Mechanism of Increased Femoral Arterial Flow During Body Warming.* (27432)

W. A. MANSON,† N. S. ASSALI AND A. HEMINGWAY
(With the technical assistance of R. Beck, D. Hopper and W. Davis)
Departments of Obstetrics and Gynecology, and Physiology, University of California School of Medicine, Los Angeles

Westin *et al.*(1) have shown that in the nonshivering hypothermic dog anesthetized with pentobarbital, immersion of the body in warm water (40-45°C) evoked an immediate and striking rise in the femoral artery blood flow at a time when the blood and rectal temperatures were still falling. They suggested that this rise might be due to an active vasodilatation in the femoral arterial bed caused by a reflex mechanism triggered by the contact of the body with warm water. This reflex could act either through inhibition of vasoconstrictor or stimulation of vasodilator innervation of the femoral bed. A direct action of heat on the vascular bed of the skin and skeletal muscle would also be implicated. The present studies were designed to investigate these various possibilities.

Material and methods. The experiments were performed on mongrel dogs weighing between 15 and 25 kg. Measurements of arterial pressure, and of skin, rectal or esophageal temperatures were made as previously described(1). Femoral arterial flow was measured with a gated sinewave electromagnetic flowmeter described elsewhere(2). Cardiac output was measured by either the direct Fick or the dye dilution technic using cardiogreen as an indicator.

The dogs were divided into 3 groups. Group A (intact control) consisted of 5 dogs in which femoral flow on both sides was measured with the electromagnetic flowmeter during periods of cooling and rewarming as previously described(1). In Group B (2 dogs) control measurements of femoral flow and arterial pressure were made after which one leg was skinned from the calf to the inguinal area. Another series of control measurements was taken and was followed by cooling and rewarming of the animal as in the other series. Group C (5 dogs) had control measurements of arterial pressure and femoral flows on both sides. Thereafter, the abdomen was opened and bilateral (2 dogs) or unilateral (3 dogs) abdomino-pelvic sympathectomy was performed. After a second series of control measurements was recorded, cooling and rewarming were carried out as in the other groups.

Results. Fig. 1-3 present the data on these experiments. In the graphs depicting flows, each line represents the flow to one femoral artery. In the intact dogs, cooling produced a progressive fall in femoral blood flow. Within 30 seconds of body immersion in warm water, an increase in femoral flow reaching values which were close to 200% of control occurred while the rectal temperature was still falling (Fig. 1). There was a slight fall in the arterial pressure and cardiac output during cooling and a slight rise during rewarming. These changes were in agreement with those reported by Westin *et al.* for these temperature ranges(1).

Fig. 2 illustrates results obtained from dogs with a skinned limb. The fall in femoral flow during cooling and the rise during rewarming were similar in the skinned and unskinned sides and were not significantly different from those observed in the intact dog. Therefore, the presence of the skin on the limb does not seem to be necessary for the increase in femoral arterial flow of body warming.

In Fig. 3 and Table I, the data on the sympathectomized animals are presented. In the 3 animals in which the sympathectomy was unilateral, a prompt and marked increase in femoral blood flow on the operated side occurred immediately following the sympathectomy without any significant changes in the

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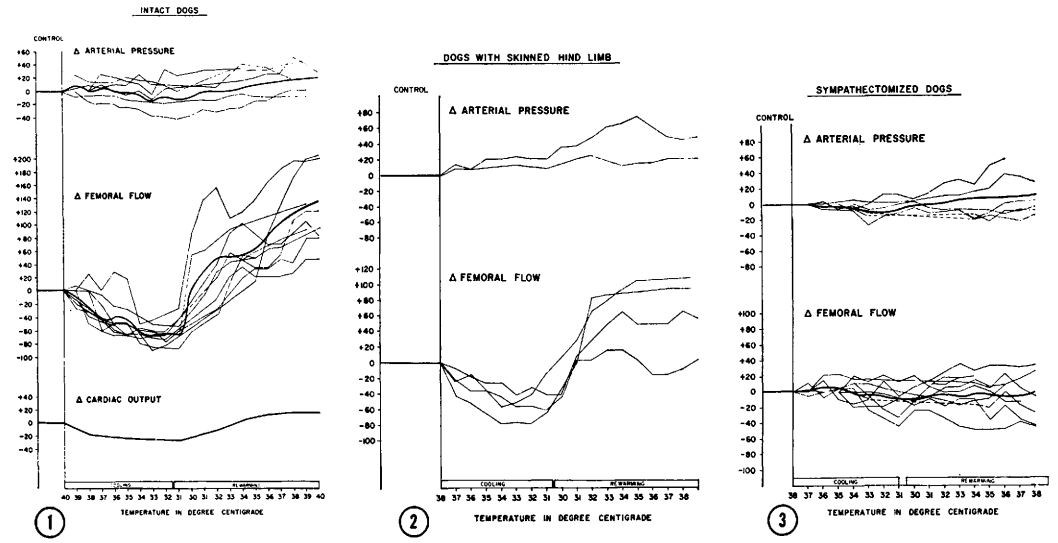

FIG. 1. Changes in arterial pressure, femoral flow and cardiac output in intact dogs subjected to cooling and rewarming. Each thin line depicts a femoral flow to one limb. The thick line represents the avg of all the measurements. In these animals, temperature was recorded with a thermistor inserted into the rectum. Note immediate rise in femoral flow upon rewarming of the body even though body temperature was still falling.

