near to the far wall by a micromanipulator in order to measure a local velocity at each sampling point.

The blood flow velocity measured near the central axis in the coronary artery showed a diastolic dominant pattern which is a characteristic of the coronary arterial flow. A typical example is shown in figure 4A. Usually 2 peaks were observed on the flow velocity pattern during the early and the late diastolic phase. This flow velocity pattern showed a good agreement with the results of the theoretical calculation for the dog coronary artery by Atabek et al.[8]. Nerem et al.[9] measured the coronary flow velocity of a horse by a hot-film anemometer. They also observed the presence of these oscillatory velocity components in the early and the late diastolic phase. However, the systolic component of the horse coronary artery was much larger than that of the dog. As the fiber tip traversed towards the vessel wall, the flow velocity decreased especially during late diastole (fig. 4B) and also during early diastole at sampling points very close to the vessel wall (fig. 4C).

In conclusion, we have found that the LDV using an optical fiber permits the measurement of the pulsatile blood flow velocity in a small sample volume. This LDV has the following advantages: (a) The sample volume of our system is roughly 100 μm which is much smaller than that of conventional methods, e.g., the ultrasound-Doppler method. The temporal resolution is also high (approximately 5 msec). (b) The optical fiber is thin and flexible. Thus, it has excellent accessibility to objects and the perturbation of the flow by fiber insertion will be small comparing with other catheter-type blood flow velocimeters. (c) Our method is free from electrical induction noises.

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Feather position of the pigeon after intrahypothalamic injections of noradrenaline, 5-hydroxytryptamine and carbachol[1]

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Summary. A slowly increasing ptiloerection was seen after the intrahypothalamic injections of NA; 5-HT was followed by preening and subsequent rapid feather fluffing, and carbachol produced an immediate sleeking of feathers. The naturally occurring complementary relationship between shivering thermogenesis and ptilomotion diminished or disappeared after the drugs, but feather position seemed to remain well related to the arousal state of the pigeon.

Hypothermia follows the injections of both noradrenergic, cholinergic, and serotonergic agonists into the anterior part of the hypothalamus of the pigeon in a cold environment[2-5]. The main cause for hypothermia after administration of noradrenaline (NA), 5-hydroxytryptamine (5-HT), and carbachol (CCh) seems to be the abolishment of shivering thermogenesis, and enhanced heat loss due to increased vasodilatation in the legs. The position of the feathers and thus the insulative properties of the plumage could be adjusted by smooth pennamotor muscles (musculi pennati)[6], and according to the hypothesis of McFarland and Budgell[7] the changes in feather position are primarily under hypothalamic control. It was therefore of interest to study whether the intrahypothalamic injections of NA, 5-HT and CCh have any effects on the regulation of feather position in the pigeon.

Materials and methods. 8 adult pigeons *(Columba livia)* of either sex, weighing 260–360 g, were used. A guide cannula was implanted unilaterally into the anterior hypothalamus as described earlier[4,8]. The coordinates were 7.8–8.0 mm anterior to the intra-aural line, 1.2 mm lateral to the midline and 9.0–10.0 mm below the surface of the calvarium. Each bird received first an injection of NA in a volume of 1 μl, after that, CCh, and finally 5-HT, with at least 1 week between successive injections. NA (L-arterenol bitartrate, Sigma, 15 μg/μl) was dissolved in 0.85% NaCl, CCh (carbamylcholine chloride, Sigma, 1.5 μg/μl) and 5-HT (as creatinine sulfate, Sigma, 15 μg/μl) in distilled water. In controls the same volume of vehicle was injected. All experiments were carried out in the afternoon between 13.00 and 17.00 h. After an equilibration time of at least 30 min in a cage located in a cold room at +5 °C, the drugs were injected via polyethylene tubing, which was connected to a 10-μl syringe located outside the cold room.

During the experiments the pigeons were observed through a TV-monitor (Philips LDH 25 camera and Finnvideo VM 12 FI monitor). The magnitude of ptiloerection was estimated once per min by applying the method of McFarland and Baher[9] originally devised for the barbary dove. The contour feathers of 6 body regions were scored according to their postures in 3 classes: 0 = sleeked, 1 = normal, and 2 = raised, and the scores of different regions were summed together to give the feather index (FI) with a minimum of 0 (fully sleeked) and a maximum of 12 (fully raised). The arousal state of the pigeons was estimated by visual observations. Axillary and foot temperatures and shivering from the pectoral muscle were measured as described earlier[10].

