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CHANGES IN RENAL SYMPATHETIC NERVE ACTIVITY, HEART RATE AND ARTERIAL BLOOD PRESSURE ASSOCIATED WITH EATING IN CATS

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SUMMARY

1. Renal sympathetic nerve activity, heart rate and arterial blood pressure were simultaneously measured in thirteen awake cats before, during and after eating which was evoked by presenting food for a period of 10–15 s.

2. With food presentation, eating behaviour occurred in 93% (191) of 205 trials, and renal sympathetic nerve activity significantly increased in 65% of the 191 trials. On the other hand, in many of food presentation trials when no eating occurred, or with presentation of an empty food box, renal sympathetic nerve activity did not change significantly.

3. Eating started 1–8 s after the food presentation. The increase in renal sympathetic nerve activity was closely related to the beginning of eating but not to the onset of food presentation. Renal sympathetic nerve activity and heart rate increased with a slight time lag of 0·5–1·5 s from the onset of eating, whereas an increase in arterial blood pressure followed the onset of eating by 5·5 s. After the beginning of eating, renal sympathetic nerve activity, heart rate and arterial blood pressure increased at a maximum of $61 \pm 18\%$ (mean $\pm$ S.E. of mean), $26 \pm 4{\cdot}0$ beats/min, and $17 \pm 4{\cdot}9$ mmHg from the control values at 1·0, 5·5 and 11·5 s, respectively.

4. Cardiac-related grouped discharges of renal sympathetic nerve activity, which were observed at rest, increased during eating.

5. When arterial blood pressure was elevated by noradrenaline (2–5 μg/kg I.V.), renal sympathetic nerve activity during resting was almost completely inhibited and the increase in renal sympathetic nerve activity during eating was not induced.

6. We conclude that renal sympathetic nerve activity increases in association with eating behaviour but not as firmly with the food presentation, and that the increase in renal sympathetic nerve activity is initiated by descending input from the higher central nervous system rather than either by the viscero-autonomic reflex due to food intake or by the baroreflex due to a decrease in arterial blood pressure.

INTRODUCTION

The sympathetic nervous system has been thought to play an important role in cardiovascular regulation associated with eating behaviour in conscious animals. Early studies showed that in human subjects and in the baboon, dog, sheep, pig, cat

and unweaned calf, kid and lamb, heart rate (H.R.) and arterial blood pressure (A.P.) transiently increased at the beginning of eating (Abramson & Fierst, 1941; Fronek & Stahlgren, 1968; Blair-West & Brook, 1969; Vatner, Franklin & Van Citters, 1970*a*, *b*; Ehrlich, Tosheff, Caldini, Abbey & Brady, 1972; Vatner, Patrick, Higgins & Franklin, 1974; Bloom, Edwards, Hardy, Malinowska & Silver, 1975; Houpt, Baldwin, Houpt & Hills, 1983; Matsukawa & Ninomiya, 1985). In the dog and baboon, cardiac output and renal and mesenteric vascular resistances increased at the beginning of eating whereas coronary vascular resistance decreased (Fronek & Stahlgren, 1968; Vatner *et al.* 1970*a*, *b*, 1974; Ehrlich *et al.* 1972;). These abrupt and transient cardiovascular changes suggested that the autonomic nervous system mediated the cardiovascular adjustment during eating. Effects of autonomic blocking agents on the transient cardiovascular changes have been previously examined. The increases in H.R., A.P. and renal and mesenteric vascular resistances at the beginning of eating were reduced by α- or β-adrenergic blockers (Vatner *et al.* 1970*a*, *b*; Matsukawa & Ninomiya, 1985). Furthermore, the increases in H.R. and A.P. were almost completely abolished by hexamethonium bromide (Matsukawa & Ninomiya, 1985). On the other hand, atropine did not prevent increases in A.P. and renal and mesenteric vascular resistances and a decrease in coronary vascular resistance at the beginning of eating (Vatner *et al.* 1970*a*, 1974; Matsukawa & Ninomiya, 1985). Although it was suggested that there was a pronounced activation of sympathetic efferent activity at the beginning of eating, direct information concerning sympathetic efferent activity during eating in conscious animals was lacking.

The purpose of this study was (1) to obtain information about renal sympathetic nerve activity (r.n.a.), H.R. and A.P. in conscious cats before, during and after eating, and (2) to identify neural mechanisms responsible for cardiovascular adjustment associated with eating.

METHODS

Preparation of animals

The experiments were carried out on thirteen cats (body weight: 2·0–4·2 kg). They were anaesthetized with sodium pentobarbitone (35–40 mg/kg I.P.) for surgical implantation of recording electrodes and catheters. Additional doses of sodium pentobarbitone (5 mg/kg I.V.) were injected as necessary. Each cat was intubated with an endotracheal tube and artificially ventilated with room air. After surgery, the cats were kept in their cages; they could take dry cat food and water *ad libitum*. Antibiotics were given to the cats for 3–5 post-operative days.

