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STUDIES ON THE CIRCULATORY PATHOPHYSIOLOGY OF TRAUMA

I. *Effect of Acute Soft Tissue Injury on Nutritional and Non-nutritional Shunt Flow Through the Hindleg of the Dog*

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Observations on the effect of hemorrhagic shock on the total blood flow, and its division into nutritional flow and non-nutritional (shunt) flow, through the hindleg of the dog have been described (Lewis 1967). It was shown that despite a prolonged period of shock, nutritional flow to the tissues was still commensurate with the total flow rate. Blalock & Bradburn (1930) reported that following soft tissue trauma to one hindleg, there was an increase in oxygen saturation of the venous blood draining the traumatized leg and a decrease in oxygen saturation in the contralateral non-traumatized leg. In view of these observations which could be interpreted as demonstrating shunting of blood as a result of trauma, it was decided to investigate the effect of trauma on the total, nutritional, and shunt flow through the hindleg of the dog.

MATERIAL AND METHODS

A. *Experimental model:* Observations were carried out on 16 mongrel dogs of either sex, ranging in weight from 12 to 27 kg. The animals were anesthetized intravenously with sodium thiopental (Pentothal®) with repeated small doses as needed throughout the course of the experiment. A cuffed endotracheal tube was inserted and the animal ventilated on room air with a respirator. Through a longitudinal midline abdominal incision, the iliac arteries and veins were mobilized to just below the inguinal ligament. All collateral branches and the sacral artery were ligated and divided. The inguinal ligaments were divided to give adequate exposure to insure that complete isolation of the vascular pedicle to each hindleg was

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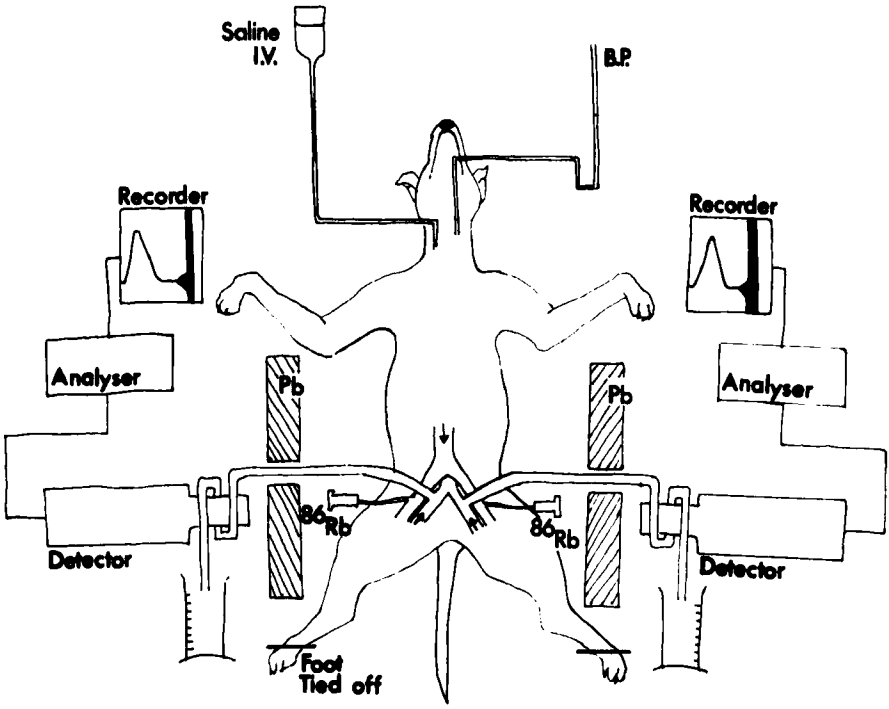

Figure 1. Experimental preparation. See text for detailed description.

accomplished. The circulation to the foot was isolated from this preparation by a tightly placed wire tourniquet around the ankle. A schematic representation of this experimental model is depicted in Figure 1.

Through the stump of the pudendo-epigastric artery located just proximal to the inguinal ligament, a short fine-bore polyethylene catheter (PE 47) was inserted into the iliac artery for injection of radioactive rubidium, approximately 60 μC/ml in saline. The tip of the catheter was carefully positioned so that it just protruded into the lumen of the iliac artery pointing upstream.

A T-tube with diameter corresponding to the vessel was placed at the midpoint of the iliac vein. Care was exercised to prevent any abnormal kink or angulation of the vein, which would impair free flow. Total venous return from the isolated hindleg was collected by diverting the flow out the sidearm of the T-tube.

Slow maintainance fluid infusion and return of collected venous blood were given through a wide-bore polyethylene catheter inserted in the external jugular vein. Systemic mean arterial blood pressure was monitored continuously via a carotid catheter to a mercury manometer.

In dogs in whom lumbar sympathectomy was done, the entire lumbar sympathetic chain was resected. In dogs in whom blood flow to the traumatized leg was regulated, a calculated amount of partial occlusion of the iliac artery proximal to the injection site was maintained throughout the experiment.

B. *Experimental procedure:* Determination of total, nutritional, and shunt flow was done by diverting the venous return of the leg studied through the T-tube, past a scintillation detector, and into an external collecting system. The background level of radioactivity was recorded and a blood sample was taken for determining the background activity. Radioactive rubidium was then injected as a rapid bolus into the iliac artery. Simultaneously with the injection, venous outflow was collected into a graduated cylinder to measure its volume flow. When the detector indicated that the primary curve of isotope activity had passed, the venous return was rediverted back to its normal course. The time of collection was recorded with a stop-watch. The total volume of venous blood collected for the time interval was noted and after thorough mixing a representative sample was taken for determination of its activity. The blood was then infused back into the animal. Only one leg was studied at a time. At the end of the collection period, blood samples were also taken from both iliac veins and carotid artery for determination of hematocrit and pO_2. The legs were removed at the completion of the experiment and their weight was recorded without the foot.

