13 Gm. In the second series the figure for 54 men was 16.21 ± 0.11 and for 55 women, 14.50 ± 0.10. In the first series the women were given 7 mg. of iron as ferrous sulphate for five months, and although there was an increase in the hemoglobin concentration, this also occurred in the men who received no supplement. In the second series a more detailed experiment was carried out and it was found that both men and women receiving ferrous sulphate tablets showed an increase in hemoglobin concentration compared with control groups, but that at the end of the experiment there was still a difference of about 1.5 Gm. Hb./100 ml. between the hemoglobin levels of treated males and treated females.—R.H.G.


In healthy young women the hemoglobin value of venous blood was found to vary from day to day by as much as 3.0 Gm. per 100 ml. The mechanism of these variations was not investigated, but it was thought that they were due to either fluctuations in the plasma

The values for serum iron and iron-binding capacity in nine cases of hemochromatosis, from some of their relatives, and from seventeen cases of anemia of varying etiology are given. It is shown that the percentage saturation value is more consistently characteristic of conditions affecting iron metabolism than the serum iron value alone.—G.C. de G.

STUDIES ON SERUM IRON AND IRON BINDING CAPACITY OF SERUM IN NORMAL AND ANEMIC SUBJECTS. II. OBSERVATIONS ON THE LEVEL OF SERUM IRON IN PATHOLOGICAL STATES. S. C. Agarwal and S. S. Misra. From the Department of Medicine, King George's Medical College, Lucknow, India. Jour. Assoc. of Physicians of India, 2: 1-8, 1954.

Employing the method of Kitzes and Elvehjem, serum iron level was determined in 65 patients suffering from different diseases. Hypoferremia was observed in chronic infection, malignant neoplasm and iron deficiency anemia.—J.B.C.

STUDIES ON SERUM IRON AND IRON BINDING CAPACITY OF SERUM IN NORMAL AND ANEMIC SUBJECTS. III. OBSERVATIONS ON THE LEVEL OF SERUM IRON, UNSATURATED IRON BINDING CAPACITY, TOTAL IRON BINDING CAPACITY AND SATURATION PERCENTAGE IN NORMAL INDIVIDUALS. S. C. Agarwal and S. S. Misra. From the Department of Medicine, King George's Medical College, Lucknow, India. Jour. Assoc. of Physicians of India, 2: 9-12, 1954.

The mean values of serum iron, total iron binding capacity of serum and saturation percentage as determined on 31 normal Indian males were 121 γ (range 75-225), 328 γ (range 235-444) and 37 per cent (range 17-48) respectively.—J.B.C.


A dextran-iron preparation produced by a commercial firm for intramuscular injection was given to 40 cases of iron deficiency anemia. Before this, experiments on 10 volunteers showed that discomfort lasting 4 to 12 hours occurred and was similar to that produced by other intramuscular injections. Skin staining occurred but not if a needle 2½ inches long was employed and a Z-shaped track of the needle used. In both normal persons and anemic patients given a single intramuscular injection of 4 or 5 ml., serum iron levels reached a variable peak in one or two days and returned to normal levels in six or seven days. The urinary excretion of iron was not increased. A mean calculation from the entire series showed that 100 mg. of iron was required to raise the Hb. 0.34 Gm. per 100 ml. The time taken to achieve the maximum hemoglobin response was 4-9 weeks. There were no toxic reactions although the serum iron reached levels as high as 13.8 mg. [sic] per 100 ml.—R.H.G.


The dextran-iron complex "Imferon" which contains 50 mg. of iron per ml. was given intramuscularly to 68 pregnant women followed for six weeks or more and to 13 women delivered within six weeks of the commencement of therapy. The estimated average utilization of the complex was 0.20 Gm. of hemoglobin per 100 mg. of iron. Local and general reactions were mainly mild and infrequent, and of 225 patients treated with the new preparation, only one had a severe reaction. This consisted of vomiting followed by syncope. Mild general reactions occurred in about 20 per cent of all cases.—R.H.G.
ABSTRACTS


In two Liverpool hospitals between September 1948 and January 1954 there were obtained from the case records details of eleven cases of men with idiopathic hypochromic anemia. It is stated that there was no evidence of hemorrhage or of steatorrhea, but a fat balance test was done on only one patient. Detailed dietetic studies were not carried out. The patients responded to oral iron therapy and did not relapse thereafter. Nine of the patients were under the age of 25.—R.H.G.


