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Cardiovascular changes during naturally elicited fighting behavior in the cat

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Adams, David B., Giorgio Baccelli, Giuseppe Mancia, and Alberto Zanchetti. *Cardiovascular changes during naturally elicited fighting behavior in the cat.* Am. J. Physiol. 216(5): 1226–1235. 1969.—Fighting was elicited in unanesthetized cats as a natural reaction to another cat, in which attack was induced by brain stimulation. The cats were implanted chronically with a femoral cannula, for measuring arterial pressure, and three electromagnetic flow probes around the ascending aorta, the superior mesenteric artery, and one external iliac artery. Muscle blood flow was judged from external iliac flow when its visceral branches were tied off and paw circulation eliminated by an occluding cuff. The most striking cardiovascular event during fighting was a strong, short-latency dilatation of muscle blood vessels, related to muscle activity itself. At the same time there was a visceral vasoconstriction and a vasoconstriction in inactive muscles. No evidence was found that a sympathetic muscle vasodilatation is a dominant factor in the cardiovascular changes during fighting.

emotional behavior; hemodynamics; muscle blood flow; visceral blood flow; cardiac output; arterial pressure; heart rate; cholinergic sympathetic vasodilator system

Despite considerable interest in the bodily changes and especially cardiovascular aspects of emotion, from the time of the psychological controversies of William James and Sir Charles Sherrington to the present day, our knowledge is still quite limited (33). Progress has been made recently since Folkow's group (18, 21), as well as other authors (1), have carefully described the cardiovascular changes induced in anesthetized cats or dogs by electrically stimulating an area in the hypothalamus where stimulation without anesthesia can evoke defense behavior resembling the naturally occurring one. As a result of this work a pattern of cardiovascular changes, usually referred to as the cardiovascular pattern of emotion, has been reported to consist of arterial pressure increase (25); sympathetically induced cardiac stimulation (increase in cardiac output (19), heart rate (19, 25), stroke volume (19), and contractile force of the heart (25)); sympathetically mediated visceral (renal (16), intestinal (14, 15, 21)) and cutaneous (15) vasoconstriction; and dilatation in muscle blood vessels (1, 15), a selective response brought about through the so-called cholinergic sympathetic fibers (30). The bearing of these investigations on the problem of cardiovascular adjustment during emotion depends, however, on three unverified assumptions: *1*) that electrically induced behavior is really comparable to the natural one; *2*) that anesthesia (or decerebration), which abolishes or severely hampers defense behavior, may not modify its cardiovascular concomitants; and finally *3*) that none of the reported changes results from costimulation in the hypothalamus of cells or fibers having no relationship to defense behavior. In a recent study in which the aforementioned cardiovascular pattern was evoked under anesthesia by the appropriate hypothalamic stimulation, and then stimulation was repeated in the unanesthetized state, it was found that all cardiovascular effects were produced at voltage levels below those at which overt behavior was obtained, as well as from other nearby electrode sites from which emotional behavior could not be elicited at all (11, 31).

Unfortunately, only few studies have measured cardiovascular changes during naturally elicited emotional behavior, and most of them suffer from important limitations either in recording technique or in the behavioral model employed. Cannon and associates (13) mentioned only indirect recording of arterial pressure in cats during "excitement," and more recently Abrahams et al. (2) monitored femoral venous blood temperature as an index of local blood flow in the cat and reported a cholinergically mediated vasodilatation in muscle vessels in response to light sensory stimuli simply inducing alerting, a response he regarded as preparatory to emotional activity. Excitement following a loud noise has been reported to produce an increase in heart rate, cardiac output, pressure, and coronary flow in the dog (24).

Investigation on human subjects has been carried out during stressful interviews or elaborate fear- or anger-producing situations (6, 26), but cardiovascular measurements have been rather limited, usually indirect, and often unreliable (26). The most detailed pattern is provided by the careful work of Brod and colleagues (7, 12, 17), who have employed as a stimulus a mental arithme-

tic task. They have observed increase in blood pressure, heart rate, and cardiac output, decrease in renal and cutaneous flows, and an increased muscle blood flow, a pattern not dissimilar to that described by Folkow in his brain-stimulated anesthetized cats.

The experiments which are to be reported here have been planned to avoid the shortcomings of previous work in animal and man. First, we induced in unrestrained unanesthetized cats a natural type of emotional behavior, namely fighting in response to an attack by a second brain-stimulated animal. This behavior is a comparatively natural one, nonetheless it can be easily controlled by the investigator and reliably reproduced many times in the course of the experimental session without changes in behavior or injury to the animal. Second, indwelling electromagnetic flow probes around the ascending aorta, and superior mesenteric and external iliac arteries, as well as an arterial cannula for pressure recording, provided faithful, direct, and continuous assessment of cardiovascular adjustment during fighting.

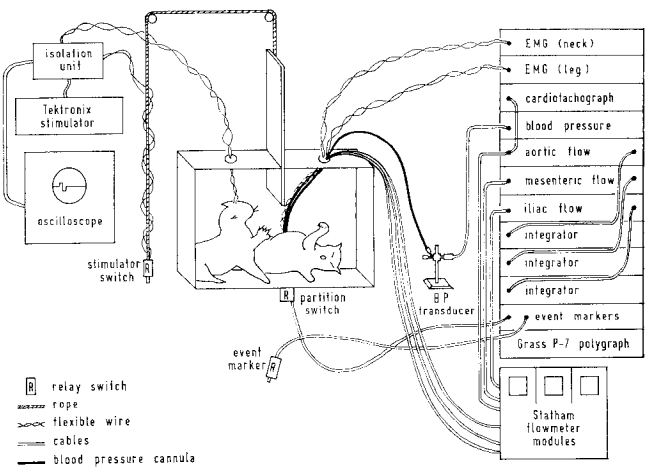

FIG. 1. Schematic representation of general experimental setup, showing partitioned cage with brain-stimulated cat (on left) attacking subject cat (on right), which fights back while several cardiovascular variables are recorded by means of implanted devices on polygraph on extreme right.