Baseline flow studies were done bilaterally allowing 30 min between each injection to assure a stable background level of rubidium in the blood. Following the control measurements, the left thigh was traumatized with 200 blows using a hard rubber mallet. A collection was then made on the traumatized side immediately after the trauma, and at hourly intervals (i.e. 1, 2, 3, 4, etc. hours after the trauma). The non-traumatized side was studied 30 minutes after the trauma, and at hourly intervals (i.e. 1.5, 2.5, 3.5, 4.5, etc. hours after the trauma). Flow studies were thus made at 30 min intervals, alternating legs, for a 6-hour period. In the group of dogs with unilateral sympathectomy, only the traumatized leg was studied and injections (30 μc) were made into this leg at 30 min intervals. In those animals in whom bilateral sympathectomy was performed, control measurements were done both before and after the sympathectomy. The animals were then subjected to soft tissue trauma.

C. *Theory of the rubidium technique and calculations:* Rubidium behaves chemically similar to potassium, which is principally an intracellular ion. Thus, when a trace amount of rubidium diffuses out of an exchanging capillary vessel, it eventually enters the cells. The differences in the intracellular and extracellular potential pools are so great that the cells can be considered a sink for tracer amounts of rubidium. In an exchanging capillary vessel, if one assumes that the blood flow and fluid diffusion characteristics are such that complete diffusion equilibrium exists by the time the blood leaves the capillary, then this blood will contain no rubidium when it enters the venule. The blood will have been completely cleared of rubidium. Such a vessel (A) is shown schematically in Figure 2. On the other hand, if the vessel in question is not an exchanging capillary but a non-exchanging vessel (i.e. a shunt), then the rubidium will have no chance of leaving the blood and all that entered on the arterial side will be still present when the blood enters the venules (cf. vessel B, Figure 2). If one could be certain that all vessels, under both normal and abnormal circumstances, were either Type A or Type B as shown in Figure 2, then the percentage of the injected rubidium appearing in the venous blood would reflect accurately the division of the total flow into these two pathways, i.e. nutritional (A) and shunt (B). However, there

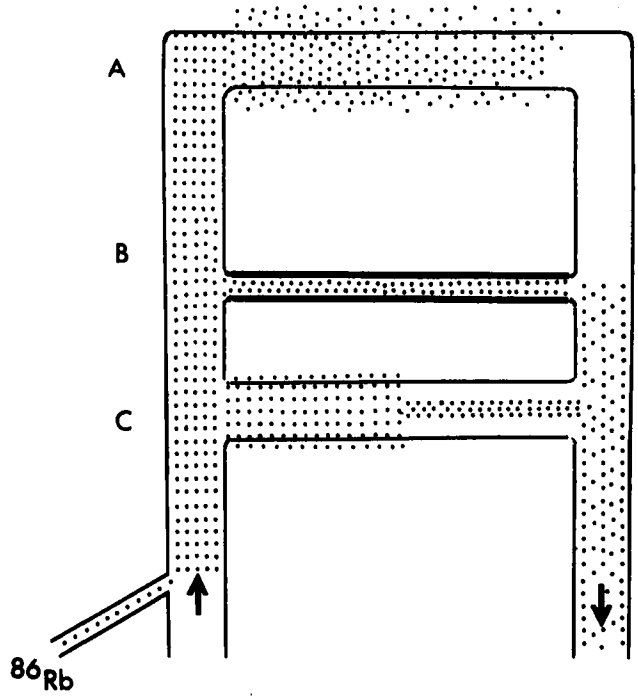

Figure 2. Schematic representation of rubidium transport in vascular bed. Type A vessel: capillary with diffusion and transport characteristics permitting complete clearance (100 per cent "nutritional" flow). Type B vessel: anatomical arteriovenous communication with no rubidium clearance (100 per cent "shunt flow"). Type C vessel: incomplete rubidium clearance ("physiological" shunting).

is no certain proof that blood leaving an exchanging capillary is always completely cleared of rubidium. This clearance may be only partial, such as depicted in vessel C, Figure 2. In this situation, one can consider that part of the total flow passing through the capillary is completely cleared of rubidium and the remainder is not cleared. If one then defines that this theoretical remainder of rubidium is shunted (physiological shunting), then the original concept of nutritional flow and shunt flow is still valid. It must be remembered, however, that in this definition, nutritional flow is that portion of the total flow which is theoretically completely cleared of rubidium. For a detailed discussion of the theory, the reader is referred to Renkin (1959) and Friedman (1965).

The calculations of the various flows were carried out as follows: Total flow (ml/min 100 g) was calculated from the volume and duration of the venous collection and the weight of the non-traumatized leg.

The percentage of injected rubidium recovered in the venous return was calculated from the radioactivity of the collected sample minus the background activity in the blood immediately before the injection and the amount of radioactivity injected. All determinations of radioactivity were carried out in a well-type scin-

Table 1. Animals subjected to trauma alone (mean ± S. D., 6 dogs).

