cal action. The basic experiment to support this idea is the beating of an isolated frog's heart when perfused with a certain solution of a mixture of salts, one of which has to be potassium chloride. The amount of potassium in this solution is so small that a simple calculation indicates that only about one potassium atom per hour radiates in the frog's heart. Since the whole experiment can be performed in a much shorter time, it is absurd to connect in this experiment the necessary presence of potassium with its radioactivity.[2]

Conclusion. The above remarks are meant to show that tracer experiments with radioactive elements, if carefully executed, give definitely reliable information about the behavior of the normal element. If unexpected results are found they should certainly not be ascribed to a difference in the properties of the radioactive isotope.

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PSEUDOMETHEMOGLOBIN AND ITS REACTION WITH CARBON MONOXIDE AFTER REDUCTION

IN a discussion in this journal[1] recently, E. Ammundsen published her observations on the presence in the blood of "a kind of hemoglobin" combining with carbon monoxide only after reduction. With reference to that substance the author emphasizes that "for the present its composition and the cause of its appearance will have to be looked upon as unknown." In this connection the following facts may be of interest.

The normal presence of substances in human and animal blood not identical with hemoglobin but combining with carbon monoxide in a similar way was described by Barkan and Berger[2] in 1928. These authors pointed out that of the "easily split-off" blood iron ("leicht abspaltbares" Bluteisen) discovered by Barkan[3] (1925) one portion E combines with O_2 and CO following the distribution equation, while another portion E′ does not react with those gases. In 1937 we[4] demonstrated that both fractions of the "easily split off" blood iron are pseudohemoglobins which are intermediates in the formation of bilirubin from hemoglobin and accompany the hemoglobin within the red cells of the circulating blood. Pseudohemoglobins, according to our definition, are chromoproteins in which the protein is probably an unaltered globin, while the prosthetic group is an iron-containing heme or hemin derivative with an opened porphyrin ring, similar to that in Lemberg's[5] "verdohemochromogen." The two fractions E and E′ differ by the valency of the iron. The latter is bivalent in *E*, which combines with oxygen and carbon monoxide and hence was designated as a pseudohemoglobin *in sensu strictiori*. The iron is trivalent in E′, which does not combine with O_2 and CO and was designated as a "pseudomethemoglobin." By reduction with $Na_2S_2O_4$ we were able to transform the pseudomethemoglobin into pseudohemoglobin, which does combine with carbon monoxide.

Our work was published in different original articles[4, 6] and, in addition, some reviews[7] have been given. Taylor and Coryell[8] in an article (1938) dealing with the magnetic susceptibility of the iron in ferrohemoglobin confirmed our work. Using the same method as was later used by E. Ammundsen,[1] they found too that in normal blood and in oxyhemoglobin solutions there are present hemoglobin-like compounds which combine with carbon monoxide only after previous reduction.

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AN EXPERIMENT ON RETINAL AFTER-IMAGE AND JUDGMENT OF SIZE

BY an almost unconscious combination of elements, we judge the size of an object from the image it produces on the retina and from our judgment of its distance from the eye. If there is uncertainty in this latter judgment, there is corresponding uncertainty in judgment of size because we do not then know how far away to consider the object to be and do not know how to interpret the retinal image. It is common experience that we are incapable of estimating the

[2] A. J. Glazko and D. M. Greenberg (*Am. Jour. of Physiol.*, 125: 405, 1939) have shown that radioactive sodium can not replace the potassium in this experiment. Compare also a critical article by S. G. Zondek, *Biochem. Zeits.*, 121: 76, 1921.

[1] SCIENCE, 90: 2338, 372–373, October 20, 1939.

[2] G. Barkan and E. Berger, *Arch. exp. Pathol. u. Pharmakol.*, 136: 278–299, 1928; *Klin. Wochenschr.*, 7: 1868, 1929.

[3] G. Barkan, *Zeits. physiol. Chem.*, 148: 124–154, 1925; *ibid.*, 171: 179–193 and 194–221, 1927; *Biochem. Zeits.*, 224: 53–62, 1930; *Zeits. physiol. Chem.*, 216: 1–16 and 17–25, 1933; *ibid.*, 221: 241–251, 1933; *ibid.*, 236: 198–200, 1935. See also G. Barkan and O. Schales, *ibid.*, 244: 81–88, 1936; *ibid.*, 244: 257–265, 1936.

[4] G. Barkan and O. Schales, *Zeits. physiol. Chem.*, 248: 96–116, 1937.

[5] *Cf.* R. Lemberg, *Ann. Rev. Biochem.*, 7: 421–448, 1938; and original papers cited therein.

[6] G. Barkan and O. Schales, *Zeits. physiol. Chem.*, 253: 83–103, 1938.

[7] G. Barkan, Lecture in the Wiener Biologische Gesellschaft, June 14, 1937; *Klin. Wochenschr.*, 16: 1265–1268, 1937; *Dtsch. Mediz. Wochenschr.*, 64: 638–640, 1938; Kongressbericht II des XVI Internat. Physiol. Kongresses, Zürich (Schweiz), 1938, p. 250; G. Barkan and O. Schales, *Arch. Ital. di Sc. Farmacologiche*, Vol. Jubil. ad Benedicenti, 1937; O. Schales, Research Meeting of the Rockefeller Institute, Copenhagen, December 3, 1938.

[8] D. S. Taylor and Ch. D. Coryell, *Jour. Am. Chem. Soc.*, 60: 1177–1181, 1938.