METHODS

The general experimental situation is characterized in Fig. 1. In the left compartment of a cage partitioned by a movable opaque screen, we placed a cat having an electrode chronically implanted in the mesencephalic gray matter, so that electrical stimulation through this electrode invariably elicited attack behavior. This cat was only used as a stimulus for evoking a natural fighting response in our experimental subject, which was placed in the right compartment of the cage. Adult cats were selected as subjects on the basis of a consistent strong response of hissing and striking to the attack launched by the brain-stimulated cat whenever the partition was raised (ref 3 for further details). Less than one-half of the cats from the colony reacted well during preliminary tests and could be used as subjects. The data reported in this paper come from the following six cats: *cat R*, male, 3.4 kg preoperative weight; *cat G*, male, 3.0 kg; *cat O*, male, 3.1 kg; *cat S*, female, 2.5 kg; *cat B*, female, 2.8 kg; *cat M*, female, 2.7 kg.

The following cardiovascular measurements were made in the six cats, before, during, and after fighting against the brain-stimulated attacking cat. Blood flow through the initial portion of the ascending aorta, the superior mesenteric artery and the left external iliac artery was measured by means of Statham electromagnetic flow probes (K probe of the hinged type, 6–7 mm i.d., for the aorta; and Q probes of the slot type, 1.5–2 mm i.d., for the two peripheral arteries), connected to three Statham M-4001 modules and power supply. The sensitivity of flowmeters had been checked by calibration with blood and with saline before implantation in the cats, and was rechecked at the end of chronic recording, after as much as a month apart, with results that agreed within 10%. Zero-flow level was the diastolic level in the case of the aorta, whereas for the two peripheral arteries it was repeatedly established by occlusion of the descending aorta by transient pulling of a snare implanted well well above the origin of the superior mesenteric artery (above root of celiac artery). Preliminary experiments showed that occlusion of the descending aorta produced the same values for zero flow as were produced by occlusion of mesenteric and iliac arteries distal to the implanted probes.

The three flow probes as well as the aortic snare were aseptically implanted under pentobarbital anesthesia, following left thoracotomy and a wide midline opening of the abdomen. The animals usually recovered in less than 1 week during which time they were maintained in a thermostatic cage and under antibiotic treatment. The left external iliac artery was implanted after tying all its visceral branches (deep caudal epigastric artery and external pudendal artery), so that the probe recorded only the blood flow to the hindlimb. Care was taken to avoid injury to the nerve plexuses around the mesenteric and iliac arteries in order not to impair vasomotor reactions in the beds supplied by these arteries. Carotid occlusion, performed after the experiments, was used to show in all cases the appropriate vasoconstriction. Cables from the flow probes and one end of the snare were passed through the paravertebral muscles and the overlying skin, and protected in a leather packet sewn to the skin of the back. Cables from two plate electromyographic electrodes positioned on dorsal and ventral muscles of the left thigh (i.e., the same leg the flow of which was monitored) and from two needle electrodes for neck electromyogram were also passed into the leather packet.

One or two days before the recording session, under transient ether anesthesia, a polyethylene tubing of a sufficiently rigid wall to prevent damping of the pressure pulse, was inserted into the right femoral artery, and its free end also passed into the back packet. From this time on, the animal was heparinized. For recording of blood

pressure the arterial cannula was connected with a Statham strain-gauge transducer feeding into the bridge circuit of a d-c preamplifier of a 12-channel Grass P7 inkwriter. On three channels of the same polygraph were displayed the instantaneous flow signals of the three flowmeters. Cardiac output (less coronary flow), mesenteric blood flow and blood flow through the left hindlimb were continuously computed from the instantaneous flow curves by integrating amplifiers (Grass 7P10) automatically reset at 2-sec intervals, and were displayed on three other channels of the polygraph. Heart rate appeared on a further channel calculated beat by beat by a cardiotachograph triggered by the aortic flow pulse. Electromyograms from neck and leg were also displayed on the polygraph. The time during which the cage partition was opened, and the cats confronted each other, was automatically recorded on the polygraph, while the striking movements made by the recorded cat were signalled by a second event marker manually triggered by an observer.

In four of the six cats iliac blood flow was also recorded after blocking blood flow to the ipsilateral paw. To this effect the paw of the left hindleg was placed into a rubber cylinder of a suitable diameter, closed on the distal end, and the proximal end was sealed with tape to the skin of the leg above the ankle. The cylinder was connected through a tubing to an air pump which could produce an air pressure greater than the systolic blood pressure simultaneously recorded from the contralateral femoral artery of the cat. Air pressure inside this occlusion cuff was directly measured by a second Statham pressure transducer and also recorded on the polygraph. Occlusion of the hindpaw circulation was started 10–15 sec before a trial, and interrupted 20–30 sec after the trial was terminated. No animal ever behaved as feeling some pain, probably because occlusion was always short lasting.

At least six trials were recorded in each cat during each of the various experimental situations described under RESULTS. Two-second measures of cardiac output, mesenteric and iliac flow, and heart rate could be taken directly from integrator and cardiotachographic tracings. Mean arterial pressure was computed for the same interval as systolic plus twice diastolic pressure divided by three. Total peripheral, iliac, and mesenteric conductance measurements were computed for each 2-sec interval by dividing each flow by mean arterial pressure. Faced with the choice of computing conductance or resistance index in order to remove driving pressure as a factor in flow changes, we have chosen to use conductance for two reasons: first, we wished to depart as little as possible from the form of the flow changes themselves, and second, we have found it necessary on occasion to calculate zero arterial flows for which conductance measures are much better suited than resistance which must go to infinity in these conditions. Other reasons favoring the use of conductance have been presented by Stark (27).