Traumatized leg	Unit	Control	0	1	2	3	4	5	6
Total blood flow	ml/min/100 g	4.15±1.78	6.04±2.62	3.55±1.61	2.15±1.32	2.06±0.76	2.55±1.06	2.49±0.98	2.07±0.75
Nutritional blood flow	% total	77.1 ±7.0	58.2 ±3.1	73.8 ±5.0	86.7 ±8.2	87.6 ±5.7	85.6 ±10.7	88.8 ±5.4	92.7 ±1.2
PS	ml/min/100 g	6.03±1.16	5.33±2.51	4.67±1.85	4.10±1.46	4.27±1.26	5.11±2.38	5.66±2.78	5.40±1.81

Non-traumatized leg	Control	½	1½	2½	3½	4½	5½	
Total blood flow	3.20±0.78	2.18±1.10	2.00±1.09	1.62±1.0	2.02±0.97	1.88±1.40	1.97±1.37	–
Nutritional blood flow	83.9 ±7.1	88.3 ±6.6	88.6 ±6.2	89.3 ±5.5	87.0 ±5.6	90.2 ±4.0	89.4 ±2.5	–
PS	5.97±1.71	4.47±1.34	4.06±1.56	3.39±1.70	3.94±1.58	4.09±2.64	4.28±2.94	–

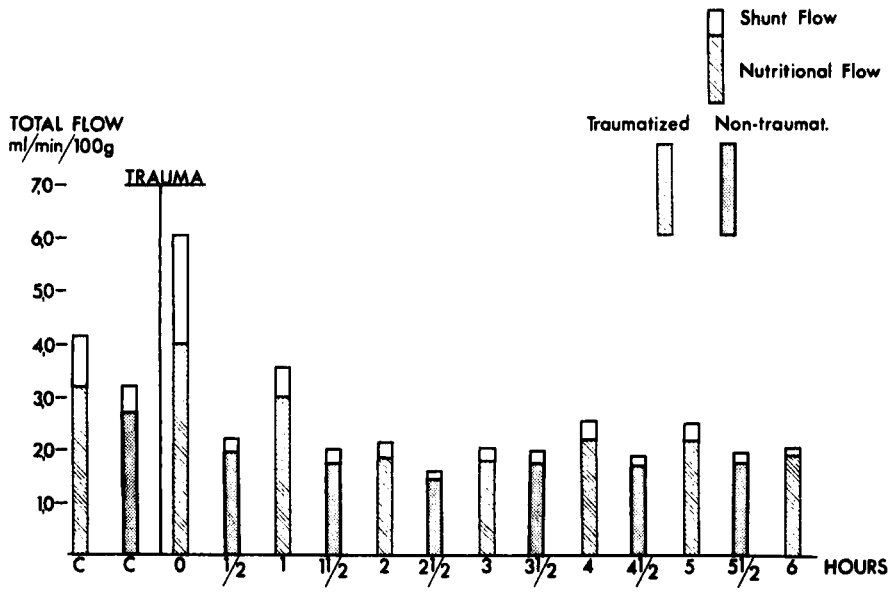

Figure 3. Trauma alone, mean values from 6 dogs. The full staple represents total flow, the upper clear portion "shunt" flow and the lower portion "nutritional" flow. Hatched bars are from the traumatized side and stippled bars from the non-traumatized side. C = control; 0–6 = time in hours after trauma.

tillation counter. The isotope curves recorded during the collection were not used for these calculations but were to insure complete collection of the isotope that was not cleared from the blood stream. Thus, shunt flow = total flow ×% injected rubidium recovered in venous return, and Nutritional flow = Total flow × (1 — per cent rubidium recovered).

PS, the permeability-surface area product introduced by Renkin (1959), was calculated from the total flow and the percentage of rubidium recovered in the venous returns. PS = — Q · 1 n (1-E), where Q = total flow in ml/min/100 g tissue and E = extraction or uptake of rubidium by the tissue (1-E = recovery in venous return). This parameter is an expression of the transport function of the capillary bed under the existing conditions.

Table 2. Animals subjected to trauma

PO_2, iliac vein, mm Hg	Control	0	½	1	1½	2
Traumatized leg	27.6±7.8	34.4±2.3	32.8±4.6	30.9±5.1	23.9±5.0	22.5±5.7
Non-traumatized leg	24.0±6.9	13.9±2.9	18.5±5.1	16.0±5.3	18.5±6.8	16.6±4.6

* Only one observation.

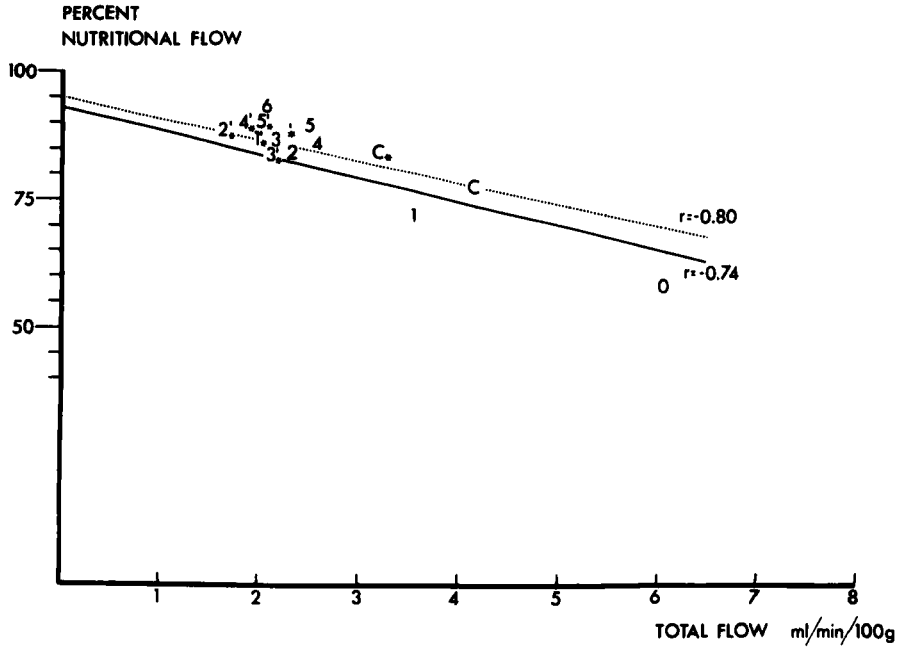

Figure 4. Trauma alone, mean values from 6 dogs. Same data as shown in Figure 3, plotted here as percentage of nutritional flow versus total flow in ml/min 100 g. The symbols C (control), O (immediately after trauma), and 1 to 6 hours (after trauma) are the mean of values from the traumatized side. The solid line is the linear regression line for all of these values. The symbols C (control), 1* (½ hour), and 1* to 5* (1½ to 5½ hours) are the mean values from the non-traumatized side. The dotted line is the linear regression line for all of these values. For any given total flow the percentage of nutritional flow is lower for the traumatized leg than for the non-traumatized. This suggests that there is more shunting as a result of trauma, but the difference between these two lines is not statistically significant.*

alone (mean ± S. D., 6 dogs).