Implantation of recording electrodes and catheters

Under aseptic conditions, either the right or left kidney was exposed retroperitoneally. A branch of the renal nerve (approximately 0·5–1·0 cm in length) was separated from the renal plexus and surrounding connective tissue located near the renal artery and vein (Ninomiya, Yonezawa & Wilson, 1976; Ninomiya & Yonezawa, 1979), using a dissecting microscope (Zeiss). The r.n.a. was recorded by a pair of silver-wire electrodes (0·1 mm in diameter) insulated by a silicone tube (Dow-corning, No. 602-105, 0·64 mm in outer diameter). The ends of the wires were carefully wound around the renal nerve branch. The wires and the nerve branch were enveloped in a thin Teflon or silicone sheet (0·05 mm in thickness) and embedded in a silicone gel. In two out of thirteen cats, the renal nerve branch was cut on the distal side of the recording electrodes; in the other eleven cats, the renal nerve branch was left intact.

A pair of silver plates (4 × 4 mm) were implanted under the skin of the left chest for electrocardiogram (e.c.g.) recording. A silver plate (4 × 4 mm) was placed as a ground electrode under the skin of the back. Heparin-filled catheters were inserted into the left carotid artery for recording

arterial blood pressure (A.P.) and into the left external jugular vein for administering drugs. The lead wires of the electrodes and catheters were brought to the exterior in the intrascapular region. During eating experiments, the wires were connected to a recording instrument by light lead cables.

Experimental protocol

Training for several days before surgery accustomed the cats to the restraint of body trunk movements as we previously described (Matsukawa & Ninomiya, 1985). Each was restrained in a cotton bag with a suitable opening for the head; the bag was mounted in a rigid frame with a hammock as illustrated in Fig. 1. Under those conditions, the cat could easily move its head downward to take food.

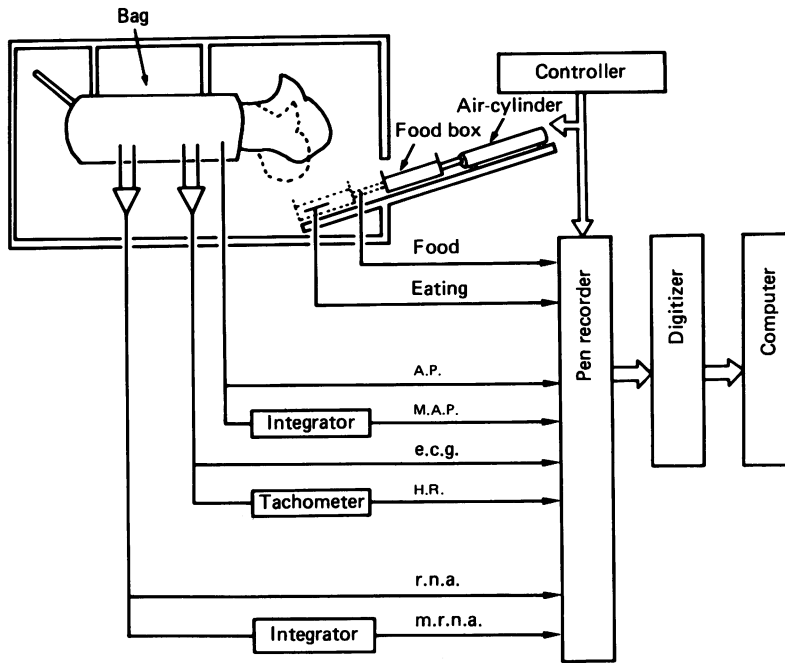

Fig. 1. A diagram of the experimental set-up. A food box was presented to the cat manually or automatically by an air-cylinder device. The cat's torso was restrained in a bag and mounted in a frame with a hammock. Food, indicates voltage signal to monitor the moment of food presentation; eating, voltage signal detected by an electrode within the food box to monitor eating behaviour; A.P., arterial blood pressure; M.A.P., mean arterial blood pressure; e.c.g., electrocardiogram; H.R., heart rate; r.n.a., renal sympathetic nerve activity; m.r.n.a., mean renal sympathetic nerve activity.

Eating experiments were conducted no earlier than 48 h after the end of anaesthesia. On the experimental day, the cat was brought into the experimental room and mounted in the frame. Then, a food box with canned tuna was abruptly presented to the cat either by hand in ten of thirteen cats or automatically by a specially designed air-cylinder device (Matsukawa & Ninomiya, 1985) in the other three cats. Changes in r.n.a., A.P. and H.R. during eating were the same whether eating was evoked by presenting food manually or automatically. In this study, 205 food presentation trials were conducted on thirteen cats 2–8 days after implantation surgery. Eating behaviour was evoked in 93% of the food presentation trials but not in 7%. In each food presentation trial, a food box was continuously present for 10–15 s. The intertrial interval was 2–5 min.

