of the book is the chapter dealing with the effects of anoxia on the digestive tract. The author himself and his co-workers have studied this subject quite intensively over a period of years.

The remaining chapters deal with the secretion of urine, the endocrine glands, metabolism, heat regulation, nutrition, water distribution in the body and the nervous system.

In the last chapter the author discusses the physiological effects of anoxia on the nervous tissue, its circulation and function. This is followed, without adequate integration, by a discussion of the psychological effects of anoxia. This section, as the author admits, is especially lacking in detail and completeness of bibliography. The most interesting observations in this field relate to the striking and insidious changes in behavior produced by a lack of oxygen to the nervous tissue. The author fails to interpret the intimate relationship which must exist between an adequate and constant supply of oxygen to the nervous tissue and mental functions, such as memory, judgment and reasoning, which are essentially psychological in nature. This is important, not only from the point of view of research relating to the possible cause of certain mental disorders, but also in an understanding of the very foundation of mental processes or behavior in general.

The discussion of the visual effects is particularly sketchy and incomplete. Little attempt is made to interpret the role of these findings in an understanding of the nature of the visual mechanism itself. Several errors in this last chapter might be pointed out. Psychological tests, such as speed of apprehension, memory tests and repetition of auditory patterns, commonly termed mental tests, are erroneously referred to as "psycho-somatic tests" (p. 234). In reporting the various studies on sensory function, the expression "decrease in threshold" is used where "increase" is obviously meant (pp. 244, 246).

The author might have emphasized more clearly that the great variations found in the responses and adaptive processes to anoxia are not only related to the type of stimulus (acute, intermittent, chronic) but are also attributable in great part to the different *degrees* of anoxia used in experiments and found at different levels of altitude.

The chief criticism concerning the style in which this monograph was written is that frequently mere statements of experimental results are given, without mention of the methods employed by the investigators, or critical appraisal of other factors which might be of value in judging their validity. This deficiency is noted particularly when opposing experimental results are cited. More interpretation and integration of the findings of the various investigators would have improved this monograph.

For the reader who is not familiar with the literature, this monograph provides a valuable summary. It is, however, not as complete in its bibliography as might be desired. A number of important studies are omitted which would have assisted the author in interpreting several controversial topics. In the section on hematology, for example, the important paper by Talbott (1936) from the International High Altitude Expedition is not mentioned. Also, a great part of the work done in Peru has been neglected. It seems regrettable that Hoff and Fulton's more complete and excellent "Bibliography of Aviation Medicine," also published in 1942, was not available to Dr. Van Liere during his preparation of this book. Also the lack of an authors' index in a work of this kind is unfortunate.

The need for a current book reviewing the work done on this subject has been very great. In spite of the above criticisms, Dr. Van Liere's monograph serves as a useful introduction to the literature on the physiology of oxygen deficiency. It will be of value not only to research workers in physiology and psychology, but also to those concerned with the problems of high altitude in aviation. The clinician, as well, will find it of interest in view of the role of anoxia in various diseases.

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SPECIAL ARTICLES

ABOLISHMENT OF ALIMENTARY LIPEMIA FOLLOWING INJECTION OF HEPARIN

DURING the course of determining red cell circulating mass in dogs by the donor-isotope red cell procedure, an occasional animal of irregular eating habits showed a marked lipemia in the initial control blood sample. When injected with whole blood containing tagged cells, in the instances in which heparin was used as an anticoagulant, this lipemia had disappeared completely in the blood sample taken three to five minutes later. This phenomenon was so striking, even in the instances where the degree of lipemia was such that the plasma was suggestive of light cream, that it seemed advisable to determine how specific the reaction might be, ruling out any special donor-recipient peculiarities which might be responsible for the change.

In the first experiments the lipemia was due to the dilatory eating habits of the animals. Later, in order

to be able to depend on the presence of lipemia the food was not given in the afternoon as usual but reserved until morning, or sometimes an extra meal consisting of 100 gms each of salmon bread, Klim and cod liver oil was fed from two to four hours before sampling.

Both blood samples were taken from the same jugular vein, the needle being left in place. The injected material was also introduced through this needle. The degree of lipemia was not quantitated except roughly as follows: 0 – water clear plasma; 1 + – slight turbidity; 2 + definite lipemia; 3 + sufficient lipemia to obscure the meniscus of the plasma completely; 4 + – same as 3 + but with frank lipid layer on top the plasma layer.