2½	3	3½	4	4½	5	5½	6
21.4±4.3	20.8±5.0	20.3±3.7	24.2±5.1	23.5±2.8	21.3±4.2	18.8*	19.7±3.1
18.2±4.5	14.9±3.2	19.1±5.2	17.8±3.0	19.3±6.7	12.0±9.9	12.0±5.7	20.5*

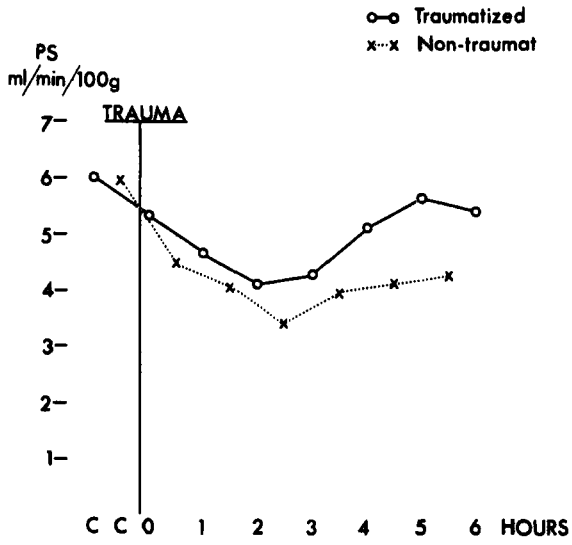

Figure 5. Permeability-surface area product (PS) calculated from the data for trauma alone. PS decreases bilaterally after trauma, slightly more but not significantly on the non-traumatized side, immediately after trauma, but tends to return towards the control values.

RESULTS

A. *Trauma alone (6 dogs):* Table 1 shows the mean values for total flow, nutritional flow, PS; and Table 2 shows PO_2 in the dogs subjected to trauma alone. There was an immediate increase in total flow to the traumatized leg, which was on the average over after 1 hour. This increase occurred in all animals. On the non-traumatized side the total flow decreased and remained low. Figure 3 shows the mean values for the total and division into nutritional flow (lower part of staple) and non-nutritional or shunt flow (upper part of staple). Note that the increase in total flow immediately after the trauma was due almost entirely to the increase in shunt flow. After this phase there was very little difference in the flow between the two legs. In Figure 4 the same flow data have been plotted in another manner to bring out the relationship between total flow and nutritional flow. Note that as total flow increased, the percentage of the flow which was nutritional decreased and vice versa. The solid line is the regression line for all of the values obtained on the traumatized side, and the dashed line that for the non-traumatized side. These two lines are not statistically significantly different ($P > 0.05$). Figure 5 shows the mean values for the calcu-

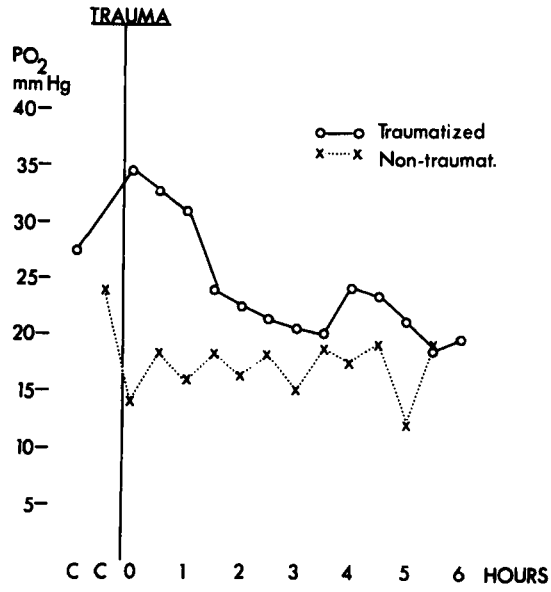

Figure 6. Venous PO_2 from the hind legs. Trauma causes an immediate increase in PO_2 from the traumatized side and a decrease on the non-traumatized side. With time the value on the traumatized side declines and is nearly the same as on the non-traumatized side.

lated permeability surface area product (PS) in these same animals. Note that there was a smaller PS bilaterally after trauma, but no significant difference between the two sides. In Figure 6 are shown the mean venous PO_2 data on these animals. The PO_2 changes varied directly with the changes in flow. The arterial PO_2 values were always within the normal range in all groups.

B. *Trauma + sympathectomy (7 dogs):* The mean values for the various parameters are given in Table 3. On the basis of the experiments from the previous group, these animals were to have been followed for a maximum of 4 hours. Even so, none of the bilaterally sympathectomized animals survived more than 3½ hours after the trauma. Figure 7 shows the division of total flow into nutritional and shunt flow. There was on the average an increase in total flow, mainly due to increased shunt flow, following sympathectomy, i.e. the same pattern as that seen in the previous group following trauma. In this group, trauma after sympathectomy was not associated with any further increase in total flow. The flows bilaterally declined and even-

Table 3. Animals subjected to trauma + sympathectomy

Traumatized leg	Unit	Control		0	½
		before sympath.	after sympath.		
Total blood flow	ml/min 100 g	4.47±2.29	5.08±2.64	4.51±2.88	6.95±2.09
Nutritional blood flow	% total	77.0 ±8.2	69.1 ±13.3	64.3 ±17.2	57.3 ±6.0
PS	ml/min 100 g	6.53±2.73	5.61±1.52	4.00±0.60	5.81±1.27
PO_2, iliac vein	mm Hg	23.4 ±5.1	27.5 ±7.4	30.3 ±6.4	31.0 ±7.8
Non-traumatized leg					
Total blood flow		2.71±1.18	3.07±0.89		2.32±0.72
Nutritional blood flow		86.1 ±6.1	77.1 ±10.8		80.1 ±12.4
PS		5.15±1.21	4.59±1.33		3.94±1.49
PO_2, iliac vein		21.5 ±4.2	23.1 ±4.7	18.0 ±3.2	23.3 ±1.5

• Only one observation.

