

Fig. 6. The effect of distribution of the melanosomes on the early photo-voltage of frog skin. Upper frame: melanosomes aggregated, skin light. Lower frame: melanosomes dispersed by 2 h exposure to MSH ( $0.7 \times 10^{-4}$  g/ml.), skin dark. Both responses were recorded with the internal skin potential at +50 mV. Calibration: 200  $\mu$ V, 10 ms.

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## Cardiovascular Changes during Preparation for Fighting Behaviour in the Cat

It has been suggested recently that animals such as the cat and dog, when warned of an impending emotional or exertional task, anticipate the task with centrally induced cardiovascular changes similar to those that take place during the emotion or exertion itself<sup>1,2</sup>. Particular emphasis has been placed on muscle vasodilatation, preparatory to and independent of muscle contraction, mediated through sympathetic fibres supposed to be cholinergic, because the vasodilatation can be abolished by atropine<sup>1,3</sup>. Most of the data on which the "preparatory pattern" hypothesis is based, however, have been obtained by electrical stimulation of certain regions in the hypothalamus or subthalamus, from which are evoked either cardiac changes similar to those during exercise but without concomitant muscle activity<sup>2</sup> or cardiovascular changes as well as manifestations of defence behaviour<sup>1,4</sup>.

It should be realized, however, that the most quoted experiment on unanaesthetized, naturally behaving animals that may be relevant to this hypothesis, is that by Abrahams *et al.*<sup>5</sup> which showed an increase in muscle venous temperature, blocked by atropine, when "alerting" was induced by various sensory stimuli. The evidence it provides, however, rests on unverified assumptions that the venous temperature increase accurately reflected muscle vasodilatation, that there was no muscle activity which could have provided a metabolic vasodilatation, and that the behaviour was one preparatory to exertion or emotion.

We have analysed the cardiovascular changes during naturally elicited fighting in the cat<sup>6</sup> and found in our experimental situation a rather simple test of the "preparatory pattern" hypothesis with respect to emotion. Data<sup>6</sup> reported previously as well as those we present here were obtained from cats in which fighting was elicited as a natural response to an attack launched by another cat, which was electrically stimulated through electrodes chronically implanted in a portion of the midbrain from which attack could be evoked at will. The two cats, the attacking cat and the subject of the recording experiment, remained on opposite sides of a partitioned cage between trials, and there was fighting whenever the partition was raised, the brain-stimulated attacking cat was moved towards the other cat, and the latter responded with hissing and striking. As soon as the subject responded with hissing and striking the attacking cat was withdrawn, the partition was lowered and the trial was terminated.

The cardiovascular changes of preparation for fighting could be seen in the interval between the moment that the partition was opened and the moment that the subject responded with hissing and striking to the attack of the brain-stimulated cat. Some changes began even before the opening of the partition in response to the noise made while the attack cat was grasped to prepare for the trial. To see these changes more clearly and to follow their time course, we alternated in a random order fighting trials with other trials in which the same procedure was used up to the point of moving the attack cat towards the subject cat, but then no brain stimulation was given, no attack took place, and the subject simply flinched, retracted its ears and watched the attack cat closely. These trials of confrontation without fighting we have called trials of preparation for fighting.

The following cardiovascular variables were continuously measured in the subjects. Mean arterial pressure was obtained from a catheter implanted in the right femoral artery. Cardiac output, superior mesenteric flow and external iliac flow were obtained from electromagnetic flowmeters chronically implanted on the ascending aorta, superior mesenteric artery and left external iliac artery, the instantaneous flow records being electronically integrated at 2 s intervals. Heart rate was recorded by cardiostachygraphy. Total peripheral, superior mesenteric, and external iliac conductances were computed for each 2 s interval by dividing flow by mean pressure. Conductance was preferred to its inverse, resistance, as an index of vascular diameter changes for reasons pointed out elsewhere<sup>6,7</sup>. In addition to the direct cardiovascular measurements electromyograms from left hind limb (plate electrodes on dorsal and ventral muscles of the thigh) and neck muscles were recorded on a twelve channel Grass P7 polygraph. Statistical analysis of the data was performed by *t* test of differences.

Results during fighting and preparation for fighting obtained from one of the six cats that were studied for all variables are compared in Fig. 1. The cardiovascular changes are average responses from six episodes of preparation for fighting and from six episodes of fighting during which the cat used the hind limb for support. Preparation for fighting, both in the shorter periods which preceded the actual fighting, and in the longer trials in which no fighting was elicited, was associated with bradycardia,