The pattern and time course of the cardiovascular changes which occurred during and after the short flurry of striking of each fighting episode was remarkably constant from trial to trial within each cat and experimental condition. Thus it was possible to meaningfully average the data from many trials using striking as the fixed point in time and then carry out statistical analysis. Another independent and constant pattern of cardiovascular changes could be seen as related to the opening of the partition between the two cats which occurred from 2 to 10 sec prior to the striking. To meaningfully average and analyze the pattern we made a separate series of means for the changes occurring before striking, using opening of the partition as a fixed point in time. This procedure is reflected in the figures where a break will be seen between the mean data before striking, time locked with the opening of the partition, and the mean data during and after striking, time locked with the first striking. The number of means calculated after opening the partition is different from cat to cat because of the different interval between partition opening and striking in the various cats. Statistical analysis was accomplished by use of a t test of differences, comparing values of a given 2-sec interval after striking or opening of the partition with independently obtained base-line values. Base-line values were obtained from 2-sec intervals randomly selected from a 30-sec period of quiet resting immediately prior to the trial.

RESULTS

All data were obtained from episodes of naturally elicited fighting behavior which included hissing, striking, and ear retraction. In order to understand the cardiovascular events during fighting we found it necessary to distinguish several stages of the event: a base-line period of quiet resting, a "preconfrontation" period, a "confrontation" period, period of the fighting itself, and a recovery period. During the preconfrontation period, usually from 2 to 6 sec in duration, the experimenter prepared the attack cat for its brain-stimulated attack, causing some noise which could alert the other cat that an attack could soon occur. The confrontation period, which varied from 2 to 10 sec in duration, was the time beginning with the opening of the partition between the two cats and ending with the first striking movement by the cat from which we were recording. Ear retraction and flinching usually occurred during the confrontation period as the cat presumably prepared to defend itself. The fighting itself was purposely confined to a few second duration in most cases and then the partition was immediately closed, separating the cats once again. Some trials of longer fighting (more than 10 sec) were also carried out in each cat. Cardiovascular changes during longer fighting episodes were similar to those of shorter fighting, but they varied more from trial to trial, depending on the timing and sequence of movements.

It was observed from the beginning that the cardiovascular events during fighting were heavily dependent on the amount and form of muscle activity of the cat. Therefore, two standardized types of fighting were

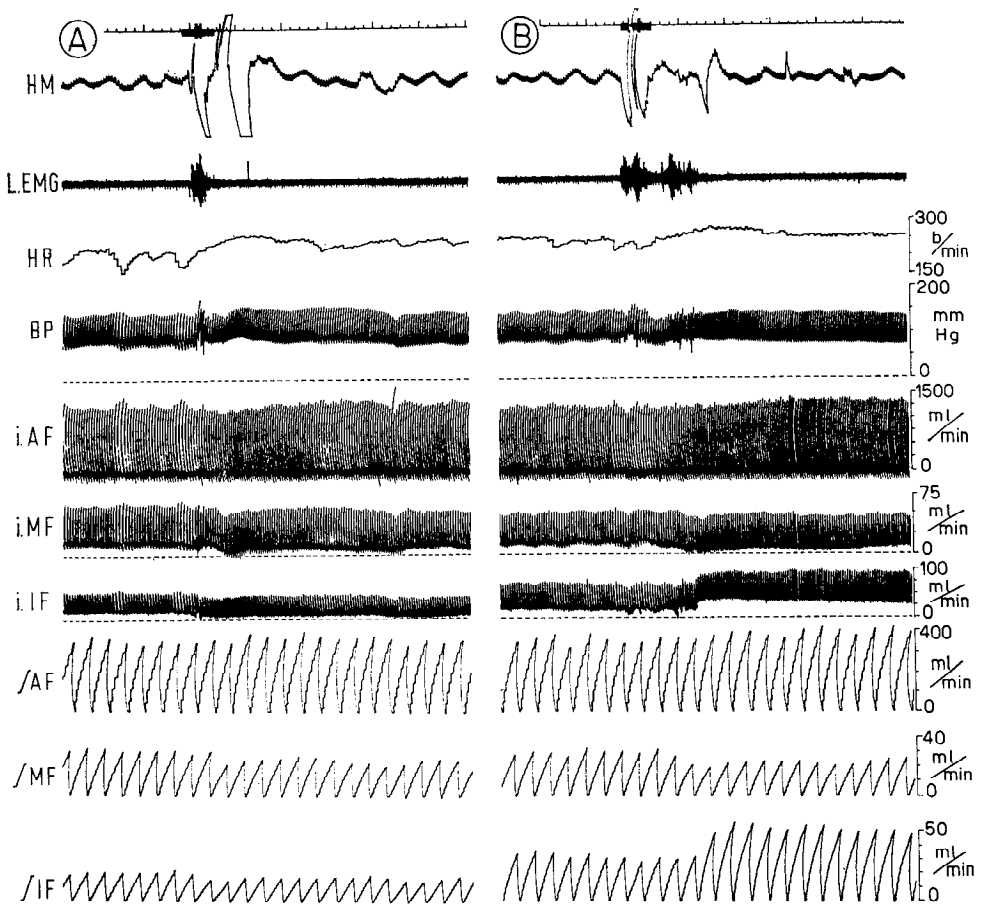

FIG. 2. Cardiovascular changes during nonsupportive (*A*) and supportive (*B*) fighting in the same cat. In this figure, as well as in Fig. 7, the first tracing from the top is time (1 and 5 sec), and event markers for raising the cage partition (horizontal heavy line) and for forelimb strokes (vertical heavy lines). HM: head movements; LEMG: electromyogram of the left hindlimb; HR: heart rate; BP: arterial blood pressure; iAF: instantaneous flow through the ascending aorta; iMF: instantaneous flow through the superior mesenteric artery; iIF: instantaneous flow through the left external iliac artery; ∫ AF ∫ MF, ∫ IF: 2-sec integrations of the above flows. Calibrations on right for both *A* and *B*.