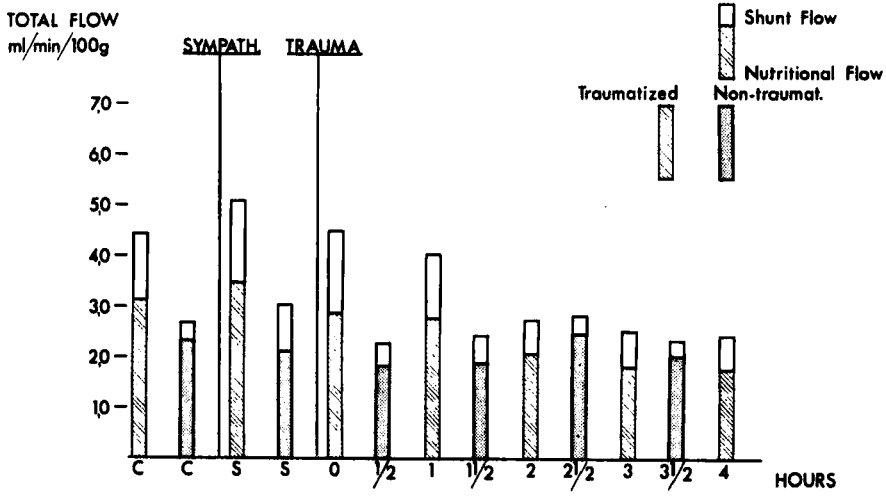

Figure 7. Sympathectomy + trauma, mean values from 7 dogs. C = control, before sympathectomy. S = control after sympathectomy. 0–4 = hours after trauma. Staples as in Figure 3. Note the increase in total flow after sympathectomy, which is virtually all "shunt" flow. With subsequent trauma there is no additional increase in flow.

(Mean ± SD, 7 dogs: 4 bilateral sympath., 3 unilateral)

1	1½	2	2½	3	3½	4
4.07±2.10	4.06±1.14	2.74±0.99	2.65±0.80	2.55±0.35	2.54±0.49	2.45±0.73
69.5 ±11.8	64.1 ±16.2	75.7 ±12.3	74.9 ±10.9	73.4 ±17.9	77.6 ±13.8	73.2 ±23.1
4.53±1.68	4.09±0.83	3.83±0.86	3.61±0.35	3.57±1.35	3.89±0.89	3.41±1.33
31.7 ±5.9	29.1 ±2.6	28.4 ±5.0	25.0±8.4	25.3 ±3.5	23.3 ±5.0	23.0 ±3.5
	2.43±1.39		2.87*		2.34*	
	77.3 ±15.9		87.0*		87.6*	
	3.34±1.39		5.85*		4.85*	
20.3 ±3.4	23.3 ±4.7	17.4 ±6.4	21.1 ±4.3	19.6 ±9.6	20.5 ±6.1	21.5 ±4.9

tually became about the same as the condition of the animals became worse. The same inverse relationship between total flow and percentage of nutritional flow was observed in these animals (Fig. 8) as in those with trauma alone. Here also the two regression lines are not significantly different ($P > 0.05$). Sympathectomy did not alter the PS value very much, which declined as in the previous group after trauma (Figure 9). Figure 10 shows the mean venous PO_2 values indicating, as in the animals not sympathectomized, the close parallel between PO_2 and flow.

C. *Trauma + restricted inflow* (3 dogs). In these animals the iliac artery proximal to the injection site was partially occluded just prior to traumatization so as maintain consistently a flow level somewhat less than that in the control period. In this group observations were made at 30 min intervals for 4 hours just on the traumatized side. Table 4 shows the mean values of the various parameters. With a constant degree of arterial constriction, there was a gradual decline in total flow and blood pressure as the animal's condition worsened. However, the smaller total flow was, as in the previous two groups, associated with a progressive reduction in shunt flow (Figure 11) and

Table 4. Animals with restricted inflow

Traumatized leg	Unit	Control	0	½	1
Total blood flow	ml/min	5.41 ± 1.97	3.15 ± 0.35	3.11 ± 0.31	2.89 ± 0.56
Nutritional blood flow	% total	70.2 ± 7.2	86.0 ± 5.4	75.3 ± 9.0	72.8 ± 8.9
PS	ml/min	6.35 ± 1.52	6.39 ± 1.64	4.61 ± 1.86	4.02 ± 1.89
PO_2 iliac vein	mm Hg	28.9 ± 6.0	23.4 ± 3.3	22.8 ± 4.0	23.3 ± 3.8
Non-traumatized leg, PO_2, iliac vein	mm Hg	28.3 ± 3.0	26.1 ± 3.0	26.8 ± 3.2	23.3 ± 3.6