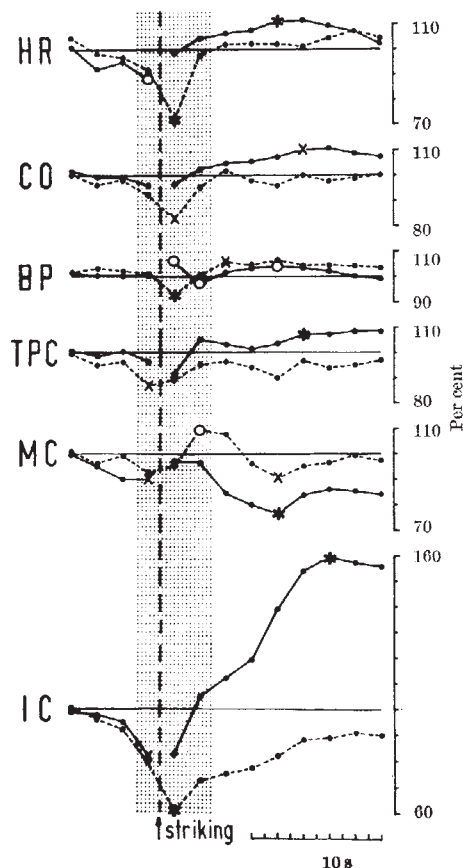


Fig. 1. Each dot is the mean of measurements taken during six episodes of preparation for fighting (dashed lines) or of fighting (continuous lines) in cat *M*. The shaded area represents the time during which partition was opened, and the vertical line (related to fighting episodes only) indicates the first striking of the attacked cat, that is the beginning of fighting. Interruption of the continuous lines immediately before the strike signal is due to the variable time between opening of partition and beginning of fighting in the different fighting episodes. All values are expressed as percentage changes with reference to baseline measurements when the cat was quiet before the trials. Absolute values for baseline are as follows (baseline means for preparation for fighting precede those for fighting). Heart rate (HR), 198 and 226 beats/min; cardiac output (CO), 338 and 350 ml/min; mean blood pressure (BP), 100 and 103 mmHg; superior mesenteric flow, 26.2 and 24.7 ml/min; left external iliac flow, 21.5 and 25.8 ml/min. Total peripheral (TPC), mesenteric (MC) and iliac (IC) conductances were calculated from flows and BP. Results of statistical analysis of data are indicated by asterisks ( $P < 0.01$ ), crosses ( $P < 0.05$ ) and open circles ( $P > 0.05$ ).

decreased cardiac output, decreased total peripheral conductance, mesenteric vasoconstriction and a stronger constriction of the external iliac bed. On the other hand, beginning of fighting, signalled by the first forelimb strike and marked in Fig. 1 by the vertical dashed line, was accompanied by cardiovascular changes many of which were in the reverse direction to those noticed during preparation for fighting. Heart rate, cardiac output and total peripheral conductance all increased for a prolonged period, and the iliac vascular bed underwent an extremely strong dilatation. Only the mesenteric bed showed vasoconstriction both in fighting and preparation for fighting, though the constriction was stronger during fighting. Blood pressure was not greatly changed in either condition, but it seemed to be maintained by different mechanisms: a fall in cardiac output opposed by overall vasoconstriction in preparation for fighting, while increased cardiac output was opposed by overall vasodilatation in fighting.

The cardiovascular pattern of fighting was extremely reliable, obtained in all of the cats tested, enduring for some time after the fighting episode and, with one slight exception, always statistically significant. The pattern of preparation for fighting was weaker and not usually as

consistent, however. It was also of shorter duration, even in those trials in which preparation was not rapidly superseded by fighting. The heart rate and cardiac output decreases were rather variable responses, present and quite conspicuous in some cats and trials, and absent in others. But a change opposite to the basic pattern, namely tachycardia instead of bradycardia, was observed only once. On the other hand, the decreases in iliac, mesenteric and total peripheral conductances were the most constant and consistently observed effects, and were statistically significant in all cats. Statistically significant iliac vasoconstriction occurred during preparation for fighting even when the sympathetically controlled portion of cutaneous circulation (which in the cat is limited to the paw<sup>8</sup>) was excluded by introducing the paw into a sealed plethysmographic cuff the pressure of which was then brought to values higher than the simultaneously recorded arterial pressure. In these conditions iliac vasoconstriction represented purely muscular vasoconstriction.

Our experiments suggest two principal conclusions. One is that cardiovascular changes during naturally elicited preparation for fighting are different from those implied from electrical stimulation of the hypothalamus in anaesthetized animals. In particular, there is no muscle vasodilatation unless the muscle is contracted, this dilatation being apparently metabolic in origin<sup>9</sup>. If vasodilatation of sympathetic muscle ever occurs during preparation for fighting it must be completely concealed by an overwhelming vasoconstrictive influence. Of course, we do not dispute that an atropine-sensitive muscle vasodilatation can be induced in the cat by proper electrical stimulation of the brain. Our results suggest, however, that the sympathetic vasodilating mechanism does not play an important part in preparation for emotional behaviour.

The second conclusion from these experiments is that cardiovascular changes during preparation for fighting and during fighting are different, at least insofar as the skeletal musculature is in use during fighting, and not in use during preparation for fighting. Some of these differences may derive simply from the exertion component of fighting which may overwhelm and conceal other central neural influences common to fighting and preparation for fighting. Before judging this, we need to know which of the cardiovascular changes are centrally elicited, and which are reflexly induced or modified. We hope that results of forthcoming experiments using sino-aortic deafferentation, vagotomy, sympathectomy or adrenalectomy, will enable us to answer these questions and shed some light on the mechanisms underlying the changes observed.

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