

THE OXYGEN CONTENT OF THE VENOUS BLOOD OF THE DOG AFTER UPPER GASTROINTESTINAL TRACT OBSTRUCTION.

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(Received for publication, July 14, 1927.)

The color of the venous blood of the dog after gastrointestinal tract obstruction is a good index of the degree of toxemia. With the characteristic rise in non-protein nitrogen the blood becomes increasingly darker in color. Cyanosis has also been recognized as one of the cardinal clinical signs of such toxemia in man. The change of color is due to the presence of an increased amount of reduced hemoglobin. To measure this change, determinations of the oxygen content and oxygen capacity of the venous blood of the dog have been made after experimental obstruction at different levels.

Method.

All operations were done under ether anesthesia with aseptic technique. Obstruction of the cardia and pylorus was accomplished by ligating with heavy tape. The intestine was obstructed by section of the upper end of the jejunum and inversion of the cut ends.

Blood for chemical analysis was withdrawn from the jugular vein. Specimens for the determination of oxygen content were collected under oil. The total oxygen capacity was determined on venous blood after thorough shaking with air. The oxygen saturation of arterial blood was determined on samples collected under oil from the femoral artery. All oxygen determinations were made by Van Slyke's technique (1). The non-protein nitrogen was determined by the method of Folin and Wu (2), and the chlorides on the tungstic acid filtrate after the method suggested by Gettler (3).

EXPERIMENTAL OBSERVATIONS.

After obstruction of the cardiac end of the stomach there is a rapid and marked fall in the oxygen content of the venous blood (Table I). Thus the oxygen saturation of the blood of Dog 2 before

operation was 54 per cent and on the 3rd day after obstruction was only 3 per cent. The findings after pyloric obstruction (Table II) are equally marked. There is also a decrease in oxygen saturation after intestinal obstruction although this is not so striking as with obstruction at higher levels.

TABLE I.
Oxygen Saturation of Venous Blood after Cardiac Obstruction.

Dog No.	Day after	Blood				Remarks
		Hema- to- crit reading (per cent of cells)	Oxygen saturation of venous blood	Chlorides	Non-pro- tein nitrogen	
		<i>per cent</i>	<i>per cent</i>	<i>mg.</i>	<i>mg.</i>	
1	0		56		36.1	
	1		9		41.6	
	2		14	395	89.8	O ₂ saturation of arterial blood, 95 per cent
2	0	49	54	430	47.6	
	1	50	26	460	46.0	
	2	51	46	430	65.5	
	3	54	3	460	101.0	
	3	35	56	600		Immediately after receiving
	4	42	35	590	107.0	50 cc. 10 per cent NaCl
3	0	51	57	460	44.4	
	1	55	33	410	60.0	
	2	60	30	390	45.6	
	3	54	33	370	54.5	
	4	52	41	360	118.0	
	5	52	7	350	183.0	
4	0	54	53	450	25.9	
	1	62	50	380	81.0	
	2	60	19	370	114.0	O ₂ saturation of arterial blood, 93 per cent

DISCUSSION.

The average oxygen saturation of the venous blood of 12 dogs before operation was 60.8 per cent and only 16.8 per cent at the end

of the experiment. This marked decrease in oxygen saturation must be due to incomplete oxidation in the lungs or to an increased reduction of normally saturated hemoglobin in the capillaries. The oxygen content of the arterial blood was determined a number of

TABLE II.
Oxygen Saturation of Venous Blood after Pyloric Obstruction.

Dog No.	Day after operation	Blood				Remarks
		Hema- to- crit reading (per cent of cells)	Oxygen saturation of venous blood	Chlorides	Non-pro- tein nitrogen	
		<i>per cent</i>	<i>per cent</i>	<i>mg.</i>	<i>mg.</i>	
5	0	55	50	475	40.0	
	1	60	14	440	71.3	
	2	53	5	390	198.0	
6	0	45	63	450	28.0	
	1	58	21	430	63.5	
	2		9	410	133.0	Obstruction released
	3	48	22	390	205.0	
	4	42	17	390	147.0	
	5		26	380		
7	0	48	46	450	36.1	
	1	53	8	440	27.0	O ₂ saturation arterial blood, 100 per cent
	2	54	37	410	48.7	
	3	61	22	340	167.0	
8	0	53	88	460	46.9	O ₂ saturation arterial blood, 95 per cent
	1	57	16	400	40.0	
	2	60	50	340	60.5	O ₂ saturation arterial blood, 99 per cent
	3	52	10	280		O ₂ saturation arterial blood, 88 per cent

times when the oxygen content of the venous blood was very low and found always within normal limits, showing there is no disturbance of oxidation in the lungs.

The increased deoxidation in the capillaries may be due to a slower

flow than normal. Such occurs after exercise if not compensated for by an increased rate of flow or in heart disease after decompensation (4). After upper gastrointestinal tract obstruction the viscosity of the blood is much increased (5), tending to slow the flow. Dehydration may also be a factor although the relative cell mass is little

TABLE III.
Oxygen Saturation of Venous Blood after Intestinal Obstruction.

Dog No.	Day after operation	Blood			
		Hematocrit reading (per cent of cells)	Oxygen saturation of venous blood	Chlorides	Non-protein nitrogen
		<i>per cent</i>	<i>per cent</i>	<i>mg.</i>	<i>mg.</i>
9	0	44	41	460	26.5
	1	51	57	460	33.0
	2	54	26	390	116.0
10	1	46	71	430	
	2	47	36	390	25.7
	3	49	30	300	31.9
	4	46	27	250	65.0
11	1	57	84	440	34.5
	3	60	62	350	41.4
	4	58	49	320	63.0
	5	58	61	300	122.0
	6	53	62	270	105.0
	8	55	60	235	126.0
	10	55	50	200	179.0
	13	53	23	190	234.0
12	1	51	66	450	46.8
	2	65	32	360	86.3

changed as shown by hematocrit readings. Keith (6) studied the oxygen saturation of the venous blood after experimental dehydration and found no marked decrease even with a very great increase in viscosity. The rate of flow through the capillary bed is much slowed also in shock.

It seems probable that each of the factors enumerated may play

a part in the decreased oxygen saturation. It is also possible that there may be present in the blood stream and body tissues active reducing bodies which are wholly or in part responsible for the greatly increased deoxidation. Thunberg (7) has demonstrated such reducing bodies in other conditions by their action on methylene blue. These, he thinks, are intermediate products of metabolism. In upper gastrointestinal tract obstruction there is much evidence to suggest the view that such reducing bodies are present. This evidence will be discussed fully in a later paper.

SUMMARY AND CONCLUSIONS.

There is a rapid fall in the oxygen content of the venous blood of the dog after upper gastrointestinal tract obstruction. This increased deoxidation is probably due to a combination of several factors.

There is much evidence to suggest that active reducing bodies are present in the blood.

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