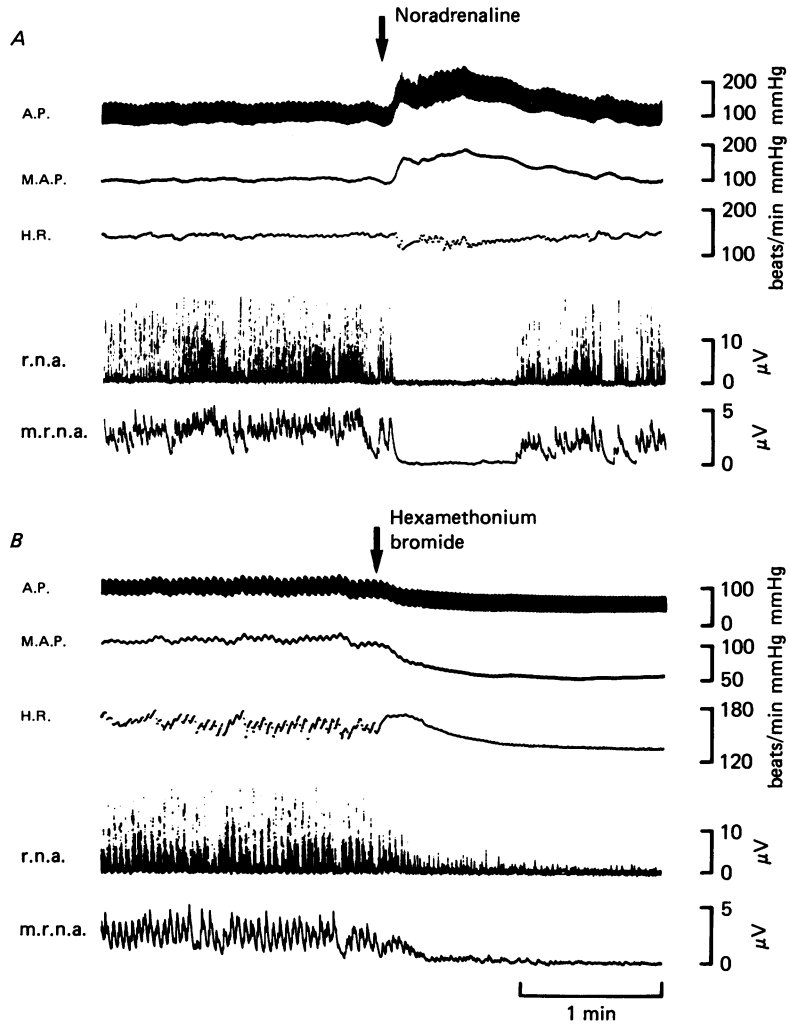

Fig. 2. Decrease in renal sympathetic nerve activity (r.n.a.) and mean renal sympathetic nerve activity (m.r.n.a.) induced in a resting awake cat by noradrenaline (2 μg/kg I.V.) (*A*) and hexamethonium bromide (3 mg/kg I.V.) (*B*). *A*, when arterial blood pressure (A.P.) was elevated by noradrenaline, r.n.a. and m.r.n.a. decreased approximately to the noise level. *B*, after injection of hexamethonium bromide, r.n.a. and m.r.n.a. decreased approximately to the noise level. M.A.P., mean arterial blood pressure; H.R., heart rate. The records were obtained on the second day after implantation surgery.

Recording of renal sympathetic nerve activity

The original nerve signal, denoted as original r.n.a., was amplified by a differential pre-amplifier (Nihon Kohden, S-0476) and was continuously monitored on a storage oscilloscope (Hitachi, VC-6015). The amplified signal was filtered between 50 and 3000 Hz and then rectified by a full-wave rectifier circuit. The output signal was integrated by an R–C integrator with a time constant of 50 ms. We refer to the integrated signal as r.n.a. Mean renal sympathetic nerve activity (m.r.n.a.) was obtained by integrating the rectified nerve signal with a time constant of 1 s. The r.n.a. examined in thirteen conscious cats showed grouped discharges synchronized with the cardiac cycle

as previously reported in conscious cats (Schad & Seller, 1975; Ninomiya *et al.* 1976; Ninomiya & Yonezawa, 1979). The grouped discharges of r.n.a. were modulated by respiratory movement (Ninomiya & Yonezawa, 1979). As shown in Fig. 2*A*, on administration of noradrenaline (2–5 μg/kg I.V.), the grouped discharges synchronous with the cardiac cycle and respiration disappeared and r.n.a. and m.r.n.a. were reduced approximately to a noise level. Also, after administration of the ganglionic blocker (hexamethonium bromide, 3 mg/kg I.V.), the grouped discharges disappeared and r.n.a. and m.r.n.a. were reduced approximately to noise level as shown in Fig. 2*B*. These findings suggest that the r.n.a. measured during resting conditions was baroreceptor dependent and originated from post-ganglionic efferent fibres as previously reported in anaesthetized or conscious cats (Ninomiya, Irisawa & Nisimaru, 1973; Ninomiya *et al.* 1976).

Data and statistical analyses

Eating behaviour was monitored by an electrode which was placed in the food box (Fig. 1) as we previously described (Matsukawa & Ninomiya, 1985). A voltage (10 kHz, 1 V) was delivered at the electrode through a series resistance of 5 MΩ. When the cat touched food, the electrode was electrically shunted to ground through the body of the animal. We defined the onset of the voltage change at the electrode as the onset of eating. We measured A.P. with a pressure transducer (Gould, P23ID) which was connected to the carotid artery catheter. Mean arterial blood pressure (M.A.P.) was electrically obtained by integrating the A.P. signal with an R–C integrator which had a time constant of 0·5 s. The H.R. was obtained from a tachometer (NEC San-ei, 2140) triggered by an R-wave of the e.c.g. signal. The r.n.a., m.r.n.a., H.R., A.P., M.A.P. and eating behaviour were simultaneously recorded on a heat-pen writing recorder (NEC San-ei, 8K23) and also displayed on an oscilloscope (Nihon Kohden, VC-10) as illustrated in Fig. 1.