Results and discussion. All 8 pigeons showed the hypothermic response to NA but only 7 of them to CCh and 5 to 5-

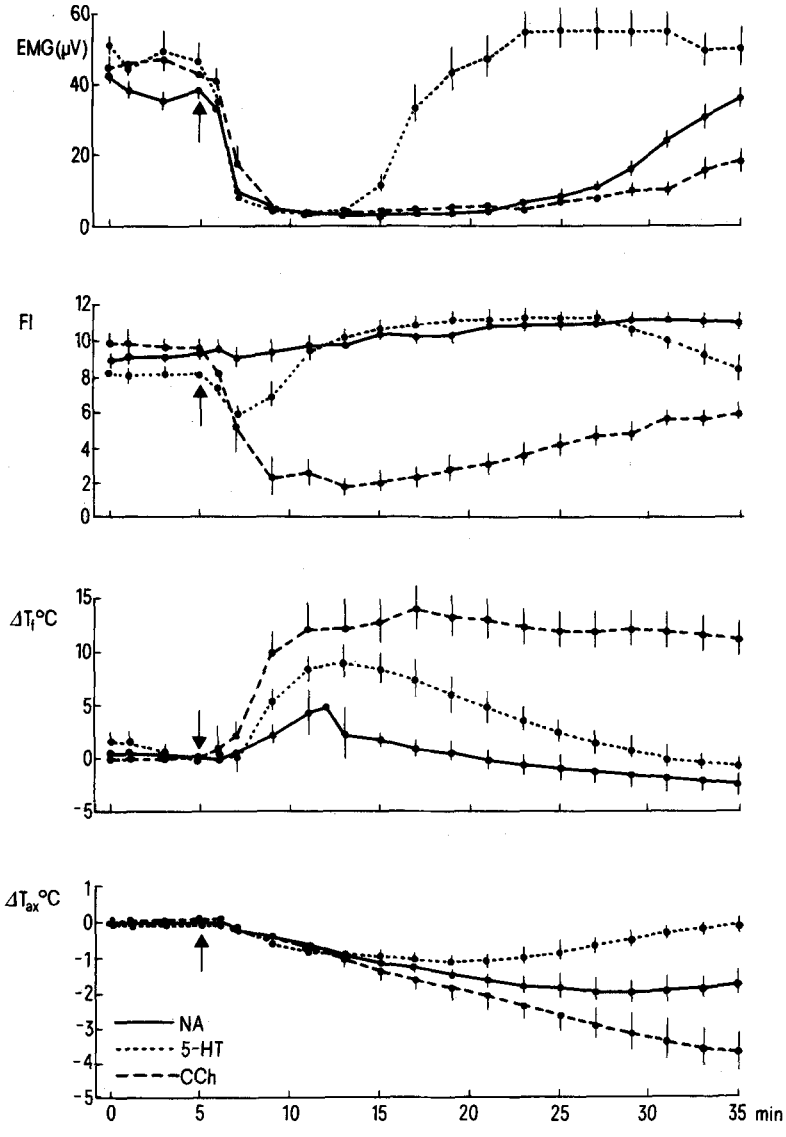

Figure 1. The effects of intrahypothalamic injections of NA (15 μg), 5-HT (15 μg), and CCh (1.5 μg) on shivering (integrated pectoral muscle EMG), feather index (FI), and foot (ΔT_f) and axillary (ΔT_{ax}) temperatures of the pigeons at an ambient temperature of +5 °C. Results are expressed as a mean value for 8 pigeons with NA injections, 7 pigeons with CCh injections and 5 pigeons with 5-HT injections. Vertical bars indicate ± SEM.

HT (fig. 1). Control injections with vehicle were without any marked effects. The injection sites in the anterior hypothalamus are shown in figure 2.

Before each injection the pigeons were observed to stand motionless with their feathers partly fluffed (FI was on the average 9.1). A slow increase in FI ($p < 0.05$ within 14 min of injection) and drowsiness was seen after NA injection, but 5-HT caused an intensive preening, lasting 5 ± 2.2 min ($\bar{x} \pm SE$) immediately after application. After preening and accompanying sleeking of feathers, a rapid and significant ($p < 0.01$) feather fluffing occurred, and the birds fell asleep with their beaks tucked into their breast feathers. CCh injection was immediately followed by a behavioral arousal: feathers were sleeked ($p < 0.05$ already within 1 min of injection) and the birds moved and raised their wings actively.

The present results suggest that the effects of NA, 5-HT and CCh were quite different on ptilomotion, although they uniformly produce hypothermia[2-4]. NA and 5-HT increased drowsiness and provoked sleep, which was accompanied by typical sleep postures. Increased drowsiness was associated with a rise in FI, which presumably reflects an improvement in the insulation of the plumage[11,12]. The other responses, i.e. abolishment of shivering and stimulated

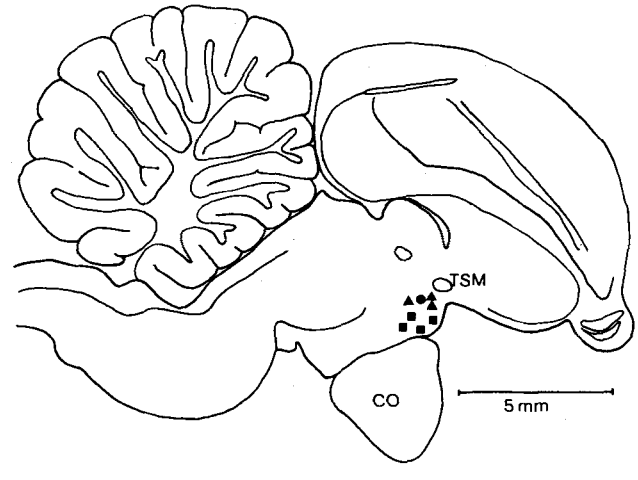

Figure 2. Sagittal section of pigeon brain 1.0–2.0 mm lateral to the midline[8]. The symbols indicate the injection sites: ■, response to NA, 5-HT and CCh; ●, response to NA and 5-HT; ▲, response to NA and CCh. CO, chiasma opticum; TSM, tractus septomesencephalicus.

vasodilatation, were, on the contrary, hypothermic. Only after CCh could all thermoregulatory responses studied be uniformly attributed to hypothermia.

