green shade of fluorescence is due mainly, but not entirely, to the contained bacilli. It is possible that the method devised for separating bacilli from the lesions and for collecting them in large numbers, which we have found useful in rats, may be helpful when extended to leprous lesions of humans.

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Influence of Anoxia on Glycogenolytic Action of Adrenalin.*

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In the course of a study of the interaction of hypoglycemia and anoxia in the rabbit[1] it was found that a short period of anoxia (7% oxygen for 15 minutes) enhances the return of the blood sugar to the control level, whereas the inhalation of 7% oxygen for 2 hours greatly aggravates the hypoglycemia. In spite of the fact that in the latter group the blood sugar averaged less than 30 mg % and was maintained at this level for 2 hours, no convulsions occurred, thus confirming McQuarrie and Ziegler's[2] experiments. The question was studied whether the differential reaction of the blood sugar to anoxia in the 2 groups of experiments is related to the effect of adrenalin on the liver. If this were the case a greater hyperglycemic effect of adrenalin would be expected after a short period of anoxia than is observed under control conditions. Furthermore, prolonged periods of anoxia should lead to a diminished glycogenolytic response of the liver.

Fifty-six experiments were carried out on rabbits which were *starved* for 18 hours and injected with adrenalin 1.9 γ/kilo 3 times in intervals of 10 minutes. The maximum rise of the blood sugar averaged 49% in this control group. The reaction was twice as great (average rise 97.9%) when this adrenalin experiment was repeated during the last half hour of an hour experiment in which the rabbits inhaled 7% oxygen. If, however, this period of anoxia was more prolonged (2 hours) and the same amount of adrenalin was

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1 Coffee, Ann, and Gellhorn, E., *Proc. Am. Physiol. Soc.*, Toronto, 1939, p. 51.

2 McQuarrie, I., and Ziegler, M. R., PROC. SOC. EXP. BIOL. AND MED., 1938, **39**, 525.

administered during the last half hour of inhalation of 7% oxygen it no longer elicited a hyperglycemia (average rise of blood sugar 3%). It is interesting to note that even after readmission of air the glycogenolytic action of adrenalin remains reduced for considerable periods of time. Eight experiments were carried out in which adrenalin was reinjected ½, 1, 1½, and 4 hours, respectively, after the end of the anoxia period. In only one experiment was the response normal, but was greatly reduced in the other experiments. The average rise in blood sugar after adrenalin was 17.6%, as compared with 49% under control conditions.

Further studies showed that adrenalin loses its glycogenolytic effect on the liver during prolonged anoxia in spite of the fact that the liver still contains considerable amounts of glycogen. Seven experiments were performed in which adrenalin was given during the last half hour of a 2-hour period of 7% O_2 inhalation producing a rise of blood sugar of only 4.2% on the average, although the average glycogen content of the liver determined immediately at the end of the anoxia period was 3.3%. It seems to be of principal interest to state that an anaerobic reaction (glycogenolysis produced by adrenalin) is greatly modified by a moderate degree of anoxia. The influence of anoxia on the effect of other glycogenolytic factors is being studied at the present time.

Conclusions. 1. Short periods of moderate anoxia (7% oxygen) act antagonistically to insulin hypoglycemia, whereas prolonged periods (2 hours) aggravate hypoglycemia. 2. The glycogenolytic action of adrenalin is increased during a short period of anoxia but greatly decreased or lost completely after a prolonged period of anoxia. 3. This loss of the glycogenolytic effect of adrenalin on the liver is not due to a depletion of the glycogen reserves of the liver.