The average value of r.n.a. during a period of 0·5 s was calculated by measuring the area of the r.n.a. tracing on the pen writing chart using a digitizer (Hewlett–Packard, 9874A) assisted by a minicomputer (DEC, PDP-11/60). In the same manner, the average values of H.R. and A.P. during 0·5 s were determined. The average r.n.a., H.R. and A.P. data, which were sequentially obtained in each food presentation trial, were treated as follows.

First, the average r.n.a. in each food presentation trial was used to evaluate whether or not r.n.a. changed significantly in response to food presentation. In each trial, we estimated a control variation of r.n.a. from the mean and standard deviation (S.D.) of the average r.n.a. during the 10–15 s control period preceding the food presentation. If a change in the average r.n.a. following the food presentation exceeded a threshold level which we defined arbitrarily as the mean $\pm 3 \times$ S.D., we recognized this as a significant response in r.n.a.

Secondly, we measured in thirteen cats the magnitude of responses in the average r.n.a., H.R. and A.P. during eating behaviour which was evoked by the food presentation. After the average r.n.a., H.R. and A.P. were collected from seven to twenty-three trials during eating in an individual cat, the relative changes from the control values were determined. Thereafter, we calculated grand mean values of the relative changes in thirteen cats. A statistical significance of the changes in r.n.a., H.R. and A.P. during eating was estimated by the one-way analysis of variance (Snedecor & Cochran, 1980) and by the modified least-significant difference method (Winer, 1971). The level of the significance was defined at $P < 0.05$. The data in the Figures and in the results are expressed as means $\pm$ S.E. of means with sampled numbers.

RESULTS

Response in renal sympathetic nerve activity to food presentation

Fig. 3 shows an example of changes in m.r.n.a. and H.R. during four successive food presentation trials in an awake cat. Eating was evoked in all of the food presentations, and m.r.n.a. and H.R. increased as indicated by filled stars in Fig. 3.

A significant increase in r.n.a. was observed in 60% (124) of 205 food presentation trials conducted on thirteen cats, but r.n.a. did not change significantly in 40%. In two out of the thirteen cats, when the renal nerve was sectioned distal to the recording electrode, r.n.a. increased in response to food presentation just as it did when the

renal nerve was intact. Eating behaviour was evoked in 93% (191) of 205 food presentation trials but not in 7%. We therefore examined the question of whether or not the increased response in r.n.a. to food presentation was related to eating behaviour.

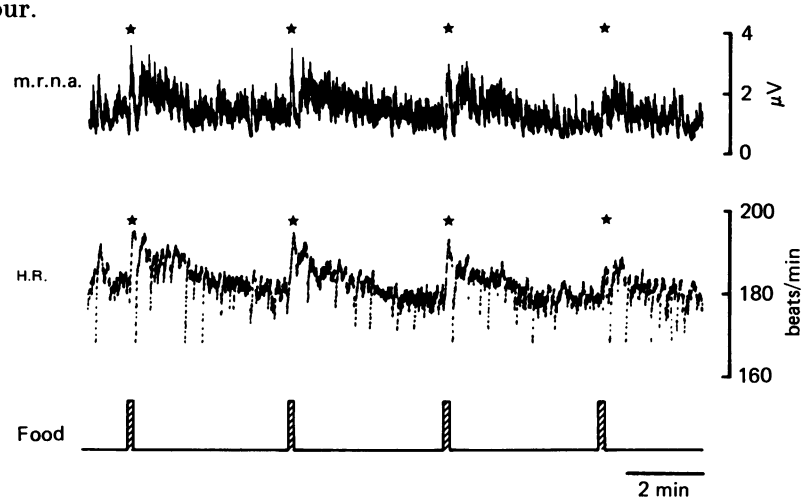

Fig. 3. Changes in mean renal sympathetic nerve activity (m.r.n.a.) and heart rate (H.R.) during four successive food presentation trials in an awake cat. In each trial, food was presented to the cat for 10 s at 4 min intervals as shown by hatched areas. Eating was evoked in all of the food presentations, and m.r.n.a. and H.R. increased significantly as shown by filled stars. The records were obtained on the fourth day after implantation surgery.

Response in renal sympathetic nerve activity associated with eating behaviour

Fig. 4 compares changes in r.n.a., m.r.n.a. and H.R. in a food presentation trial with eating behaviour (*A*) and in the succeeding trial when there was no eating behaviour (*B*) in an awake cat. Fig. 4*A* shows that r.n.a., m.r.n.a. and H.R. increased almost simultaneously with the onset of eating (vertical dashed line). The m.r.n.a. and H.R. significantly increased by 195% and by 14% (26 beats/min) from the control values respectively, as indicated by filled stars in Fig. 4*A*. In 65% of 191 food presentation trials with eating behaviour, significant increases in r.n.a. were observed. On the other hand, when an empty food box was presented and there was no eating behaviour (Fig. 4*B*), r.n.a. and m.r.n.a. did not increase significantly although H.R. seemed to increase slightly. In all of six trials involving presentation of an empty food box, no significant changes in r.n.a. were observed. Similarly, in 71% of fourteen food presentation trials when there was no eating behaviour, no significant changes in r.n.a. were induced.