In an aboriginal Malay boy with iron-deficiency anemia, one obvious cause for which was ancylostomiasis, the blood findings were Hb. 0.7 Gm./100 ml. by a cyanmethemoglobin method, red cells 1,000,000 per cu. mm., P.C.V. 7 per cent, M.C.V. 70 c, M.C.H.C. 10 per cent. The hair was dry, lusterless and rough, with a well marked brown color not normally found in his tribe, and this was of special interest. There was a good response to treatment with iron by the intravenous route and by mouth, followed by deworming.—R.H.G.


A 27 year old man suffering from endogenous hemochromatosis and chronic hepatitis was treated by repeated venesection. The patient had recently suffered an attack of acute infective hepatitis and it was difficult to assess the relative roles of hepatitis and hemochromatosis in the genesis of his cirrhosis; there was no evidence of diabetes. The removal of 25 liters of blood produced a calculated negative iron balance of 10 Gm. in one year and resulted in a return to normal of his serum iron level and of his saturated serum iron-binding protein. The plasma of the veneseected blood was returned to the patient. Neither anemia nor hypoproteinemia developed. The importance of serial determinations of serum iron content and latent iron-binding capacity in assessing depletion of body iron stores in stressed —G.C. de G.


Two patients were treated with intravenous Versenol for periods of 2 and 4 days respectively. The results were insignificant. The authors point out that phlebotomy is a much more effective means for the removal of iron from the body. Untoward reactions to the Versenol were not noted.—W.N.J.

METHODS

A NOTE ON THE DECREASE IN AVIDITY OF THE COOMBS REACTION BY GRADUAL ELUTION OF RH ANTIBODIES FROM THE SENSITISED CELLS. Jean Barrie, Vera L. Krieger Department of Pathology, Women's Hospital, Melbourne Victoria. M.J. Australia 1: 247 1954.

The authors show that the elution of Rh antibodies at 56 C. noted by other workers also takes place at lower temperatures, although more slowly. Therefore it is important to test the sensitized cells in either direct or indirect Coombs tests within a short time after the saline washing in order to avoid weakening of the reactions and sometimes even negative
results. If specimens for the direct Coombs test cannot be handled immediately, whole blood and not saline suspensions of the infant’s cells should be sent for examination.—G.C. de G.

**Papain Treated Red Cells in the Detection of Incomplete Antibodies. K. Goldsmith.**


Details are given of a method for preparing and using papain-treated red cells to detect incomplete antibodies. 61 sera containing rhesus antibody were examined: parallel titrations were done with red cells suspended in saline solution and in 20 per cent bovine albumin, by the Coombs antiglobulin technique and by trypsin and streptomyces albus techniques. The tube technique with papain-treated red cells was the most sensitive method. In 3,370 maternal sera examined, papain gave only 11 inconstantly false positive results and no false negatives. It is believed that most false positive results develop as a result of cooling of the cell serum mixture. During tests with papain-treated red cells in other blood-group systems enhancement of agglutination was noted with Anti-A, Anti-B, Anti-M, Anti-N, Anti-Le a and Anti-Le b. No agglutination took place with Anti-Fy a.—R.H.G.


It is demonstrated that anti-M and anti-N antibodies are thermostable at 70°C for 60 minutes. Previous studies demonstrated that such treatment of dilute immune rabbit serum results in the destruction of heterologous hemagglutinins for A, B and O human red cells. Simple absorption of this dilute, heated anti-M or anti-N serum with any A, B or O red cell of the heterologous M or N factor results in a specific antiseraum.—J.H.A.


The plasma expander Vinisil, a 3.5 per cent solution of polyvinyl-pyrrolidone in saline, is a suitable substitute for plasma or serum in making a diluting fluid for mixing small portions of fresh bone marrow either on a glass slide or within a capillary pipette. This method has also been used in the preparation of autoradiograms of isotopically labeled bone-marrow cells.—O.P.J.


The loss of water from leucocytes by osmosis during the drying of a blood smear is discussed. It is shown experimentally that this loss may produce an 18 per cent reduction in the mean diameter of lymphocytes in certain parts of a thin, normally dried blood smear, and a 9 per cent reduction in the mean diameter of neutrophilic cells. It is shown that this loss can be largely prevented by drying the smear rapidly, and it is suggested that investigation into the finer points of morphology of cells in blood or bone-marrow be carried out on smears that have been dried rapidly.—G.C. de G.