elected for investigation: nonsupportive and supportive fighting. In nonsupportive fighting the animal lay on its side, hissed, and struck with the forelimbs during fighting, but did not support itself with the hindlimbs or use them in the fighting. In this type of fighting there was minimal change in muscle activity of the hindlimbs as measured by the EMG. In supportive fighting the animal supported itself on its hindlimbs during the base-line period, in either a sphinx or a sitting position, and then used the hindlimbs for balance and support during the fighting. Muscle activity in the hindlimb was considerable during this behavior according to the EMG but not always evident in terms of overt movements. Other types of fighting responses, those in which the cat rose from a lying position to standing during the fighting, or in which the cat rolled over onto its side changing from standing to lying during the fighting were not analyzed since the results were considered a mixture of fighting and posture change.

In spite of the many fighting episodes in which the cat participated, there were not usually any consistent changes in base-line cardiovascular values before and after the initial episode or during the course of the experimental sessions, such as might have been expected from circulating hormones or changing central influences on the resting state of the cardiovascular system.

Original records from nonsupportive and supportive fighting are presented in Fig. 2, showing, besides arterial pressure, instantaneous as well as integrated measures of cardiac output, external iliac flow, and superior mesenteric flow. Neck and left hindleg movement, forelimb striking, and raising of the cage partition are also monitored. Figures 3–6 summarize the data from all cats relating to heart rate and cardiac output (Fig. 3), arterial pressure and total peripheral conductance (Fig. 4), mesenteric flow and conductance (Fig. 5), and iliac flow and conductance (Fig. 6). For each variable the data are presented as means of six episodes of supportive fighting, and separately of nonsupportive fighting, for each of the six cats. The changes have been expressed in percent to facilitate comparison between cats, but absolute values are also indicated. Statistical significance has been indicated for selected points.

Heart rate (Fig. 3, left column). Fighting was followed by a significant tachycardia in all cats and conditions, which reached its peak from 4 to 10 sec after initiation of fighting and then returned gradually towards base-line values often remaining slightly above base-line for as much as 1 min later. The peak tachycardia ranged from 10 to 40% above base-line values. There appeared to be some inverse relationship between resting heart rate and the magnitude of the response: the greatest increases in heart rate occurred when base-line heart rate was lowest. On the whole tachycardia was of similar magnitude both in supportive and in nonsupportive fighting. No tachycardia

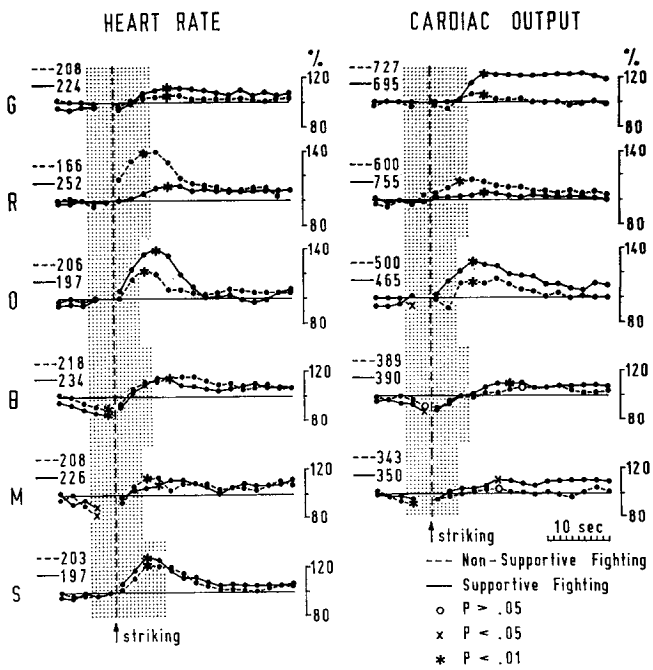

FIG. 3. Changes in heart rate (left column) and cardiac output (right column) during nonsupportive fighting (dashed lines) and supportive fighting (continuous lines). In this figure, as well as in Figs. 4–6, each dot represents the mean of 2-sec measurements performed during six fighting episodes in each of the two different kinds of fighting in each of six cats (identified by letters on the left). Shaded area is time during which the cage partition was raised, while vertical line indicates first striking of the attacked cat (i.e., beginning of fighting). Interruption of line joining dots is due to the variable time between raising of partition and beginning of fighting in the different trials and cats. Data are expressed as percent changes with reference to base-line values measured while the animal was quiet prior to the trial. Means of absolute base-line values (= 100%) are given for each cat at the left of each column, both for nonsupportive and supportive fighting trials. Heart rate is given in beats/min, and cardiac output in ml/min. Results of statistical analysis, when performed, are indicated by asterisks ($P < 0.01$), crosses ($P < 0.05$), and open circles ($P > 0.05$).

was ever seen during the preconfrontation and confrontation periods; instead there was sometimes a significant bradycardia.