Time relation between the onsets of the increase in renal sympathetic nerve activity, food presentation and eating behaviour

The interesting question is whether the increase in r.n.a. recorded during eating behaviour is temporally related to the onset of food presentation or to the beginning of eating. Fig. 5 superimposes eleven traces of m.r.n.a. during eating behaviour in an

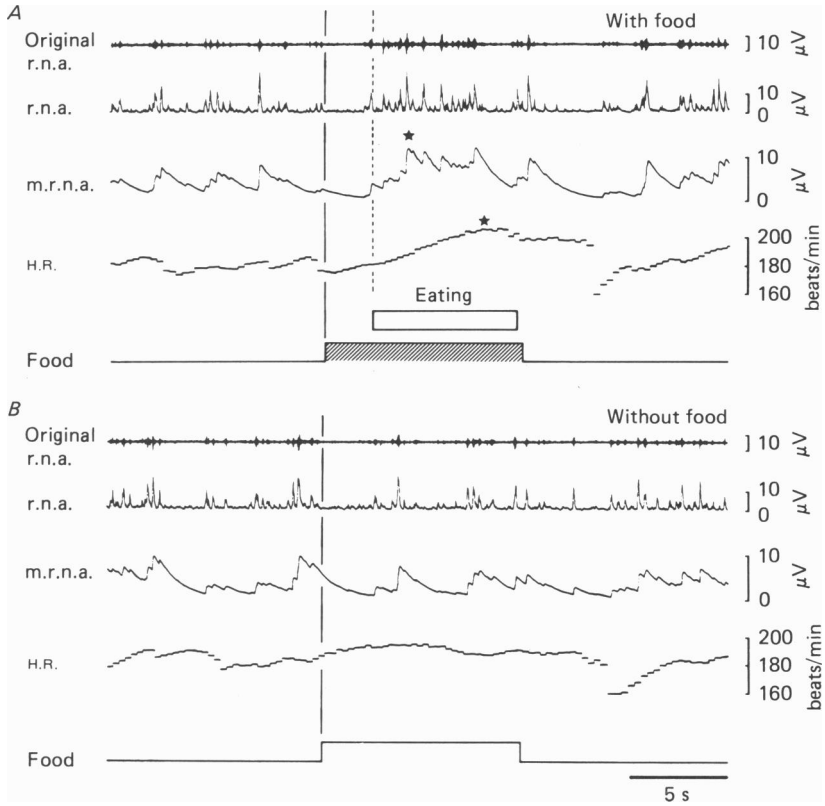

Fig. 4. Changes in renal sympathetic nerve activity (r.n.a.), mean renal sympathetic nerve activity (m.r.n.a.), and heart rate (H.R.) recorded in an awake cat in a food presentation trial with eating behaviour (*A*) and in the succeeding trial without eating behaviour (*B*). In *A*, eating behaviour is indicated by the open bar, food presentation by the hatched area. The r.n.a., m.r.n.a. and H.R. increased almost simultaneously with the onset of eating (a vertical dashed line). Filled stars show where m.r.n.a. and H.R. increased significantly. *B*, on presentation of an empty food box as indicated by the stimulus marker, r.n.a. and m.r.n.a. did not change significantly although H.R. seemed to increase slightly. Records in *A* and *B* were obtained on the fourth day after implantation surgery.

awake cat. In Fig. 5*A*, each trace of m.r.n.a. is aligned at the onset of the food presentation as indicated by a vertical continuous line. The time interval between food presentation and eating onset, as indicated by small filled arrows, varied from 1·5 to 7·9 s (2·8 ± 0·54 s: mean ± S.E. of mean). Also, the beginning of the increase in m.r.n.a. varied considerably around the onset of eating (Fig. 5*A*). In Fig. 5*B*, each trace of m.r.n.a. is aligned at the onset of eating as indicated by a vertical dashed line. The m.r.n.a. increased almost concomitantly with the onset of eating. Therefore, the increase in m.r.n.a. was closely related to the beginning of eating but not to the onset of food presentation.

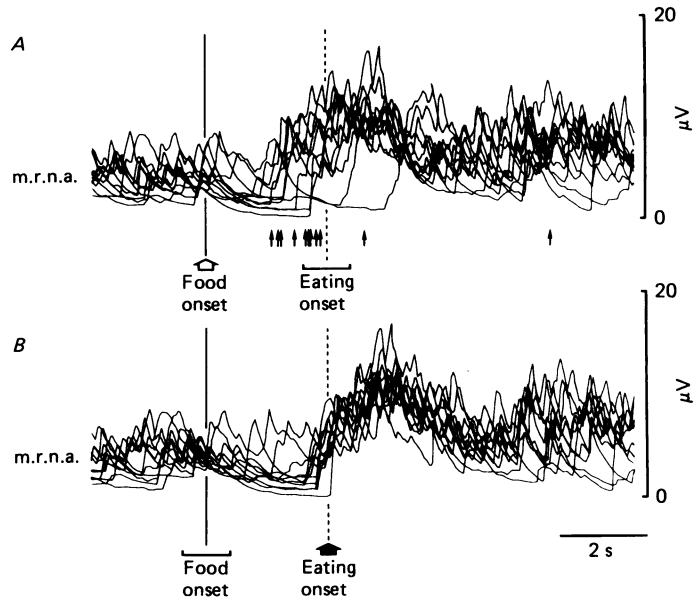

Fig. 5. Time relationship between the onsets of the increase in mean renal sympathetic nerve activity (m.r.n.a.), food presentation (food onset) and eating behaviour (eating onset) in an awake cat. Eleven traces of m.r.n.a. are superimposed. *A*, each trace of m.r.n.a. is aligned at the onset of food presentation as indicated by a vertical continuous line. The time interval between food onset and eating onset, as indicated by small filled arrows, varied considerably, and the mean and ±S.E. of mean are shown by the vertical dashed line and the horizontal bar which crosses it. *B*, each trace of m.r.n.a. is aligned at the onset of eating as indicated by a vertical dashed line. The m.r.n.a. increased almost concomitantly with the onset of eating. The mean ± S.E. of mean of the time interval between food onset and eating onset are shown by the vertical continuous line and the horizontal bar which crosses it. The records were obtained on the third day after implantation surgery.

