

REACTIONS OF BLOOD VESSELS OF THE CAT'S STOMACH TO EXPOSURE OF THE HIND LIMBS TO WET AND COLD

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In acute experiments on 22 cats, cooling the limbs in most cases led to vasoconstrictor responses of the gastric vessels; less commonly dilatation of the gastric vessels or bi-phasic reactions were observed. As a rule these reactions began without a latent period. After division of the skin and nerves at the base of the limb no reactions of the gastric vessels to cooling of the limb were observed.

Investigations into the effects of local wet and cold on animals have been undertaken for several years at the writer's clinic [1-3]. During prolonged exposure of the dog's limb to wet and cold, sensory disorders were found, and persistent spasm of the limb vessels were detected by contrast angiography. Meanwhile the dogs lost their appetite, refused to eat, vomited, and lost up to 2.5 kg in weight.

These observations served as the basis for further experiments to study the effects of local exposure to wet and cold on the gastric vessels.

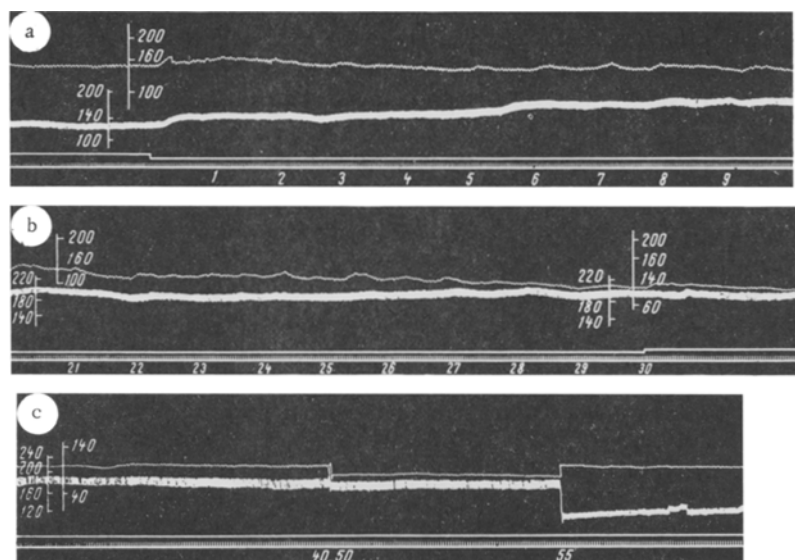


Fig. 1. Reaction of vessels to local cooling of cat's limbs in ice-cold water for 20 min. From top to bottom: general arterial pressure, perfusion pressure in gastric vessels, marker of stimulation, time marker. a, b, c) Successive recordings.

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EXPERIMENTAL METHOD AND RESULTS

In acute experiments on cats, tracheotomy, dissection of the vessels, and laparotomy were performed under ether anesthesia, and thereafter the experiment was carried out without anesthesia. The arterial pressure was recorded by a mercury manometer in the left carotid artery, and changes in the resistance of the vessels were measured by means of a perfusion pump in the left gastric artery under conditions excluding a collateral circulation to the stomach. The animals were immobilized with listhenon and maintained on artificial respiration. The hind limbs were immersed in ice-cold water up to the mid-thigh. Exposure to cold continued for 10 to 30 min. In 22 experiments 44 observations were made.

The latent period of reactions of the vessels to limb cooling was absent in 33 cases, and in 11 cases changes in pressure appeared after 10-40 sec. In 30 cases during exposure to cold the perfusion pressure was increased by 10-30 mm or more, in 4 cases it was reduced by 20 mm or more, and in 7 cases the response was biphasic in character; in 3 cases there was no response. As a rule the biphasic reaction began with a small or moderate increase in perfusion pressure followed by a gradual decrease to the initial level or below it for 20-30 mm.

The initial and subsequent reactions of the gastric vessels were clearly differentiated on the resistograms (Fig. 1). The initial reaction, with very few exceptions, was almost instantaneous and sharp. The subsequent increase in perfusion pressure took place gradually, to reach a maximum during the first 1-3 min of cooling, but in some cases only towards the end of exposure to cold.

In 4 tests, throughout the period of cooling regular, periodic rises and falls of pressure to the initial level or slightly below it were observed. These fluctuations of pressure ceased simultaneously with the end of cooling.

In nearly all experiments a prolonged after-effect of cold stimulation was observed, consisting of a persistent increase or, less frequently, decrease in the perfusion pressure. The longer the duration of cooling of the limb, the more persistent the after-effect. In four experiments the perfusion pressure continued to rise after the end of cooling for several minutes longer. Spontaneous recovery of the perfusion pressure 10-30 min after cooling was observed in only 8 cases, and in 36 tests no return to the initial level took place.

The reactions of the general arterial pressure to local cooling of the limb were characterized by an initial, transient elevation by 10-20 mm. Later, as a rule, the pressure fell gradually and considerably. Only in 7 cases was the pressure in the common carotid artery not reduced below its initial level. The perfusion pressure either continued to rise or, having reached its maximum, remained stable.

The decrease in arterial pressure developed much more slowly and was less marked in its degree than the increase in resistance in the perfused vessel. Against the background of a lowered arterial pressure, all visible fluctuations in its level corresponded to changes in the perfusion pressure and developed simultaneously with them, and vice versa. However, in these cases also the fluctuations of pressure in the gastric vessels were higher than in the common carotid artery. Usually the arterial pressure did not subsequently rise above its initial level, nor did it return spontaneously to normal after cooling.

The decrease in general arterial pressure in these experiments can be considered to be due to trauma associated with the method and to general hypothermia developing at the end of prolonged cooling of the limbs.

The instantaneous reaction of these resistive vessels to local cooling indicated the reflex mechanism of this phenomenon. This hypothesis is confirmed by the fact that a circular incision in the skin at the base of the limbs depressed the responses of the arterial and perfusion pressure only partially, while division of the skin and the femoral and sciatic nerves at this same level abolished the initial reaction of the vessels to cooling. Meanwhile, in two of the four experiments carried out after division of the skin and nerves the perfusion pressure in the left gastric artery was increased, and in one experiment it was reduced, 10 min or more after the beginning of cooling, indicating the action of humoral factors accumulating in the circulating blood during this period. Other evidence in favor of this hypothesis is given by the later responses of the vessels during cooling, and also by the prolonged after-effect.

The processes lying at the basis of responses of the gastric vessels to limb cooling may be regarded as taking place in two stages. The first stage is that of a rapid reaction, in the majority of cases one of spasm of the vessels due to reflex influences. This is followed by the action of vasoactive humoral substances.

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