Cardiac output (*Fig. 3, right column*). Cardiac output generally followed the changes in heart rate. When there was a bradycardia in the confrontation period, there was a corresponding fall in cardiac output; and along with the tachycardia which began with the onset of fighting there was a rise in cardiac output. The slope of the increase in cardiac output was never as steep as that of the tachycardia, however, as stroke volume fell slightly during the period of rapidly rising heart rate. Peak values of cardiac output ranged from means of 4 to 30% above base-line values and were significant for all cats in supportive fighting and in three out of five for nonsupportive. After the peak values of heart rate and cardiac output had been reached in some cats during supportive fighting, the cardiac output remained higher than would have been expected from heart rate alone, the stroke volume increasing above base-line levels.

Arterial pressure (*Fig. 4, left column*). The general pattern of mean blood pressure changes during short fighting episodes consisted of five stages, not all of which were present in each episode: *1*) a slight fall in pressure during confrontation, consistently present in one posture condition in four cats; *2*) transient and highly irregular increases, sometimes for only one stroke in duration, corresponding to striking motion by the cat, inconsistent and not usually statistically significant; *3*) a fall in pressure for the first few seconds after striking, almost always significant in supportive fighting and sometimes also in nonsupportive fighting; *4*) an increase in pressure beginning 4–6 sec after striking, the pressure rising above base-line values in nonsupportive fighting, but usually simply returning towards base line in supportive fighting; and *5*) a return to base-line values within 10–20 sec after striking. In some cats the blood pressure remained rather low for 1 min or so following supportive fighting only.

The same pattern of mean blood pressure changes could be seen in both supportive and nonsupportive fighting, but in general the pressure tended to fall more and remain lower for a longer time in supportive fighting, and to rise in nonsupportive fighting. Only in supportive fighting did mean blood pressure remain significantly lower than base-line values for more than a few seconds after fighting, and only in nonsupportive fighting did mean pressure ever increase significantly above base-line values.

A more detailed view of diastolic and systolic components of the mean blood pressure changes may be seen in

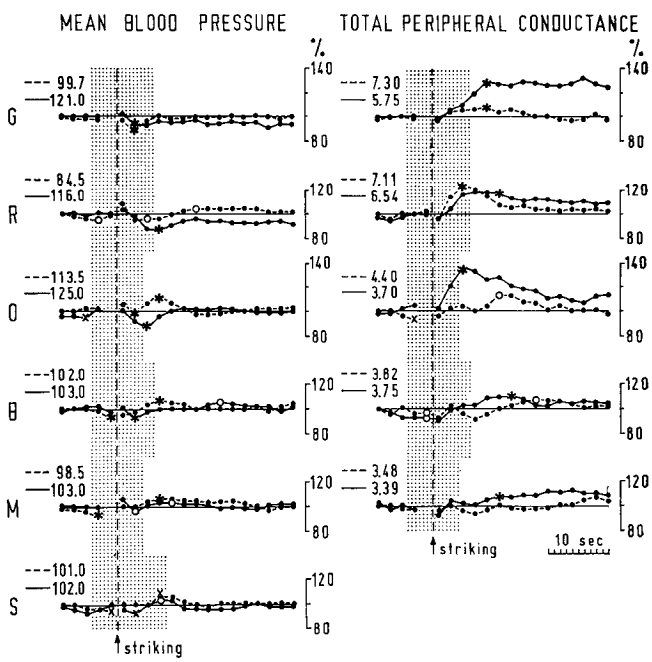

FIG. 4. Changes in mean blood pressure (left column) and total peripheral conductance (right column) during nonsupportive fighting (dashed lines) and supporting fighting (continuous lines). Absolute base-line values are given on the left of each column in mm Hg (mean blood pressure) and in units (ml/min per mm Hg) (total peripheral conductance). All other explanations as in Fig. 3.

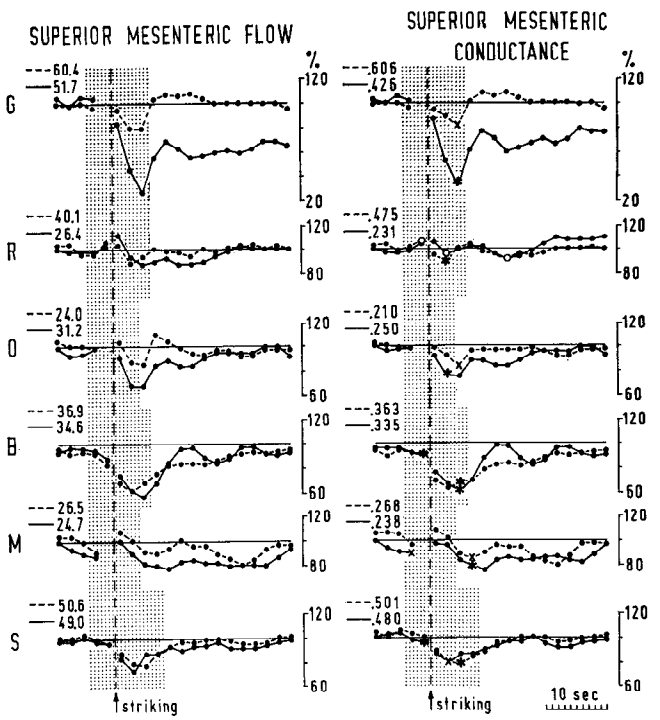

FIG. 5. Changes in superior mesenteric flow (left column) and superior mesenteric conductance (right column) during nonsupportive fighting (dashed lines) and supportive fighting (continuous lines). Absolute base-line values are given on the left of each column in ml/min (flow) and in units (ml/min per mm Hg) (conductance). All other explanations as in Fig. 3.

the examples of Fig. 2. The fall in pressure during confrontation and the transient increase during striking were usually diastolic changes for the most part, whereas the initial fall in pressure following striking was often mostly systolic. The final rise in pressure, toward or above base line usually began as a more pronounced diastolic rise, but systolic pressure then continued to rise later and differential pressure increased. Thus, in general, differential pressure tended to be slightly higher during confrontation, drop below normal as soon as striking occurred, and then return to or above normal as mean pressure returned to base line afterwards.