Time courses and magnitudes of responses in renal sympathetic nerve activity, heart rate and arterial blood pressure during eating

During resting in the restrained state, the control value of H.R. obtained from thirteen awake cats was 166±6·9 beats/min; the control value of A.P. was 100±2·6 mmHg.

Fig. 6 illustrates relative changes in r.n.a., H.R. and A.P. obtained from thirteen awake cats before, during and after eating. Eating behaviour started 1–8 s after food presentation and persisted for 10·8±0·51 s ($n = 13$ cats) as shown by a horizontal open bar. The r.n.a. increased significantly with a latency of 0·5 s from the onset of eating (vertical dashed line) ($P < 0·001$). The increase in r.n.a. rapidly reached the peak value of 61±18% (open arrow in the upper panel) at 1·0 s after the onset of eating ($P < 0·001$). Thereafter, r.n.a. gradually decreased to approximately the control value by the end of the eating period. The r.n.a. decreased by 12±9·9% below the control value immediately after the end of eating; but, the decrease in r.n.a. was not statistically significant ($P > 0·05$).

H.R. increased significantly with a latency of 1·5 s from the onset of eating ($P <$

0·01). The increase in H.R. reached the peak value of 26±4·0 beats/min (16±2·4% of the control value, an open arrow in the middle panel) at 5·5 s after the onset of eating ($P < 0·001$). Then, H.R. gradually decreased approximately to the control value by the end of the eating period.

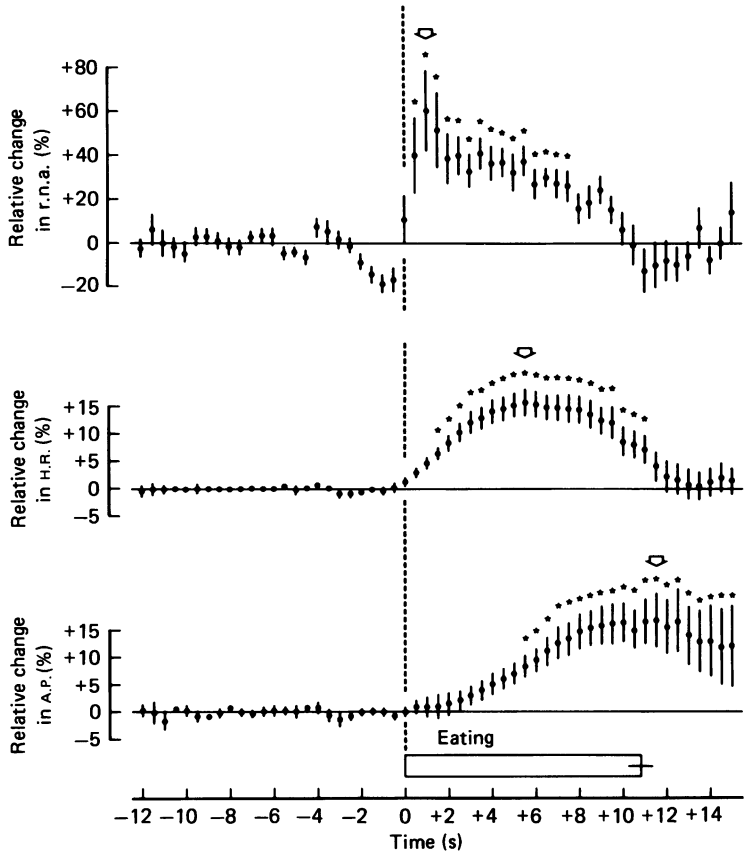

Fig. 6. Average percentage changes of renal sympathetic nerve activity (r.n.a.), heart rate (H.R.), and arterial blood pressure (A.P.) before, during and after eating obtained from thirteen awake cats are shown in upper, middle and lower panels, respectively. Average changes in r.n.a., H.R. and A.P. at 0·5 s intervals collected from seven to twenty-three trials in each individual cat and from these figures grand mean values were calculated for the thirteen cats. The means±S.E. of mean of each variable are indicated by dots and vertical bars. The onset of eating is shown by the vertical dashed line. The mean±S.E. of mean of the duration of eating ($n = 13$ cats) is shown by the horizontal open bar and line. Significant changes in each variable from the control variation are indicated by filled stars ($P < 0·05$).

A.P. increased significantly after a latency of 5·5 s from the onset of eating ($P <$ 0·01). The elevation in A.P. developed progressively during eating and reached the peak value of 17±4·9 mmHg (17±4·9% of the control value) at 11·5 s after the onset of eating (i.e. at 0·7 s after the end of eating), as shown by an open arrow in the lower panel ($P < 0·001$). The time courses and magnitude of the changes in H.R. and A.P. during eating were very similar to those recorded in our previous study (Matsukawa & Ninomiya, 1985).

