function of the kidney is eliminated by ureter ligation. To a certain degree, the severity of the shock depends on the amount of the injected microspheres. After the injection of 10 mg into each kidney, the serum urea rose transiently, but no symptoms of circulatory failure occurred, and all animals survived for a period of more than 3 months. In contrast to this, the injection of 25 mg into each kidney always resulted in the rapid development of a severe shock-like state, and all animals succumbed within 12 h.

The shock-like state is prevented, if a contralateral, non-embolized kidney is present, independent of its excretory function. If the intact renal tissue is removed after several hours, a shock-like state still develops as a consequence of the preceding injection of microspheres into the other kidney.

It is suggested that the shock-like state after massive embolization is elicited by the release of a substance from the affected kidney. At first, it was supposed that renin might have been excessively released, and the high plasma concentration of angiotensin II seems to support such an assumption. However, the stimulation of renin secretion and the enhanced formation of angiotensin II should result in an increase in blood pressure. Furthermore, in studies with the isolated perfused kidney, only a transient release of renin for about 10 min was observed[4], but the amount of microspheres injected in those experiments was only a fraction ($^1/_{50}$) of that given to the rats in which a shock-like state developed. The high plasma concentration of angiotensin II is probably the consequence of a secondary activation of the renin-angiotensin system. The reduction in intravascular volume, caused by the increased capillary permeability, is the major stimulus for the renin-angiotensin system. In addition, renin might have been released also from the ischaemic renal tissue. A release of renal prostaglandins has also been considered, but when the rats were pretreated with indomethacin (2–3 mg/kg), the shock-like state could not be prevented. However, these experiments do not exclude a possible contribution of prostaglandins to the shock syndrome. Another possibility would be the release of kininogenases from the kidney and the formation of kinins, such as kallidin and bradykinin. If bradykinin was infused into rats, the blood pressure fell in relation to the dose, which ranged from 2.5 γ/kg/min to 125 γ/kg/min. The response to vasopressor agents, such as noradrenaline and angiotensin II, was reduced. A bolus injection of 50 γ bradykinin per rat (corresponding to 250 γ/kg) was followed by a decrease in blood pressure and heart rate, and the rats died within 10–40 min. Although these observations seem to support a causal role of bradykinin in the development of the shock syndrome after renal embolization, they do not explain the protective role of intact renal tissue. Furthermore, the amount of bradykinin that may be formed in the rats is limited by the amount of substrate present. Hence, convincing evidence is still lacking that the kininogenase-kinin system is the only factor involved in the pathogenesis of the observed syndrome. On the other hand, bradykinin has been claimed to be responsible for the shock that occurs after injection of trypsin and other proteolytic enzymes[6]. Studies with the protease inhibitor aprotinin could not be undertaken because of its high toxicity in rats.

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Hornet ventilation noise: Rhythm and energy content

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Summary. A new technique is used to measure the hornet's wing movement. It enables one to measure precisely the frequency of this movement, even in the presence of spurious 'noise'. This autocorrelation technique revealed that the hornet's wing stroke is divided into 3 smaller strokes when they are tired. The energy content in each stage can be measured using the autocorrelator.

Thermoregulation is a common phenomenon among Vespinae[1], who use both fanning with the wings and water transport to cool overheated nests[2–5]. In the case of the Oriental hornet, Vespa orientalis, several workers arrange themselves around the nest entrance, facing out in nests above ground or up in subterranean nests. At temperatures higher than optimal, these workers commence a constant and rapid fanning movement of their wings. The fanning workers are most frequently dispersed on the outside of the nest, at some distance from the entrance[6], but may also arrange themselves behind and around it, in the galleries between the entrance and the combs, and on the combs proper. According to Steiner[3], the adult wasps begin fanning their wings when the temperature of the surface of the brood comb reaches about 35 °C. Because the Oriental hornet builds its nests underground, the workers may commence fanning even at temperatures around 20 °C, provided the relative humidity is higher than 80%. This can be induced experimentally by moistening the soil around the nest, and, in the laboratory, by keeping the floor of the artificial breeding box (ABB) wet. In this case, one to several workers engage in thermoregulation, and their fanning activity can be observed and recorded over long periods without interruption.

In order to obtain accurate data on the fanning activity and the noise produced by it, we employed a technique well known in astronomy and related fields, but never before used in analyzing hornet ventilation noise. A series

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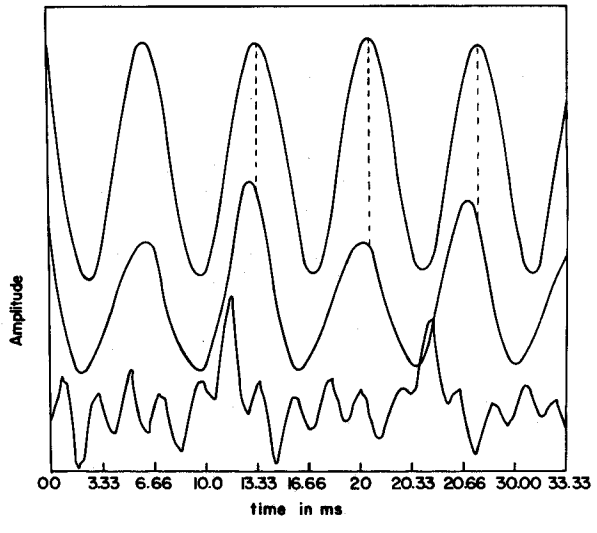
Energy content

