

BLOOD FIBRIN IN UPPER GASTROINTESTINAL TRACT OBSTRUCTION.

BY RUSSELL L. HADEN, M.D., AND THOMAS G. ORR, M.D.

(From the University of Kansas School of Medicine, Kansas City, Kansas.)

(Received for publication, November 11, 1926.)

Foster and Whipple (1-4) have studied the blood fibrin in a number of experimental conditions. They have shown that fibrin is a very labile protein with wide fluctuations due to a variety of stimuli. The site of production is mainly the liver where there is a constant manufacture to supply the continuous utilization in the animal body. Injury to any tissue except the liver causes some increase in the fibrin content of the blood. If the liver is seriously injured a decrease is observed. With mild liver injury there is a rise instead of a fall.

Dead or injured tissue and non-specific inflammation seem the only factors needed to excite an increased production. It seems quite probable also that any cell injury even without inflammatory reaction will cause this characteristic rise. McLester (5) has attempted to utilize blood fibrin determinations as an index of liver function. Foster (6) made fibrin determinations in numerous clinical conditions. He found a marked elevation with the toxemia of pregnancy, the fibrin level running parallel with the severity of the symptoms. With nephritis no increase was observed. Liver atrophy showed a very low value and lobar pneumonia a very high value.

We have made a series of plasma fibrin determinations for comparison with data previously observed in the toxemia incident to upper gastrointestinal tract obstruction.

Methods.

All experiments were done on dogs. All operations were done under anesthesia with aseptic technique. The cardiac end of the stomach and pylorus were obstructed by ligation with tape. The jejunum was obstructed by severing the intestine and turning in the cut ends. The hematocrit reading was obtained by centrifuging 10 cc. of blood mixed with 2 cc. of 1.6 per cent sodium oxalate. The

fibrin determinations were made on this oxalated plasma by the method of Foster and Whipple (1). The non-protein nitrogen was determined by the method of Folin and Wu (7) and the chloride after the manner suggested by Gettler (8).

TABLE I.
Obstruction of Cardiac End of Stomach.

Dog No.	Day after operation	Hematocrit reading (per cent of cells)	Fibrin (mg. per 100 cc. plasma)	Blood (mg. per 100 cc.)	
				Non-protein nitrogen	Chlorides (as NaCl)
		<i>per cent</i>	<i>mg.</i>	<i>mg.</i>	<i>mg.</i>
11	0	55	605	47.6	530
	1	51	980	46.0	490
	2	51	1350	65.5	480
	3	54	1717	101.0	470
	4	43	1608	106.0	570
4	0	50	569	44.4	520
	1	56	1020	60.0	470
	2	61	1210	45.6	440
	3	52	1468	54.5	420
	4	50	1554	118.0	440
	5	55	1600	163.0	490
7	0	50	572	50.5	520
	1	63	972	28.9	490
	2	63	1006	78.0	500
	3	64	1414	130.0	520
8	0	58	665	28.9	470
	1	64	1170	31.9	450
	2	67	1679	82.5	440
	3	61	1828	139.0	400
12	0	54	520	37.3	480
	1	62	975	140.0	410
	2	60	1122	152.0	380

EXPERIMENTAL OBSERVATIONS.

Successive determinations of plasma fibrin after obstruction of the cardiac end of the stomach are shown in Table I. In each animal there is a marked increase, the fibrin being almost tripled. The fibrin

increase parallels quite closely the degree of toxemia as indicated by the level of the non-protein nitrogen of the blood.

TABLE II.
Obstruction of Pylorus.

Dog No.	Day after operation	Hematocrit reading (per cent of cells)	Fibrin (mg. per 100 cc. plasma)	Blood (mg. per 100 cc.)		Remarks
				Non-protein nitrogen	Chlorides (as NaCl)	
		<i>per cent</i>	<i>mg.</i>	<i>mg.</i>	<i>mg.</i>	
13	0	53	368	32.6	480	
	2	61	564	67.5	460	
	3	53	1721	139.0	440	
14	0	46	388	38.9	390	
	1	53	926	36.6	—	
	2	54	840	48.7	450	
	4	61	1212	167.0	420	
10	0	45	566	33.0	470	
	2	60	1380	68.3	420	Obstruction released
	4	48	1301	113.0	320	
	5	42	1148	91.0	440	
15	0	53	456	59.7	490	
	1	57	513	75.0	400	
	2	60	1326	70.0	360	“ “
	3	50	1348	107.0	330	
9	0	66	324	30.0	490	
	1	64	1109	30.3	420	
	2	64	1241	59.5	330	“ “
	3	58	1036	41.6	390	
	4	58	1062	41.0	380	
	5	69	766	32.6	380	
	6	57	756	38.9	390	
	7	45	751	31.9	400	
	8	43	719	27.0	400	
	9	40	677	35.3	390	
10	42	845	32.6	420		

With obstructions of the pylorus (Table II) the findings are quite similar. The increase here is even more marked and rapid. In three

animals the obstruction was released after onset of the toxemia. In two the fibrin level remained high and the animals died. One recovered. In this animal the fibrin fell but did not return to the normal level. In four dogs with the jejunum obstructed there was also an increase in the fibrin level. This was not so marked however as with

TABLE III.
Obstruction of Jejunum.

Dog No.	Day after operation	Hematocrit reading (per cent of cells)	Fibrin (mg. per 100 cc. plasma)	Blood (mg. per 100 cc.)	
				Non-protein nitrogen	Chlorides (as NaCl)
		<i>per cent</i>	<i>mg.</i>	<i>mg.</i>	<i>mg.</i>
16	0	44	686	26.5	460
	1	51	1238	33.0	460
	2	54	1358	116.0	390
17	0	47	489		430
	1	47	820	25.7	390
	2	49	1000	31.9	300
	3	46	681	65.0	250
18	0	50	670	46.8	450
	1	65	801	86.3	360
19	0	56	620	34.5	440
	1	60	813	41.4	350
	2	58	953	63.0	320
	3	58	905	12.2	320
	4	53	760	105.0	270
	6	55	650	126.0	220
	8	55	692	179.0	200
	10	53	725	234.0	180

obstruction of the cardia and pylorus. One animal (No. 19) showed an exceedingly low blood chloride and high non-protein nitrogen with no marked change in the fibrin.

DISCUSSION.

The very high blood fibrin level observed in this series of animals emphasizes the tissue injury occurring in such conditions. The rise

is especially striking after obstruction of the cardiac end of the stomach which we have shown is characterized by a toxemia more serious than that incident to pyloric or high intestinal obstruction.

These findings are of interest also in indicating that hepatic insufficiency is not the cause of death in high gastrointestinal tract obstruction as has been suggested (9). If there is any liver injury it must be mild since extensive liver injury without exception has been shown to give low values for fibrin.

SUMMARY.

After high gastrointestinal tract obstruction the blood fibrin increases rapidly.

The change is more marked in animals with obstruction of the cardiac end of the stomach.

The rise in fibrin parallels closely the toxemia characteristic of such conditions.

These results indicate that liver insufficiency cannot be the cause of death in such obstructions.

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