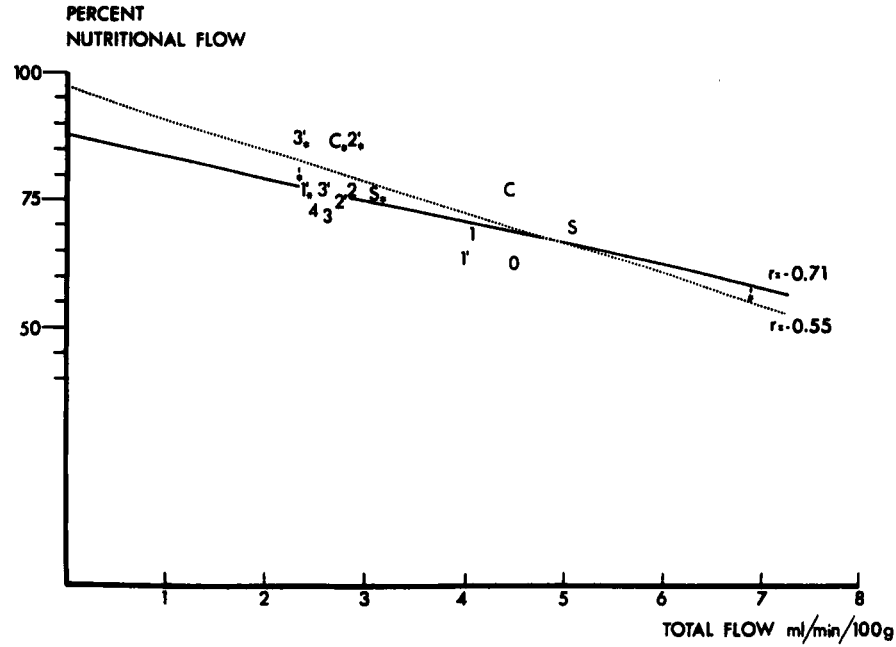

Figure 8. Same data as in Figure 7 plotted as in Figure 4 with same symbols. The solid line is the linear correlation for the traumatized side, and the dotted line for the non-traumatized side. These two are not statistically significantly different.

to traumatized leg (Mean ± S.D., 3 dogs).

1½	2	2½	3	3½	4
2.20±0.64	2.03±0.94	1.73±0.77	1.58±0.68	1.26±0.65	1.05±0.81
82.3 ±9.5	82.2 ±3.5	84.9 ±6.2	82.2 ±8.3	85.9 ±7.9	92.1 ±10.8
4.22±2.26	3.58±1.89	3.48±1.96	3.01±1.81	2.72±1.66	2.76±1.67
18.3 ±4.5	19.3 ±1.0	18.5 ±0.5	19.8 ±2.5	15.7 ±0.8	15.8 ±0.6
19.8 ±4.5	19.4 ±2.7	19.8 ±0.3	18.8 ±1.6	16.2 ±2.5	18.2 ±4.2

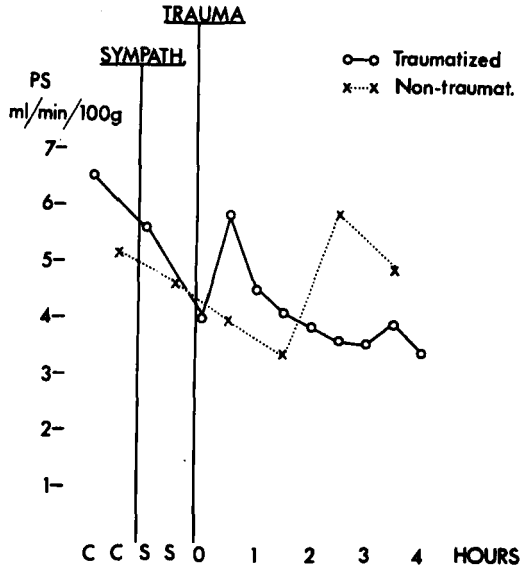

Figure 9. PS data for the sympathectomized animals. There is a decline in PS bilaterally which returns towards control on the non-traumatized side, but not on the traumatized side.

therefore a progressive increase in the percentage of nutritional flow (Figure 12). As the flow declined, the PS declined (Figure 13) as well as venous PO_2 (Figure 14).

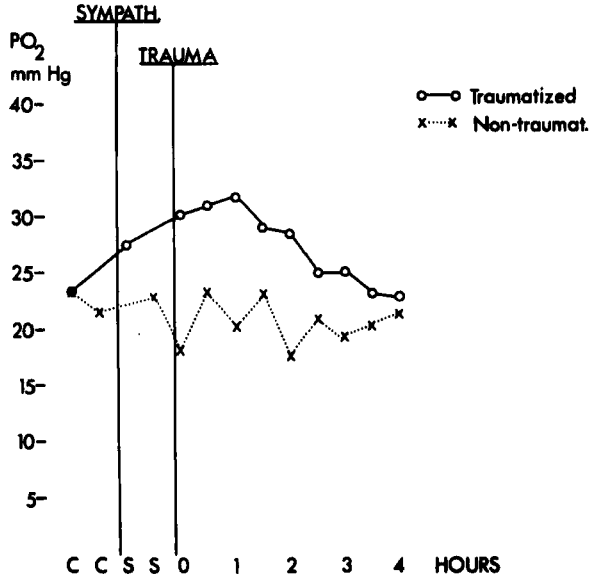

Figure 10. PO_2 values for the sympathectomized animals. Trauma further increases PO_2 on the traumatized side, but this declines as the animal's condition worsens. Sympathectomy essentially abolishes the immediate posttraumatic decrease on the non-traumatized side.