	Top curve	Middle curve	Bottom curve
Down-stroke	100%	79%	21%
Upward-stroke	100%	55%	12.5%

of sensitive microphones (Brüel & Kjaer) are placed in advance in the ABB in the estimated vicinity of ventilating hornets. The electrical signal from the microphone closest to a ventilating hornet is then fed into a tape-recorder (Pioneer, Dolby system, high fidelity, frequency range 5–20,000 Hz) and recorded on a cassette tape. The ventilation noise is recorded from start to finish for each fanning hornet (and its subsequent replacement).

The individual recordings are next fed into an auto-correlator (Hewlett-Packard Type No. 3721A). In the correlator, the analogue signal is first digitized, i.e. every $^1/_3$ millisecond, the instrument detects the height of the signal at that moment and produces a number which is proportional to the height. There are thus 180,000 such number per min of recorded time. The auto-correlator is actually a computer that takes these numbers and processes then as follows: if the numbers are labeled a_1, a_2, $a_3, \ldots a_n$ ($n = 180{,}000$ for 1 min), the computer first calculates the expression $\sum_{i=1}^{180{,}000} a_i^2$ and memorizes it on channel 0. The product $\sum_{i=1}^{180{,}000} a_i \times a_{i+1}$ is then fed into channel 1 and the product $\sum_{i=1}^{180{,}000} a_1 \times a_{i+2}$ into channel 2, and so on up to channel 99. With our particular auto-correlator, these products are calculated continuously as more data come in and the memory channels are updated for as long as the tape output is fed into the auto-correlator. Ultimately the 100 memory channels are displayed and recorded.



The amplitude of the auto-correlator is plotted versus time. The top curve shows a repetition rate of 150 times per sec. The middle curve was taken 6 min later and it shows a faster repetition rate and smaller amplitude of the up-stroke. In the bottom curve taken 6 min later, the 2 strokes were split into three shorter swings. It took 3 min of integration to obtain each curve.

The auto-correlator technique employed here enables us to trace the exact 'shape' of signals even in the presence of spurios 'noise'. If there is a certain feature in the signal that repeats itself every so often, the display of the 100 channels shows only the repetitious feature and ignores any random feature. Thus the auto-correlation technique may be considered as giving a representative section of a signal, 'cleaned' from any random 'noise'. If nothing in the incoming signal is repetitious and the peaks and the 'valleys' occur at random times, the display will show a straight line. The main advantage of the auto-correlator, therefore, is that it can extract a repetitious signal out of a random noise which may be much larger than the signal itself. The longer the time of integration, the better the repetitious signal will stand out of the noise. The second advantage is that the time-measuring capabilities of the instrument are very precise, enabling us to determine the rate of repetition with maximum accuracy.

The top curve in the figure illustrates the result of such an analysis (at 28 °C and 90% relative humidity). A representation of 33.33 msec of real time is displayed. It shows a clear repetitious signal every 6.66 msec or 150 times per sec. In terms of wing movement this implies that a downstroke-upstroke beat of equal intensity occurs every 13.33 msec or 75 times per sec.

An interesting change is noted about 6 min after the workers start ventilating. The change is evident from the middle curve of the figure, which is different from the upper curve in 2 respects: the strokes are a little bit faster (153.8 times per sec or every 6.5 msec) and every second stroke (probably the down-stroke) is lower in amplitude (by 32%) than the first. 5 min later, the pattern is altogether different (figure, bottom curve); the down-stroke is still there, but is split into 3 peaks, and the up-stroke is also split into 3. The time between two upward strokes is now shorter (12 msec, or 83.33 times per sec). This new pattern is interesting and is probably detectable only with a correlator. It shows that at a certain moment, probably when the hornet is tired, the down-strokes are not made in one continuous swing, but are rather divided into 3 shorter swings before the upward stroke commences. The reason we suspect hornet fatigue to be responsible is that the total energy expenditure in the bottom curve is smaller than in the middle or top curve. The energy content is measured by the area under the peaks, and the area is the largest in the top curve, smaller in the middle curve and smallest in the bottom curve (even if one takes into account the higher repetition rate). The energy content values of the peaks in the figure are presented in the table.

Of the ventilating hornets, 5 followed this sequence of events in the time schedule specified above, 7 produced the sequence in a shorter time, i.e., up to 3 min for the first change, and another 3 min maximum for the second change (bottom curve of the figure), and 3 took longer (up to 14 min) to complete a sequence.

It would seem that the ventilation activity initially entails a series of two-stroke wing movements – a strong down-stroke (the power stroke) and a subsequent up-stroke. Gradually, however, the down-strokes weaken (probably as a result of fatigue), breaking up into several sub-strokers and possibly decaying altogether. It should be noted that in the natural, well populated nest, a considerable number of workers (up to 30–50 on very warm days of following rainfall) may be engaged in ventilation activity. This poses the question (now under investigation) as to how the ventilating workers synchronize their wing movements to avoid a phase difference which would interfere with coordinated ventilation.