Total peripheral conductance (*Fig. 4, right column*). There was a significant increase in total peripheral conductance during supportive fighting, although magnitude (range of the means 10–35 %) and duration of the change varied considerably in the different cats. In all except one case of nonsupportive fighting, there was little change in total peripheral conductance. The one exceptional case, in which a considerable increase was observed, is discussed below. During the confrontation period, total conductance varied slightly, but in one cat in nonsupportive posture there was a small but significant decrease.

Mesenteric flow and conductance (*Fig. 5*). There was a vasoconstriction in the bed of the superior mesenteric artery during fighting in all cats, although the initial vasoconstriction differed greatly in magnitude, ranging from means of 5 to 68 % in different cats. The magnitude of the vasoconstriction also varied considerably in different episodes within the same cat, although statistical analysis shows that the means were significantly different from controls in all except one case. The form of the vasoconstriction was curious, consisting of a series of waves at intervals of 8–14 sec, the first wave usually being the largest, followed by a return to base line and then further oscillatory constrictions. A preparatory mesenteric vasoconstriction was observed during the confrontation period in half of the cats.

Iliac flow and conductance (*Fig. 6*). If the hindlimbs were used for support during the fighting there was an immediate strong vasodilatation of the external iliac bed reaching a peak of 50–150 % increase within 12 sec and then slowly returning to base line over a period of several minutes. This vasodilatation was dependent on muscle activity itself, since if the hindlimbs were inactive during the fighting as in nonsupportive fighting, there was a vasoconstriction instead (range of means 10–40 %). In

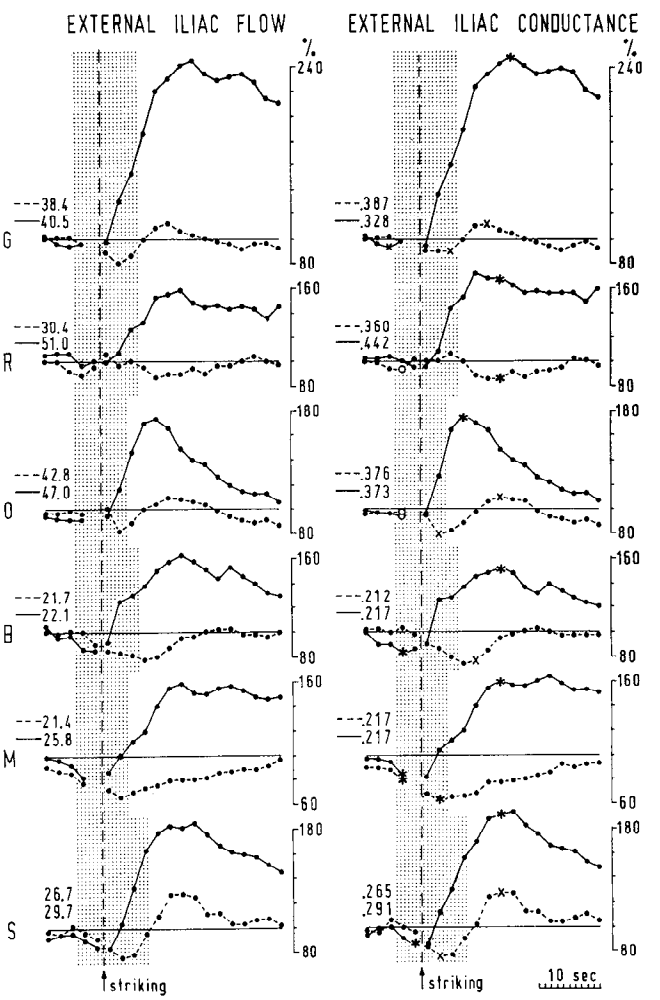

FIG. 6. Changes in left external iliac flow (left column) and left external iliac conductance (right column) during nonsupportive fighting (dashed lines) and supportive fighting (continuous lines). Absolute base-line values as in Fig. 5; all other explanations as in Fig. 3.

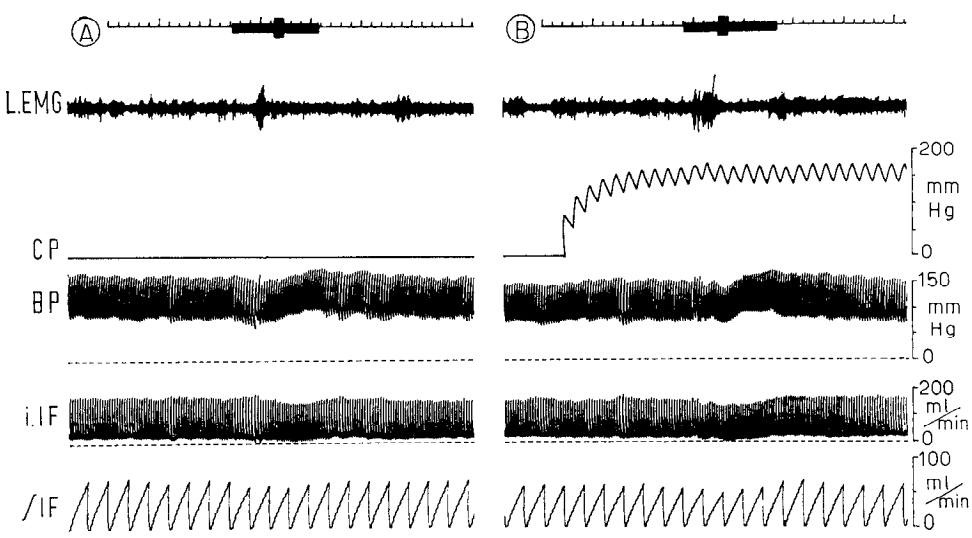

FIG. 7. Vasoconstriction in left external iliac bed during nonsupportive fighting, both with hindpaw circulation included (*A*) and excluded (*B*). CP: pressure in plethysmographic cuff around left hindpaw. All other abbreviations as in Fig. 2.