The naturally-occurring complementary relationship between insulative and thermogenic mechanisms[10] was no longer shown after application of drugs; the correlation coefficient between 1-min means of FI and integrated EMG-activity was −0.157 (NS) after NA, and after 5-HT and CCh even highly positive (0.404, $p < 0.05$, and 0.959, $p < 0.001$, respectively).

In conclusion, our results suggest that intrahypothalamic injections of NA, 5-HT and CCh have either direct or indirect effects on feather position in the pigeon. Ptilomotor changes seem to be well related to the drug-induced changes in the arousal state, but the relation to shivering thermogenesis seems to be variable.

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Conditioning of depressor responses evoked by single volleys in the aortic nerve

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Summary. Depressor responses evoked by single volleys in the aortic nerve are more facilitated by conditioning volleys exciting C fibers in the same aortic nerve than in the contralateral one. Conditioning volleys in A fibers do not facilitate the testing depressor responses.

Depressor responses evoked by activation of the aortic nerve were studied mainly under conditions of repetitive stimulation. This kind of stimulation produces temporal facilitation in the vasomotor centers and results in prolonged changes of their excitability. The duration of these changes was determined in interaction experiments with 2 repetitive stimulations applied to the same aortic nerve. It was found that at durations of stimulations amounting to 20 sec the effect of a conditioning stimulation on the size of the testing response is visible even when the interval between them exceeds 180 sec[1]. In contrast to these results the effect of single volleys in the aortic nerve is much less conspicuous. Single volleys in A fibers do not produce any changes in the arterial blood pressure. Douglas et al.[2] observed that when the strength of a single pulse is sufficient to excite non-myelinated afferents (C fibers) small blood pressure falls are encountered in approximately half of animals. These findings indicate liminal activation of the vasomotor mechanisms. We have tried to assess the extent of this activation by employing the technique of conditioning. For this purpose the testing depressor responses, evoked by single volleys in the aortic nerve, were preceded by volleys in the same or the contralateral aortic nerve.

Material and methods. 33 rabbits were anaesthetized with urethane (1.3 g/kg) and vagotomized. The arterial blood pressure was measured in the femoral artery. The right and left aortic nerves were identified electrophysiologically and each of them mounted on the pair of platinum stimulating electrodes. The nerves were covered with warm paraffin oil. Single rectangular pulses of 1-msec duration were used for conditioning and testing stimulation. The strength of pulses evoking testing depressor responses amounted to 10 V and was supramaximal for activation of C fibers[3]. The intensity of conditioning pulses was either the same or lower (usually 3 V) to activate only A fibers. The intervals between the pairs of pulses were not shorter than 5 min.

Results and discussion. Single volleys activating C fibers in the aortic nerve usually evoke very small and variable depressor responses. To make an analysis of their changes during conditioning easier we have taken into consideration the results from animals in which the size of the testing blood pressure falls was equal to or exceeded 10 mm Hg. It was found that depressor responses of that size appeared in 12 out of 33 rabbits, i.e. in 36%. The records of figure 1A and the upper curve of figure 2 demonstrate the changes in the size of the testing depressor responses when both conditioning and testing volleys are applied to the same aortic nerve. At a testing interval of 8 msec the size of the depressor response amounts to 125% of the control. At longer intervals between volleys the testing response rises abruptly to reach its maximum of 231% at the interval of 128 msec. With further lengthening of the intervals between volleys the decline of the testing response is observed and at 1024 msec its size is similar to that observed at 8 msec. The records of figure 1B and lower curve of figure 2 show

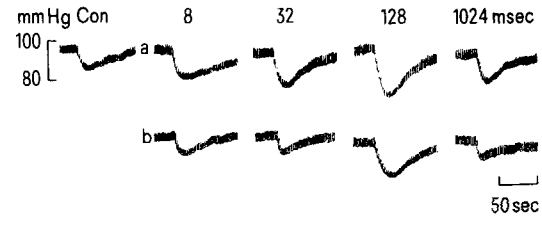

Figure 1. Conditioning of depressor responses by volleys in the same (A) and contralateral (B) aortic nerve. The depressor responses were evoked by single volleys exciting C fibers of the aortic nerve. The 1st record (Con) shows the test depressor response not preceded by a conditioning volley. The following records illustrate conditioning of the depressor response. Numbers above the records indicate intervals between conditioning and testing volleys.