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FLUID ABSORPTION BY RED BLOOD CELLS AND HEMOLYSIS IN EXPERIMENTAL VENOUS STASIS*

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Absorption of fluid by red blood cells accompanied by an increase in their volume, fragility and hemolysis was reported by Rosenfeld (1) in dogs under shock by histamine, trypsin, peptone or bothropic venom. Since these substances, with exception of bothropic venon, do not produce any change of the red blood cells "in vitro", these modifications were considered by Rosenfeld as due to shock induced by the drugs and not as a direct effect of the drugs mentioned. If that is the case, then absorption of fluid by erythrocytes should be a constant finding in shock. In part it would also explain the decrease in plasma volume, known to occur in shock, which has so far been attributed to loss of blood plasma through the walls of the blood vessels.

Rosenfeld, Nahas, Schenberg and Beraldo (2) observed later the same phenomenon in tourniquet shock in dogs. As the alterations were the same and appeared even before arterial pressure fall, they seemed to be produced by venous stasis. In order to test these hypothesis, the modifications that occurred under experimental venous stasis were studied.

MATERIAL AND METHODS

A laparotomy was done to clamp the abdominal veins or to handle the viscerae of dogs maintained under Nembutal anesthesia (35 mg/kg). The carotid blood pressure of most animals was also recorded during the experimental period. From the exposed femoral veins, 5 ml of blood were collected at each time. To avoid clotting 0.1 mg of heparin per milliliter of blood were added. Red blood cell counts were done in 1/5 mm^2 after shaking mechanically blood

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and pipets (2). Wintrobe hematocrits were used and all were centrifuged for 15 minutes at 4,000 rpm. Hemolysis was estimated in the spectrophotometer from the hemoglobin concentration present in the supernatant plasma of hematocrit determination.

RESULTS

Clamping of the vena cava just below the renal veins provoked a gradual and slight fall of blood pressure (fig. 1) followed by hemoconcentration, mean corpuscular volume increase and hemolysis (table 1 and graph 1). On clamp release, after this clamp had acted for 50 minutes, a transitorial rise of blood pressure appeared but the hematological modifications remained.

Clamping of the mesenteric vein near to its junction in the vena cava produced a gradual and more marked fall of blood pressure (fig. 2) with the same blood alterations. On clamp removal, the modifications became more marked (table 2, graph 2). In a dog (fig. 3) there was very small pressure fall, although the same blood alterations were observed (table 3, graph 3).

Removal of viscera from the abdominal cavity and its handling also provoked pressure fall (fig. 4) and the same blood modifications (table 4, graph 4) which remained even after replacement of the viscerae and normalization of the blood pressure. It was observed that during this experiment stasis occurred in the mesenteric veins as shown by dilatation and cyanosis of the intestines.

Controls were done (fig. 5, tables 5 and 6, graphs 5 and 6) in order to find out if anesthesia or the incisions to expose femoral or carotid artery were sufficient to provoke alterations of the same kind. In these experiments hemoconcentration did not occur; and a marked or constant increase of mean red blood cells volume could not be observed. In one of these animals (table 5) hemolysis appeared and the dog died during the experiment. In another control which had neither been anesthetized nor submitted to incisions, no alteration took place (table 7, graph 7).

TABLE 1

Dog 5-52-C1. Clamping of the vena cava for 50 minutes

	Before Clamping	Time in minutes			
		After Clamping		After Clamp removal	
		15	30	15	60
Red blood cells $\times 10^6$.....	6.0	6.5	6.7	6.6	6.7
Hematocrit %.....	45.0	52.0	54.5	52.0	54.5
Mean Corpuscular Volume μ^3....	74.2	80.2	81.3	79.1	81.7
Hemolysis Hb. gm %.....	0.06	0.08	0.20	0.20	0.40

TABLE 2

Dog 17-52-C1. Clamping of the mesenteric vein for 45 minutes

	Before Clamping	Time in minutes					
		After Clamping			After Clamp removal		
		15	30	45	15	150	240
Red blood cells $\times 10^6$	7.4	6.6	6.4	6.4	7.5	8.2	8.9
Hematocrit %	51.0	50.5	46.1	46.5	59.2	64.5	64.7
Mean Corpuscular Volume μ^3	68.9	76.5	70.7	73.8	80.0	78.7	72.7
Hemolysis	0	++	++	++	+ +	+++	+++