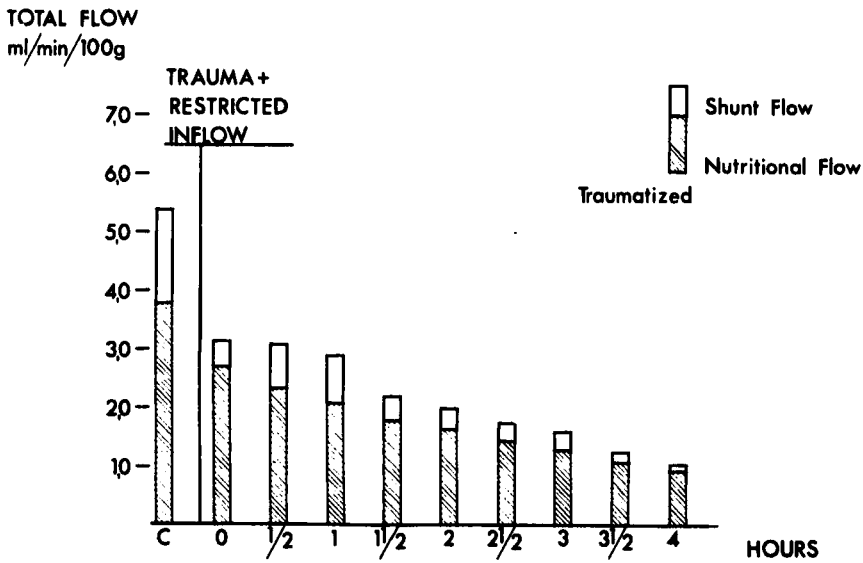

Figure 11. Trauma + restricted inflow in 3 dogs, mean values. Only the traumatized leg was studied. Symbols and staples as in Figure 3. Note that with no increase in total flow immediately after trauma, there is no increase in "shunt" flow.

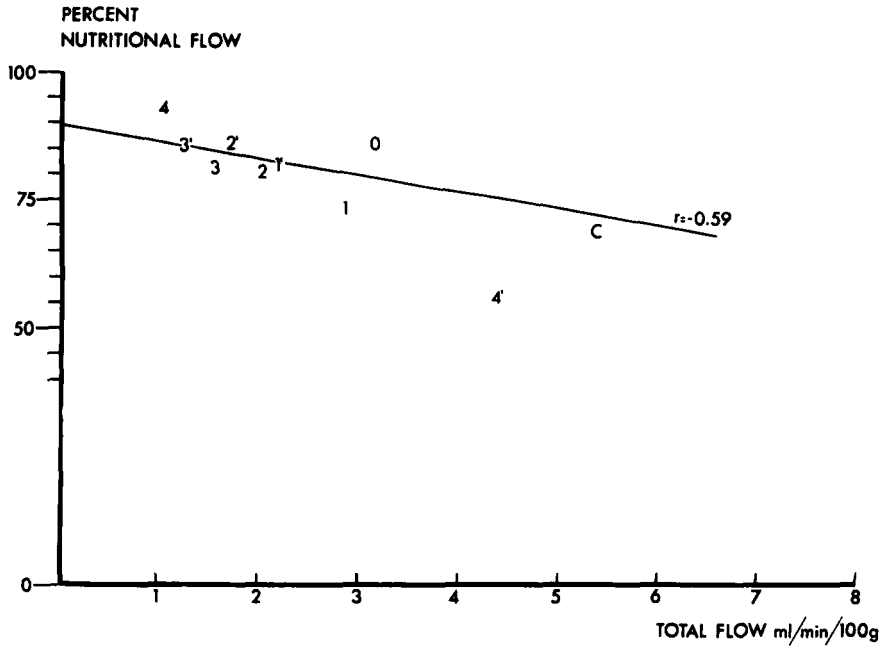

Figure 12. Same data as in Figure 11 plotted as in Figure 4. The symbols with primes (e.g., 3') represent half-hour values (e.g. 3½ hours). All values from traumatized leg. This regression line is not significantly different from the other two (trauma alone, trauma + sympathectomy) from the traumatized side.

DISCUSSION

Soft tissue trauma to the hind leg caused an immediate, but transient (up to 1–2 hours) increase in the total blood flow. Since the tissue, with the foot excluded, consisted for the most part of skeletal muscle, it can be assumed that it was muscle blood flow that increased. The mechanism of this increase has not been investigated in detail in these studies, but the lessening or even disappearance in the flow increase following trauma in sympathectomized animals suggests that the phenomenon was a release from sympathetic vasoconstrictor tone. Further studies on this point will be pursued. What changes occur at the nerve endings that produce the vasodilatation of trauma are purely speculative at this point. Several features, however, are known. Kjellmer (1965) has shown that potassium will produce marked vasodilatation. The release of this intracellular ion or some other intracellular component could cause the degree of vasodilatation seen here. Metabolic products, not as yet specifically identified, are also known to

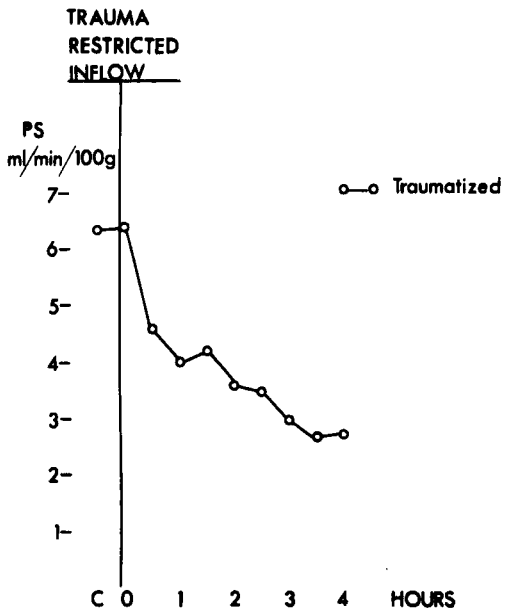

Figure 13. Trauma + restricted inflow markedly reduced PS with no tendency to return towards control.

impair the ability of the sympathetics to elicit a vasoconstrictor response (Lewis & Mellander 1962). Vasodilatation can be produced by activation of the vasodilator sympathetic fibers which probably does not play a role here. It seems hardly conceivable that trauma would interfere with the action of the constrictor fibers locally and at the same time enhance the activity of the dilator fibers. A central dilator action can be eliminated since the contralateral leg responded with constriction which was in part a generalized response to the trauma. The failure to elicit this response on the traumatized side is acceptable since it is known that locally originating dilator simuli can easily override the central or general constrictor mechanism. The pattern of response of total flow following trauma in both the non-sympathectomized and unilaterally sympathectomized animals is essentially the same as that recently reported by Liu (1968).