In Table 1 is shown a series of reactions in which heparinized whole blood from a number of donor dogs was injected, as well as plasma from heparinized blood. Two makes of heparin were used, those experiments marked with * were carried out with a preparation obtained from the Hynson and Westcott Company of Baltimore, while those marked ** were done with material of considerably greater specific potency (1 mgm = 110 units) obtained from the Connaught Laboratories of Toronto. In all experiments in which heparinized whole blood or plasma was administered the lipemia was abolished. The reaction did not occur on the injection of washed red cells derived from heparinized blood nor after injection of any fractions derived from citrated blood.

It was finally found that the same amount of heparin (**2.4 mgm) as used in the earlier experiments when dissolved in saline and given by vein would in itself abolish the lipemia.

Addition of 5 mgm of heparin to 5 ml of lipemic plasma *in vitro* with mixing showed no reaction on standing. Neither did mixing of heparin with non-lipemic plasma and subsequent mixing with lipemic plasma result in the clearing of the latter.

The time interval of 5 minutes elapsing between the injection of the heparinized material and sampling is more than necessary for the reaction to occur. In three of the experiments shown in Table 1, samples were taken at ½, 1, 1.5, 2 and 3 minutes as well as 5 minutes. In each of these instances the lipemia was practically absent in the recipient's blood at the end of 1 minute.

TABLE 1

LIPEMIA BEFORE AND AFTER ADMINISTRATION OF HEPARIN

Date	Dog		Material introduced	Degree of lipemia		Donor source
				Initial	Final	
1/8	40–115	*	20 ml heparinized whole blood	3 +	0	39–266
1/20	41–164	**	" " " "	3 +	0	1–J
1/19	1–J	**	" " " "	3 +	0	39–266
1/4	1–K	*	" " " "	3 +	0	39–266
1/8	1–K	*	" " " "	4 +	0	39–266
1/19	40–115	**	20 ml heparinized plasma	2 +	0	1–J
1/19	41–164	**	" " "	4 +	0–1 +	39–266
1/22	39–266	**	" " "	4 +	1 +	38–137
1/20	39–196	**	" " "	4 +	0–1 +	40–149
1/13	1–J		20 ml citrated whole blood	2 +	2 +	pooled
1/13	38–137		" " "	3 +	3 +	"
1/18	39–196		" " " "	3 +	3 +	39–266
1/13	40–115		25 ml citrated plasma	4 +	4 +	pooled
1/14	39–57		" " "	4 +	4 +	"
1/22	39–266	*	20 ml washed heparinized cells	4 +	4 +	38–137
1/19	40–149	*	" " " "	3 +	2 +	39–266
1/13	1–K		25 ml citrated washed cells	2 +	2 +	pooled
1/18	1–K		" " " "	4 +	4 +	"
1/23	40–115	**	20 ml citrated plasma (itself lipemic) to which was added 250 units of heparin	2 +	0	39–266
1/23	41–164	**	250 units of heparin in 5 ml saline	2 +	0	....
2/11	39–57	**	" " " " " " " "	4 +	0	....
1/28	40–115	**	" " " " " " " "	1 +	0	....
2/1	38–137	**	" " " " " " " "	3 +	0–1 +	....
2/11	39–266	**	" " " " " " " "	3–4 +	0	....

On a basis of the data presented here it would not be wise to speculate as to the nature of the mechanism involved in the abolishment of lipemia from the blood of dogs as a result of the injection of moderate amounts of heparin. Fractional lipid analyses of plasma taken before and after injection of heparin are being carried out and will be reported at a later date.

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PRODUCTION AND TREATMENT OF GRANULOCYTOPENIA AND ANEMIA IN RATS FED SULFONAMIDES IN PURIFIED DIETS

THE production of granulocytopenia and anemia in rats through the use of sulfanilylguanidine or succinyl sulfathiazole in purified diets and treatment of the animals with whole dried liver or liver fractions have been reported from this laboratory.[1] The pres-

[1] S. S. Spicer, F. S. Daft, W. H. Sebrell and L. L. Ashburn, *Pub. Health Rep.*, 57: 1559, 1942.