TABLE 3

Dog 26-52-C1. Clamping of the mesenteric vein for 60 minutes

	Before Clamping	Time in minutes				
		After Clamping			After Clamp removal	
		10	20	50	30	180
Red blood cells $\times 10^6$	5.9	6.2	6.8	7.0	7.3	7.7
Hematocrit %	36.3	46.0	49.0	51.0	53.5	56.0
Mean Corpuscular Volume μ^3	61.5	74.2	72.0	72.8	73.2	72.7
Hemolysis Hb gm %	0.1	0.1	0.1	0.05	0.1	0.1

TABLE 4

Dog 27-52-C. Visceral handling for 20 minutes

	Before visceral handling	After handling before replacement	Time in minutes		
			After replacement of intestines		
			60	180	240
Red blood cells $\times 10^6$	5.8	7.2	7.4	7.6	9.2
Hematocrit %	45.0	57.4	60.0	61.7	62.5
Mean Corpuscular Volume μ^3	77.5	79.7	81.0	81.1	67.9
Hemolysis Hb gm %	0	0.30	0.35	0.85	0.60

TABLE 5

Dog 30–52–C. Control. Anesthesia, exposure, handling of femoral veins and blood pressure recording. This animal died during the experiment.

	Before operation	Time in minutes			
		40	100	160	340
Red blood cells × 10^6	6.3	6.2	6.0	6.4	5.7
Henatocrit %	46.0	40.0	43.0	47.1	43.1
Mean Corpuscular Volume μ^3	73.0	64.5	71.7	73.6	75.6
Hemolysis Hb gm %	0	0	0.1	0.2	0.3

TABLE 6

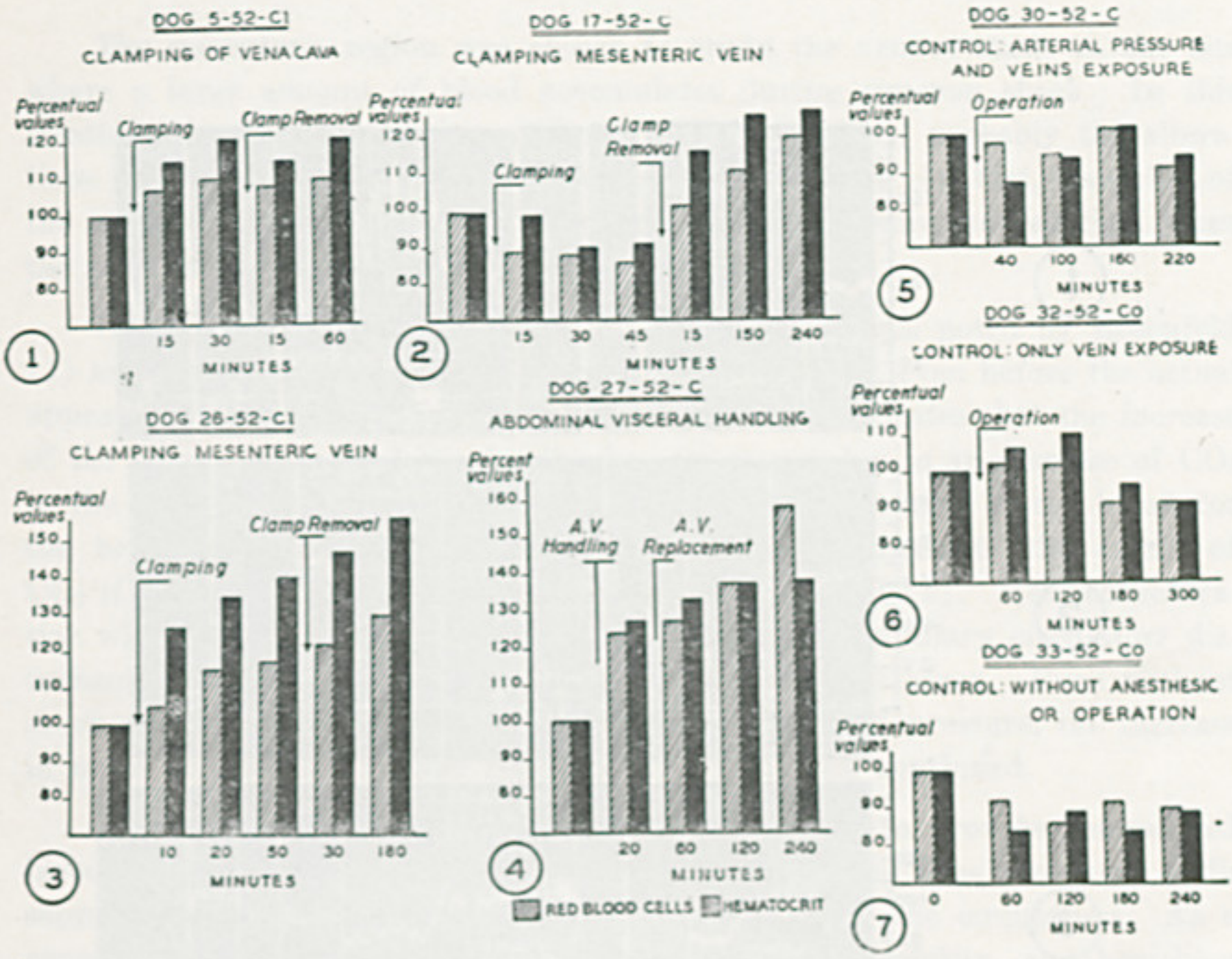
Dog 32–52–C. Control. Anesthesia and femoral vein exposure.