FIG. 2. Changes in arterial pressure and femoral flow in 2 dogs in which one limb was skinned. The increase in femoral flow upon body warming was closely similar to that of the intact dog. In these dogs, temperature was recorded from the rectum.

FIG. 3. Changes in arterial pressure and femoral flow in the series of sympathectomized dogs. Each thin line refers to a femoral flow from one sympathectomized limb. The thick line represents the avg of all the measurements. In these dogs, temperature was recorded with a thermistor inserted deep into the esophagus, because the extreme dilatation of the rectum caused by the sympathectomy made it difficult to obtain rectal temperature. Note lack of fall in femoral flow during cooling and absence of the rise in this flow during rewarming.

flow on the contralateral side (Table I). In these animals, the flow in the sympathectomized limb exhibited insignificant changes during both the cooling and rewarming periods while on the intact side the femoral flow fell during cooling and rose upon rewarming in a manner similar to the intact animals (Table I). In the bilaterally sympathectomized dogs, the femoral flow in both limbs increased after sympathectomy. When these animals were cooled and rewarmed, no significant changes in the femoral flows oc-

TABLE I. Effects of Unilateral Sympathectomy on Femoral Flow.

		Femoral blood flow			
		Left (intact)		Right (sympathectomy)	
		Flow		Flow	
	Temp. esophagus	ml/min.	% of control	ml/min.	% of control
Control	36°	88	100	162	100
Cooling	35	92	105	169	104
	34	97	110	180	111
	33	84	95	180	111
	32	69	78	170	105
Rewarming	31.2	134	152	164	101
	32	113	128	143	88
	33	135	153	136	84
	34	132	150	136	84
	35	132	150	147	91
	36	142	161	175	108

curred such as were seen in intact animals (Fig. 3).

Discussion. The present data show that in the nonshivering dog subjected to immersion hypothermia the fall in femoral flow which is observed during the cooling period is the result of stimulation of the sympathetic vasoconstrictor tone, because removal of the sympathetic chain abolished this response. Similarly, the rise in flow which occurs upon immersion of the body in warm water is the result of reflex inhibition of the sympathetic vasoconstrictor tone because it was entirely abolished by sympathectomy. Active stimulation of vasodilator fibers or centers does not seem to play a role in the femoral hyperemia of body warming, because the increase in femoral flow did not significantly exceed the control values obtained after sympathectomy but before the animal was subjected to hypothermia. Likewise, the effects of alterations in cardiac output or arterial pressure can be ruled out because the changes in these 2 parameters observed during rewarming were minor when compared with the increase in femoral flow.

The question arises as to the pathways and function of this femoral vasodilatation. Since skinning of one leg did not abolish the increase in femoral flow on that side, it is apparent that the reflex for inhibition of the sympathetic vasoconstrictor tone need not involve contact with warm water of the skin of the same limb. Furthermore, dilatation of the vascular bed in the skeletal muscle is probably the primary factor involved because the increase in femoral flow in the limb occurred in the absence of the skin. Also, in Westin's studies(1) as well as in the present one, the rise in femoral flow upon rewarming occurred while the temperature of the blood, rectum and esophagus was still falling. This indicates that the reflex initiated by contact of the body with warm water is sufficiently strong to overcome the effects of the cold blood which is presumably still bathing the vasomotor centers. These latter may in fact be more sensitive to the changes in the temperature trend in the body rather than to absolute values and this factor might also be implicated in the femoral dilatation. This question has been debated for many years but has not been definitely settled.

It is very difficult to ascribe a function to the increase in the blood flow to the skeletal muscles during body warming. Promotion of heat dissipation is not likely to be a major factor since this is primarily carried out through the skin circulation and this latter did not seem to be implicated. It is possible that this vascular response is peculiar to the nonshivering animal anesthetized with pentobarbital since it was not observed in the shivering dog anesthetized with Sernyl(1).

Summary and conclusion. 1. The mechanisms of vasodilatation in the femoral arterial bed which follow contact of the body with warm water were investigated in intact, skinned and sympathectomized dogs subjected to immersion hypothermia. 2. The femoral vasodilatation was found to be due to a reflex inhibition of the sympathetic vasoconstrictor tone triggered by the immediate contact of any part of the body with warm water.

1. Westin, B., Sehgal, N., Assali, N. S., *Am. J. Physiol.*, 1961, v201, 485.

2. Westersten, A., Herrold, G., Abbott, E., Assali, N. S., *IRE Transactions on Medical Electronics*, 1959, vME-6, 213.

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