The onset of the increase in r.n.a. preceded the onsets of the increases in H.R. and A.P. by 1·0 and 5·0 s, respectively. Also, the peak of the increase in r.n.a. preceded the peaks of the increases in H.R. and A.P. by 4·5 and by 10·5 s, respectively.

Renal sympathetic nerve activity during eating with administration of noradrenaline

Fig. 7 shows an example of changes in r.n.a. and m.r.n.a. during eating behaviour before (*A*) and 8 s after (*B*) intravenous administration of noradrenaline (2 μg/kg),

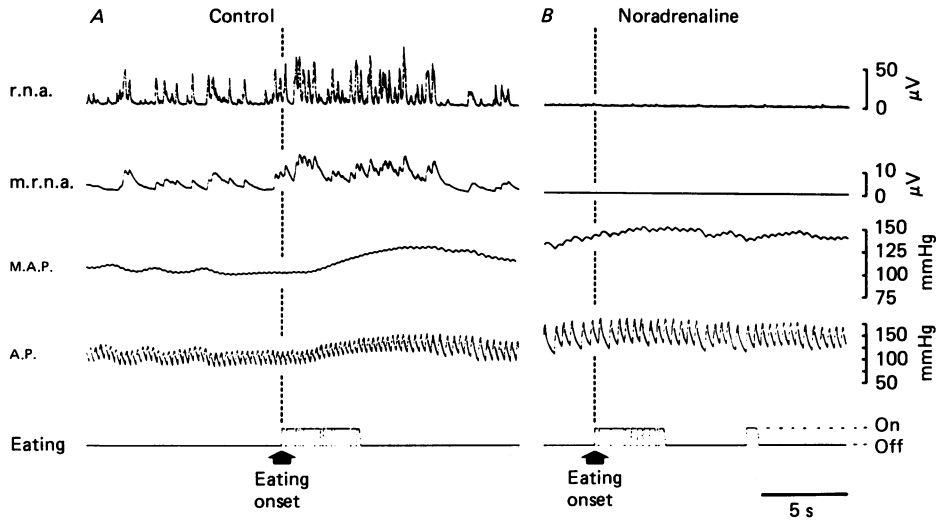

Fig. 7. Changes in renal sympathetic nerve activity (r.n.a.), mean renal sympathetic nerve activity (m.r.n.a.), mean arterial blood pressure (M.A.P.) and arterial blood pressure (A.P.) during eating before (*A*) and after (*B*) intravenous administration of noradrenaline (2 μg/kg). *A*, during eating, the cardiac-related grouped discharges of r.n.a. increased over those before eating, although A.P. was elevated. *B*, when A.P. was highly elevated by administration of noradrenaline, r.n.a. and m.r.n.a. during resting were almost completely inhibited, and there were no increases in r.n.a. and m.r.n.a. during eating. The stimulus marker indicates the voltage signal detected by an electrode within a food box; when the cat touched food, the eating signal was in an upper level (on state). The records were obtained on the third day after implantation surgery.

as well as changes in M.A.P. and A.P. In the control (Fig. 7*A*), the r.n.a. showed spontaneous grouped discharges synchronous with the cardiac cycle as previously reported in conscious cats (Ninomiya *et al.* 1976; Ninomiya & Yonezawa, 1979). Also, during eating, the r.n.a. showed the cardiac-related grouped discharges. As can be seen in Fig. 7*A*, the cardiac-related grouped discharges of r.n.a. increased during eating over those before eating. These results indicated that the pattern of r.n.a. is baroreceptor dependent, not only during resting but also during eating.

When A.P. was highly elevated by administration of noradrenaline (2–5 μg/kg I.V.) in thirteen cats, r.n.a. and m.r.n.a. during resting were almost completely inhibited for a period of 73 ± 9·4 s (e.g. in Figs. 2*A* and 7*B*). When food was presented 7–34 s after injection of noradrenaline, there was eating behaviour but the increase in

r.n.a. and m.r.n.a. was not induced as shown in Fig. 7*B*. Thus the increase in r.n.a. associated with feeding was not able to overcome the baroreceptor-dependent inhibition of r.n.a.

DISCUSSION

In the present study, on conscious cats, we measured simultaneously changes in r.n.a., H.R. and A.P. in response to food presentation. Our new findings are that r.n.a., H.R. and A.P. increased in association with eating but not as firmly with the food presentation, and that the increases in r.n.a. and H.R. occurred almost simultaneously with the onset of eating whereas the elevation in A.P. followed the onset of eating. The abrupt increase in r.n.a. strongly suggests that central activation of the sympathetic efferent activity to the kidney is involved in the cardiovascular adjustment associated with eating behaviour.

To facilitate quantitative determination of the time courses and magnitudes of the changes in r.n.a., H.R. and A.P. during eating, the cats were restrained. Therefore, there is the possibility that restriction of body trunk movements may have affected the changes in sympathetic nerve activity and haemodynamics during eating. However, the resting values of H.R. (166 beats/min) and A.P. (100 mmHg) in the present restrained cats were almost the same as those recorded in unrestrained, free-moving cats (Ninomiya, Matsukawa, Honda, Nishiura & Shirai, 1986). Moreover, the responses of H.R. and A.P. during eating in the restrained cats were almost coincident with their responses in unrestrained baboons (Vatner *et al.* 1974) and cats (Ninomiya, Nishiura, Okada & Hirata, 1981). Therefore, it seems unlikely that the restriction of movement affected the changes in sympathetic nerve activity and haemodynamics we recorded during eating, particularly because the cats were well accustomed to such restraint.