	Before anesthesia	Time in hours			
		After anesthesia and vein exposure			
		1	2	3	5
Red blood cells × 10^6	6.9	7.1	7.0	6.3	6.3
Hematocrit %	49.4	52.4	54.4	47.4	45.0
Mean Corpuscular Volumes μ^3	71.6	73.8	77.7	75.2	71.4
Hemolysis Hb gm %	0	0	—	0	0

TABLE 7

Dog 33–53–C. Control. Without anesthesia or veins exposure

	Time in hours				
	0	1	2	3	4
Red blood cells × 10^6	5.0	4.6	4.3	4.6	4.5
Hematocrit %	37	31	33	31	33
Mean Corpuscular Volume μ^3	74.0	67.4	76.7	67.4	73.3
Hemolysis Hb gm %	0.16	0.12	0.12	0.16	0.16



GRACHS 1 — 7 — Percent changes of red blood cells counts and red blood cells volume (hematocrit). Disproportional increase of the hematocrit value show increase of the volume of the red blood cells.

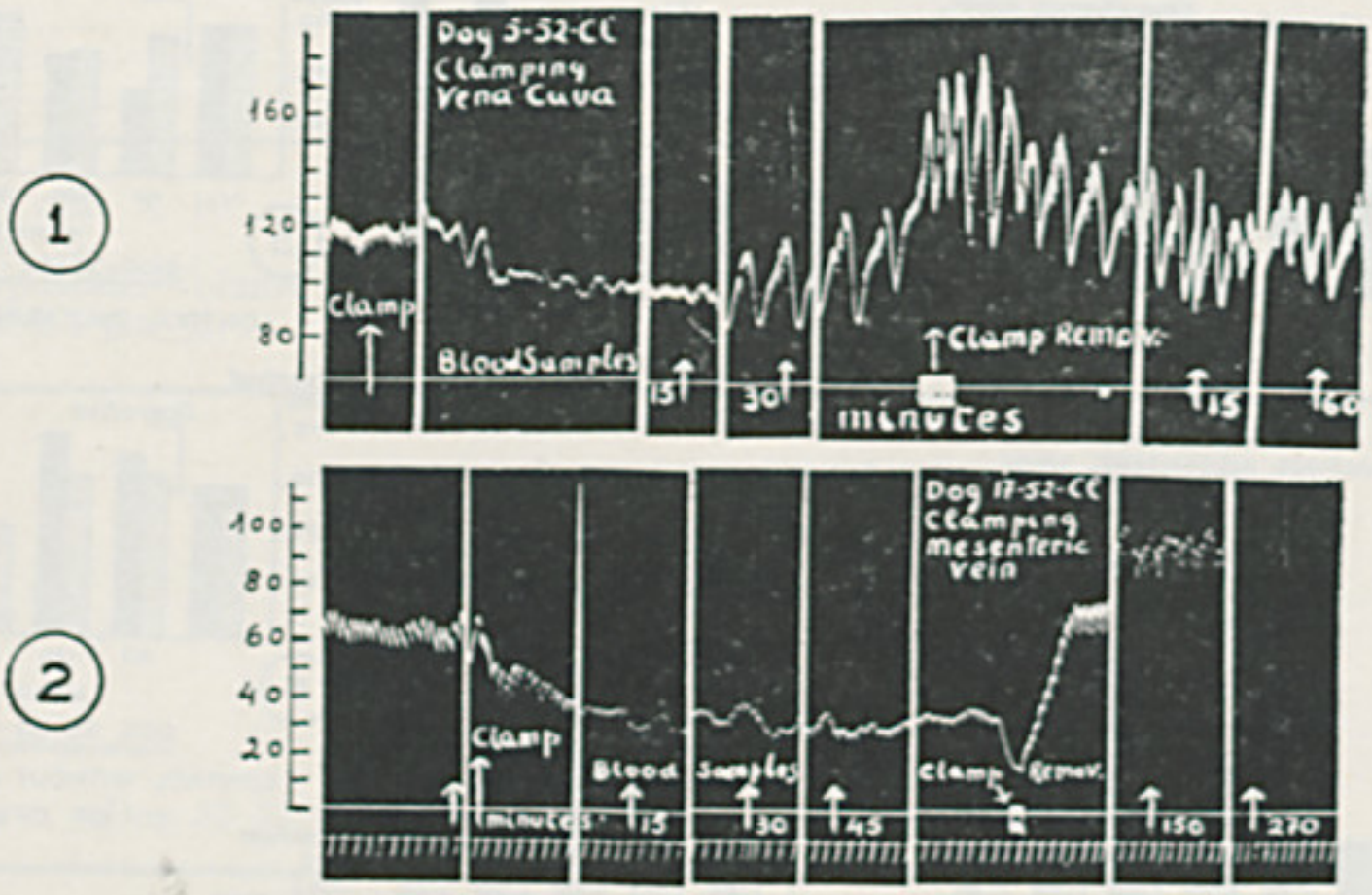


FIG. 1 — 5 — Arterial blood pressure recording.

DISCUSSION

The mesenteric region was chosen to study the venous stasis as the site where a large amount of blood accumulates during common shock. In this location a great accumulation of blood easily occurs and probably the alterations due to stasis there take place with more intensity. In fact, clamping of the mesenteric vein produces modifications which are more marked than when the vena cava is clamped.

It was observed the same hematimetric modifications noted by Rosenfeld (1) and Rosenfeld et al (2) in different kinds of shock, even before the actual appearance of shock Evidence was presented which indicates that the increase of the volume of the red blood cells observed is not due to an increase of CO_2 in the cells because the technic used for the homogenisation of the blood for the hematimetric determinations was sufficient to eliminate the excess of CO_2 if present, as was demonstrated by Rosenfeld et al (2). Hemoconcentration which according to Moon (4) is the first sign of capillary circulatory disturbance, appears even before an arterial pressure fall occur. Even without shock and even with normalization of the arterial blood pressure, the increase in mean corpuscular volume remained and hemolysis continued.