In Vespa orientalis, as in other Hymenoptera, a myogenic rhythm of wing-beat is observed. In myogenic insects, the energy to maintain a wing-beat frequency can only be drawn from fibrilar muscles. A mechanical trace from the thorax of an active wasp[7] points to a frequency of 155 Hz, whereas an electrical trace from the same wasp yields a considerably lower frequency. In the latter instance, the impulses in motor nerves supplying the main flight muscles are not synchronous with the wing beats. Our method of recording, when coupled to a processing via correlator, permits an accurate analysis of the ventilation noise of an insect without interfering with its normal activity. The recorded differences in duration of ventilation activity among various hornets probably represent differences in the physical endurance of individual hornets.

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Non-uniformity of regional vasomotor activity indicating the existence of 2 different systems in the sympathetic cardiovascular outflow

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Summary. In rabbits, 2 populations of sympathetic postganglionic fibres innervating the skin, heart, muscle and kidney could be classified by their different spike heights and their different susceptibility to noradrenaline and angiotensin amide. The ability of the 2 populations to respond to physiological stimuli in a highly differentiated manner leads to the assumption that 2 different systems in the cardiovascular sympathetic outflow exist.

Experiments performed within the last few years demonstrate the ability of the sympathetic vasomotor system to respond to various physiological stimuli with a high degree of differentiation of regional sympathetic outflow (for review of literature, see Simon and Riedel[1]). By compilation of these changes in regional sympathetic activity or changes in blood flow, a pattern results as the creation of the vasomotor control center in response to the entire information – neural and humoral – during a given stressful situation. In the analysis of the components of the diverse patterns of autonomic outflow by direct recording of regional vasomotor activity in rabbits, non-uniformity up to the degree of opposite changes of activity was found in different filaments innervating the same vascular regions or organs, e.g. the heart. This indicates the ability of the autonomous nervous system to functional differentiation of its outflow to single organs, which may be based on the existence of different fibre populations. This, in fact, could be shown for the innervation of the heart where changes of activity in some filaments always correlated with changes of heart rate and changes in other filaments appeared to be associated with changes in myocardial function[2]. Functionally different populations of vasomotor fibres have recently been identified in the vasomotor supply to the muscle by Horeyseck et al.[3] and to the skin by Gregor et al.[4] in cats. As in the heart we have found differently reacting fibre populations in the vasomotor supply to the muscle, skin and kidney of rabbits, whose function we do not yet know in detail. They showed some common intrinsic properties which may allow a general classification.

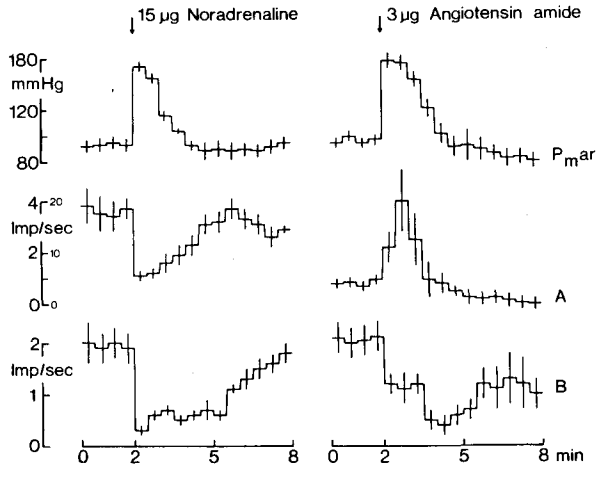

Fig. 1. Response of renal sympathetic efferents (fibre type A and B) and of arterial mean blood pressure ($P_m$ar) to i.v. injections of noradrenaline (15 µg) and angiotensin amide (3 µg). Mean values with standard deviations obtained from 9 animals. The right scale of the ordinate for fibre type A refers to the effect of angiotensin amide.

Materials and methods. 32 rabbits, weighing 3.2–4.8 kg, were anesthetized with nembutal sodium (30 mg/kg as initial dose). Subsequent anesthesia was maintained by artificial ventilation with 30% O_2 and 70% N_2O. In addition, a continuous infusion of either pentobarbital (5 mg/animal/h) or Althesin (Glaxo, 0.5 ml/animal/h) was given via the femoral vein. Relaxation was maintained with either succinyl choline (10 mg/animal/h) or gallamine (5 mg/animal/h). The femoral artery was cannulated for blood pressure recording. Sympathetic activity was recorded from fine nerve strands after removing the perineurium. Filaments containing one or few functioning fibres were put on a platinum electrode under paraffin oil. The fibre activity was amplified (band width 0.06–1.5 kHz) and displayed on an oscilloscope. Fibres discharging with different spike amplitudes could be separated by means of a window discriminator and were stored together with the original spikes and the blood pressure on magnetic tape. Numerical evaluation of the impulse rates and of arterial pressure was performed

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