We suggested previously that transient increases in H.R. and A.P. were associated with eating because H.R. and A.P. increased significantly in 86–98% of food presentation trials where eating was evoked, but they did not change significantly in 75–77% of food presentation trials where eating was not evoked, despite the presentation of food (Matsukawa & Ninomiya, 1985). In the present study, r.n.a. increased significantly in 65% of food presentation trials with eating, but did not change significantly in 71% of food presentation trials without eating. In addition, when an empty food box was presented and there was no eating, r.n.a. did not change significantly as shown in Fig. 4*B*. Therefore, it may be concluded that the increase in r.n.a. and the cardiovascular changes recorded during eating in the present experiments were associated with eating behaviour but not with food presentation.

The renal sympathetic nerve in the cat has been known anatomically (Kuo, de Groat & Nadelhaft, 1982; Kuo, Nadelhaft, Hisamitsu & de Groat, 1983) and electrophysiologically (Åström & Crafoord, 1968; Beacham & Kunze, 1969) to include both sympathetic efferent and afferent fibres. Because the renal nerve branches examined in most conscious cats in this study were intact, the possibility that the recorded r.n.a. may be partially from renal afferent activity cannot be ignored. However, the r.n.a. during resting was almost completely inhibited by intravenous administration of noradrenaline (Figs. 2*A* and 7*B*) or hexamethonium bromide (Fig. 2*B*). Even if

eating was evoked after intravenous administration of noradrenaline, there was no increase in r.n.a. during eating as shown in Fig. 7*B*. Furthermore, r.n.a. increased during eating even when the renal nerve was sectioned distal to the recording electrode. These results indicate that (1) the r.n.a. measured both during resting and during eating originated mainly from sympathetic post-ganglionic efferent fibres and not from afferent fibres, and (2) artifacts which corresponded to body movements and/or muscular activities accompanied by eating behaviour were negligible in our neural recordings.

The fact that the increase in r.n.a. occurred almost concomitantly with the onset of eating (Fig. 6) suggests that this was not a viscero-autonomic reflex evoked by food intake. Moreover, the increase in r.n.a. was not preceded by a decrease in arterial pressure so it was unlikely to have been a reflex initiated by baroreceptors. Rather, the increase in r.n.a. was followed by an increase in arterial pressure to which it probably contributed by causing renal vasoconstriction (see below). Thus, it seems most likely that the abrupt increase in r.n.a. at the beginning of eating was induced directly by the descending input from the central nervous system.

Although a baroreceptor–r.n.a. reflex did not apparently contribute in inducing the increase in r.n.a. at the beginning of eating, it seems likely that the baroreflex did affect r.n.a. during eating, for the following reasons. First, the r.n.a. showed cardiac-related grouped discharges not only during resting but also during eating. The cardiac-related discharges of r.n.a. were enhanced during eating (Fig. 7*A*). Secondly, the increase in r.n.a. was gradually attenuated as A.P. elevated progressively at the later stage of eating as shown in Fig. 6. Moreover, when A.P. reached its maximum value immediately after the end of eating, r.n.a. tended to decrease below the control level. Thirdly, the increase in r.n.a. during eating was abolished when hypertension had been induced by intravenous administration of noradrenaline (Fig. 7*B*). These findings therefore suggest that afferent input from the arterial baroreceptors exerted an inhibitory influence on r.n.a. during eating, particularly at the later stage of eating. Accordingly, elimination of the baroreceptor input might be expected to reinforce the increases in r.n.a. and A.P. during eating. Indeed, in the rat, lesions of the nucleus tractus solitarii which receives input from the baroreceptors were reported to augment the increase in A.P. recorded during eating behaviour (Buchholz & Nathan, 1984).

It is known that electrical stimulation of the renal nerve evokes a decrease in renal blood flow and an increase in renal vascular resistance (Selkurt, 1963). The abrupt and marked increase in r.n.a. at the beginning of eating would therefore be expected to evoke an increase in renal vascular resistance. In fact, a transient renal vascular vasoconstriction was observed during eating in the baboon and the dog, and the increase in renal vascular resistance during eating was reduced by α-adrenergic blocker but not by atropine (Vatner *et al.* 1970*b*, 1974). Therefore, it is likely that the transient increase in r.n.a. we have recorded is responsible for renal vasoconstriction at the beginning of eating. Such a positive correlation between changes in r.n.a. and renal vascular resistance has been reported during excitement in the conscious dog (Gross & Kirchheim, 1980).

Ninomiya *et al.* (1981) in a preliminary report stated that cardiac sympathetic

nerve activity (c.s.n.a.) increased at the beginning of eating in the unrestrained, free-moving cat. It was proposed that this abrupt increase in c.s.n.a. induced the transient tachycardia at the beginning of eating. On the basis of the present study, it seems that both r.n.a. and c.s.n.a. are activated at the beginning of eating as part of a centrally organized pattern of response.

In conclusion, the results obtained from simultaneous recording of r.n.a., H.R. and A.P. in conscious animals indicate that the abrupt increase in r.n.a. associated with eating behaviour is probably caused by a descending input from higher central nervous system rather than either by a viscero-autonomic reflex due to food intake or by a baroreflex.

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