some cases of nonsupportive fighting the vasoconstriction was interrupted and followed by a short-lasting vasodilatation which appeared to be related to some muscle contraction which was indicated by the EMG despite the absence of strong movement or use of the limb for support. The vasodilatation during supportive fighting was significant at the 1% level in all six cats, whereas the vasoconstriction during nonsupportive fighting was significant at the 5% level in all cats. In preparation for the fighting a significant iliac vasoconstriction occurred in four cats.

The vasoconstriction during nonsupportive fighting was obviously unrelated to the visceral components of external iliac flow, as these had previously been eliminated by tying all visceral branches as stated in METHODS. It could not be maintained, however, that iliac vasoconstriction represented constriction of the hindleg muscle bed, as it might result mainly or even exclusively from a constriction in cutaneous vessels. As it is known that sympathetic control of skin blood flow is mainly limited to the paw (28, 29), six trials of nonsupportive fighting were carried out in each of four cats after the blood flow to the left paw (i.e., ispilaterally to the iliac flowmeter) had been eliminated by enclosing the paw into a suitable occlusion cuff and raising the air pressure inside the cuff above systolic arterial pressure values (see more details under METHODS). As shown in Fig. 7, even in these conditions did iliac vasoconstriction regularly occur during nonsupportive fighting.

Control procedures. Additional procedures were carried out in some cats in order to check some of the experimental conditions. The possibility that the "cholinergic" muscle vasodilator mechanism was for some reason impaired in our cats, was ruled out as follows. One cat (*cat R*) from which a vasoconstriction of the iliac bed had been observed during nonsupportive fighting was subsequently anesthetized with a mixture of chloralose (45 mg/kg) and urethan (0.4 g/kg). A stereotactically oriented electrode was inserted into a convenient site of the hypothalamus, and upon stimulation with rectangular pulses (62.5/ sec, 1 msec duration, 0.45 ma) a marked iliac vasodilatation was observed. As expected, this iliac vasodilatation was completely abolished by methylatropine (1 mg/kg).

Two types of observations have been employed in order to check the hypothesis that, because of an emotional reaction of our cats to the cage environment, the cholinergic muscle vasodilator mechanism was already maximally activated by base-line conditions, and hence no further cholinergic vasodilatation could occur during fighting. First, external iliac blood flow and conductance were compared in the same animals during the intervals between nonsupportive fighting trials and later on when the cats were studied in another cage during natural sleep (23). The mean values for six trials in each cat were very similar in both conditions. In *cat G* iliac conductance was 0.387 units (base line before nonsupportive fighting) and 0.385 units (light synchronized sleep); in *cat R* 0.360 and 0.390 units, respectively; in *cat O* 0.376 and 0.352 units; in *cat B* 0.212 and 0.250 units; in *cat M* 0.217 and 0.181 units. No sleep studies were carried out in *cat S*. Second, three cats (*G*, *M*, *S*) were given large doses of methylatropine (1 mg/kg iv) so as to block any peripheral cholinergic synapses. Base-line values of external iliac flow and conductance before nonsupportive fighting were never lower after methylatropine than before. This is definitely against the possibility that a cholinergically mediated iliac vasodilatation was operative in our base-line conditions.

The possiblity that some of the flow measurements during fighting might be altered by violent movements of the cats shifting the base line (zero flow) of the flowmeter, was also ruled out in control experiments. When the descending aorta was occluded by the implanted snare during fighting movements, the base line moved to its precalibrated position, and no shift from the zero line was ever observed.

DISCUSSION

The data presented above provide a clear picture of the cardiovascular pattern during short-lasting fighting. The most striking event and the most important for an understanding of the general pattern, is a strong, short latency and a relatively long-lasting dilatation of muscle blood vessels (as judged from the external iliac bed), which is related to muscle activity itself. At the same time there is a visceral vasoconstriction (as judged from the superior mesenteric bed) and a constriction in muscle beds where there is no change in muscle activity. As a consequence, if hindlimb muscles do not participate to fighting (nonsupportive fighting), total conductance usually remains relatively constant or only slightly increases, apparently balanced by the opposing actions of vasodilatation in the active forelimbs and of vasoconstriction in viscera and inactive hindlimbs. On the other hand, if forelimbs and hindlimbs are both involved in the fighting response (supportive fighting), visceral vasoconstriction cannot prevent total conductance from increasing with a time course reflecting that of muscle vasodilatation. The rise in peripheral conductance usually produces a slight initial fall in arterial pressure, especially in supportive fighting, but pressure then returns to normal in most cases immediately, and in nonsupportive fighting it often reverses to a slight transient increase, due to an augmented cardiac output mainly caused by tachycardia. The muscle vasodilatation and consequent increase in total peripheral conductance usually continue for several minutes after the fighting is over, but at this stage arterial pressure is usually balanced by a continued higher level of heart rate, cardiac output, and, in some cases, stroke volume.