It seems that stasis in some regions is sufficient to provoke intense red blood cells modifications altering their permeability and thus provoking a disappearence of a great part of plasmatic liquid from the circulation. As a consequence, there is an increase of red blood cells fragility, and hemolysis occur, even without a traumatism.

SUMMARY

An increase of the mean corpuscular volume, hemolysis and hemoconcentration of the red blood cells were observed as a consequence of clamping of the vena cava or mesenteric vein in dogs. These changes appeared even when there was no appreciable fall of the arterial blood pressure. Venous stasis seems to provoke a modification of the permeability of the red blood cells thus inducing an increase of their volume by absorption of plasmatic fluid. Probably part of the blood fluid loss in shock is due to this mechanism.

RESUMO

Foi provocada estase sangüínea pelo pinçamento da veia cava ou da mesentérica em cães laparotomizados. Essa estase, assim como a simples manipulação das vísceras exteriorizadas, provocou aumento do volume médio das

hemácias, hemólise e hemoconcentração, observadas em sangue retirado da veia femural. Essas modificações apareceram mesmo quando não havia queda apreciável de pressão arterial. A estase venosa provoca portanto uma modificação da permeabilidade das hemácias com absorção do líquido plasmático que passa para êsses glóbulos, causando hemoconcentração e explicando parte da perda de plasma sangüínea no choque.

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