With the post-traumatic increase in total flow, there was a predominant increase in non-nutritional flow, which resembled that seen after sympathectomy. There was thus an immediate albeit transient increase in shunting as a result of this trauma. The evidence, though not conclusive, pointed to the increase in flow as the responsible fac-

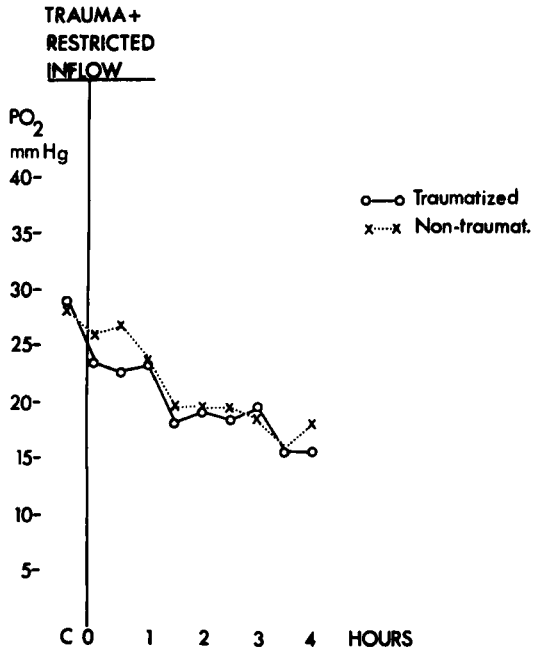

Figure 14. Preventing the immediate post-traumatic increase in total flow also abolished the PO_2 increase on the traumatized side.

tor and not the trauma per se. There was a direct relationship between the level of total flow and the level of non-nutritional flow. This has been seen previously in rubidium studies on normal skeletal muscle (Friedman 1965, Renkin 1959, Winbury et al. 1965), and was seen here on the non-traumatized side. The relationship between total flow, nutritional flow, and shunt flow was essentially the same on the traumatized side as on the non-traumatized side as evidenced by the similar regression lines in Figure 3.

Since the division of total flow into nutritional and shunt flow is based on the extraction of rubidium from the blood as it passes through the tissue, there is a question whether this is valid for muscle that has been traumatized. If, as was alluded to above, there might be a leakage of potassium from the cell, the same might occur for rubidium. However, with the degree of trauma employed here, the ability of cells (i.e. the leg as a whole) to take up rubidium was not significantly impaired. Following the passage of the injected bolus of the isotope, there appeared to be no significant long-term back diffusion into the blood, as evidenced by the low and constant levels of activity in the venous

blood draining the leg. This was checked on a number of occasions. The impairment of rubidium uptake following trauma would then lead to a shunt flow out of proportion to total flow. This did not occur, indicating that rubidium uptake by the traumatized tissue was not significantly impaired.

All of the traumatized legs, with the exception of those with restricted inflow, had large hematomas and weighed on the average 17 per cent greater than the non-traumatized leg (range 1 to 34 per cent). Is the rubidium method valid in the presence of this large extracellular collection? Rubidium will diffuse into this fluid if it is in close relationship with exchanging capillaries. However, the changes in flow and changes in rubidium uptake suggest that this inert extracellular pool was not to any great extent in contact with exchanging capillaries. Furthermore, in the animals without significant increase in leg weight, the same relationship between flow and rubidium was observed as in animals with great increases in leg weight.

It would appear that the increase in flow can satisfactorily explain the previous observations of Blalock & Bradburn (1930). If the flow increase is a release from sympathetic tone, then it is a non-metabolic increase. Metabolic flow increases, i.e. those in which the demand for flow and nutrients increase, as in exercise, are associated with an increase in the size of the capillary bed (Renkin et al. 1966). Non-metabolic flow increases are not associated with an increase in the size of the capillary bed (note unchanged PS after sympathectomy), nor is there an increase in oxygen consumption. With a constant oxygen consumption, an increase in flow is associated with a decrease in oxygen extraction, which is reflected by an increase in venous oxygen content and PO_2. This was seen in the present study. For technical reasons it was not possible in this study to measure oxygen consumption precisely, but this is to be investigated in detail.

While the physiological explanations in the face of an unchanged, or relatively unchanged, oxygen consumption can well explain the increased venous PO_2 and the increased shunt flow, the data of the present study do not rule out the possibility of true arterio-venous shunting, i.e. the opening up of previously closed large direct channels that by-pass the capillary bed.

Clinical applications of these experimental data would appear to be premature. However, if man responds in the same way the dog does to soft tissue trauma, one can expect an increased demand for cardiac output because of the increase in flow through the injured area.

Shoemaker et al. (1967) have recently reported that there was an acute increased cardiac output in patients after trauma.

Furthermore, the inability of the vasculature in the injured tissue to respond to the normal sympathetic stimuli, such as has been seen in prolonged reduced flow and hemorrhagic shock in the cat (Lewis & Mellander 1962, Mellander & Lewis 1963) could lead to a vicious cycle of decompensation. This may be a common denominator for the peripheral circulatory collapse in the morbidity and mortality associated with different syndromes, e.g. crush syndrome.

SUMMARY AND CONCLUSIONS

1. Total blood flow, nutritional flow, non-nutritional blood flow, and venous PO_2 determinations have been done on 16 dogs following soft tissue trauma to one of the hind legs.

2. Trauma was associated with an immediate increase in total blood flow, which was for the most part non-nutritional. The rise in venous PO_2 observed previously by Blalock & Blackburn has been confirmed.

3. Evidence is presented sugesting that the phenomenon represents a temporary release from sympathetic vasoconstrictor tone. It further suggests that this presumably non-metabolic flow increase is associated with the expected increase in "physiological" shunting and most likely not with a true anatomical arterio-venous communication.

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