Our experimental design, and the means employed for analyzing the data, also provided information on the cardiovascular pattern preparatory for fighting, i.e., the responses during preconfrontation and confrontation periods. This pattern consists of no significant change in heart rate, or else a bradycardia, fall in cardiac output and in arterial pressure, and vasoconstriction in mesenteric and iliac beds. Tachycardia, increased blood pressure, or a cholinergic preparatory vasodilatation of muscle beds were never observed. The confrontation period usually endured only a few seconds before fighting however, giving little time for preparatory changes to take their course. Longer trials of confrontation without any fighting were therefore run for each cat. Although detailed data on these experiments will be published elsewhere (4), it can be reported that the cardiovascular changes during prolonged confrontation not followed by attack were similar in direction to those occurring during the short preparatory period preceding fighting.

Comparing the cardiovascular changes we have observed during natural fighting behavior with those described by earlier investigators in much more artificial experimental conditions (see above), it is apparent that we have confirmed some of the previous data: tachycardia (19, 25), increased cardiac output (19), mesenteric vasoconstriction had been described (14, 15), and vasodilatation in contracting muscles could be easily expected. Two important disagreements with the previous literature have arisen, however.

First, the strong muscle vasodilatation seen during supportive fighting appears to be the result of local metabolic factors related to muscle activity, rather than to a central mechanism such as the cholinergic vasodilator system as might have been expected from experiments of brain stimulation (1, 18). In fact, if there is a primary nervous effect in addition to the metabolic factors it would appear to be a vasoconstriction instead, such as we have observed in the iliac bed during nonsupportive fighting. Results from the experiments eliminating flow to the skin of the paw support the view that this is a vasoconstriction of the muscle components of the iliac flow, not just of skin. Of course we do not dispute the existence of an atropine-sensitive, or cholinergic, central nervous mechanism of muscle vasodilatation in the cat, which may be obtained by electrical stimulation of the brain (30, 31). We simply question its relevance to natural behavior, as has already been done by other authors (11). However, if it appears certain that an active sympathetic vasodilatation is not a dominant factor in the cardiovascular changes during fighting, we cannot rule out a minor effect of sympathetic vasodilatation masked by other more important metabolic vasodilator and sympathetic vasoconstrictor effects. It is well known that vasoconstrictor influences usually suppress the cholinergic vasodilating effect (20), whereas metabolic vasodilatation might easily overwhelm, or mask, a similar sympathetic influence. Forthcoming data using cholinergic or adrenergic blockade will be crucial to reveal a possible cholinergic vasodilatating component.

Data from human subjects during emotional behavior usually indicates a vasodilatation (7, 9, 17, 22, 32) but one cannot determine from the literature whether or not it is due to muscle activity. As a matter of fact, according to an earlier report by Blair and associates (9) muscle vasodilatation was largely reduced by atropine, but these data could not be confirmed by later work of Blair et al. (10) and of Barcroft et al. (7), both groups reporting a very limited, and inconstant, decrease of the vasodilating effect after atropine. Likewise, cervical sympathectomy was found largely, though not invariably, effective in reducing forearm vasodilatation by Blair et al. in their first paper (9); but this procedure was ineffective according to Barcroft et al. (7), who found however a moderately reduced vasodilatation after acute stellate blockade. On the other hand, there is some evidence against a simple explanation in terms of metabolic effect of muscle activity since it is reported after partial paralysis of the muscle (10).

As to the second point of disagreement, one might have expected from previous literature (see 18) that the most important blood pressure change would be a long-lasting increase in pressure; instead, however, we have seen relatively slight changes in blood pressure and a quick return to resting levels. In fact, the most consistent change in

supportive fighting was a slight fall in blood pressure during the first few seconds after the striking movement, corresponding apparently to the beginning of muscle vasodilatation and the consequent increase in total peripheral conductance. This discrepancy cannot only result from the much shorter duration of fighting in our experiments: even in longer lasting fighting episodes (5) the dominant effect during supportive fighting was a fall in blood pressure, and in no cases were there very large increases in pressure. The complete, or almost complete, abolition of fighting movements in experiments on anesthetized, and sometimes also curarized animals, might be an important factor in accounting for the rise in blood pressure reported by other authors.

Although we can now present a relatively detailed picture of the cardiovascular changes that occur during fighting, we can only advance hypotheses on the underlying mechanisms, and design the experiments to test them. Are most of the cardiovascular changes we have observed during fighting the secondary reflex consequence of a primary event, the metabolically mediated vasodilatation of active muscle beds? In principle, tachycardia, augmented cardiac output, and iliac and mesenteric vasoconstriction might all be mediated through sinoaortic reflexes trying to maintain arterial pressure in face of vasodilatation in active muscles. Some of these changes however, might also be partly induced by direct influences from the brain. In fact, the appearance of visceral and iliac vasoconstriction during confrontation, when there is not yet any movement, suggests that at least these events may be centrally mediated rather than only reflexly induced. On the other hand, tachycardia which is absent in confrontation might largely result from reflex mechanisms, and augmented cardiac output both from reflexes and increased venous return. All these possibilities, however, cannot be answered until experiments using animals with sinoaortic deafferentation and pharmacological or surgical blockade of the sympathetic system are performed.

The importance of muscle activity in determining at least part of the cardiovascular events during fighting poses another question. Are the changes we have described during fighting peculiar to emotional behavior or are they more generally related to activity and exercise? According to a recent review of cardiovascular changes during exercise in man (8), the same qualitative changes occur as we have seen in fighting. Therefore, it should be stressed that the question concerning the cardiovascular events directly related to emotion will not be correctly answered until the emotional behavior under study is selected with the greatest care so as to avoid the participation of exercise.

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