J. Physiol. (1982), **326**, *pp.* 309–318
With 6 text-figures
Printed in Great Britain

THE EFFECTS OF NUCLEUS RAPHE MAGNUS LESIONS ON AN ASCENDING THERMAL PATHWAY IN THE RAT

BY DAVID C. M. TAYLOR*
From the National Institute for Medical Research, Mill Hill, London, NW7 1AA

(*Received 4 August 1981*)

SUMMARY

1. In the thalamus and hypothalamus of rats, anaesthetized with Urethane, single unit recordings have been made from cells which respond to small innocuous changes in scrotal skin temperature applied with a water-perfused brass thermode.

2. Once a scrotal temperature-sensitive neurone had been isolated the brain stem was electrolytically lesioned through implanted tungsten electrodes to determine whether the input from the scrotal skin temperature sensors ascends through the brain stem lemniscal pathways or the mid-line raphe nuclei. All recording sites and lesions were identified histologically.

3. Thirty-six neurones have been studied of which half were located in the ventrobasal thalamus, six were located in the anterior thalamic nuclei and the remainder were in the medial hypothalamus.

4. The nucleus raphe magnus was lesioned on eighteen separate occasions; in each case the temperature-responsive activity of the thalamic or hypothalamic neurone was abolished.

5. Extensive brain-stem lesions which spared only the mid-line nucleus raphe magnus had no discernible effect on the responses of the thalamic or hypothalamic neurones to scrotal skin temperature.

6. The ascending pathway from the thermal sensors of the rat scrotal skin must pass through, or relay in, the nucleus raphe magnus.

INTRODUCTION

This paper is concerned with the anatomy of the pathway ascending from the thermal sensors in the rat scrotum. It will be shown, using a combination of acute lesion and electrophysiological recording techniques, that the ascending fibres from the ventrolateral quadrant of the spinal cord do not pass directly to the thalamus through the lateral lemniscus but pass through, and possibly relay in, the nucleus raphe magnus.

Acute spinal cord lesion experiments in rats have shown that the input from the periphery to thalamic neurones which respond to scrotal skin temperature exclusively passes up the ventrolateral quadrants of the spinal cord, and that each of the

* Present address: Max-Planck-Institut für Physiologische und Klinische Forschung, W.G. Kerckhoff-Institut, Parkstrasse 1, 6350 Bad Nauheim, W. Germany.

quadrants serves the thalamus on the same side (Taylor, 1979). In this respect the pathway follows a similar course to the thermoafferent pathways described in other species, since unilateral lesions of the dorsal part of the ventrobasal funiculus have been shown to abolish contralateral temperature sensitivity in humans (Foerster & Gagel, 1932) and seriously impair the sensitivity in cats (Norrsell, 1979).

Histological studies, in which the ascending fibre tracts were severed and the resulting degeneration was traced, have shown that the ventrobasal quadrants of the spinal cord do not only comprise ascending fibres which terminate in the ventrobasal thalamus. Some terminal and preterminal degeneration has been observed in the nuclei of the brainstem, notably the nucleus raphe magnus, in both rats (Zemlan, Leonard, Kow & Pfaff, 1978) and cats (Brodal, Walberg & Taber, 1960). The degeneration could have been due to either terminating ascending fibres or collaterals of lemniscal fibres. Since some neurones in the raphe nuclei respond to scrotal skin temperature (Jahns, 1976) they might receive an input from collaterals of ascending spinothalamic fibres. An alternative is that the scrotal thermoafferent pathway actually relays in the raphe nuclei before ascending to the thalamus and hypothalamus. To decide between these two possibilities the nucleus raphe magnus, medial or lateral lemnisci were lesioned through implanted electrodes whilst activity was recorded from thalamic or hypothalamic neurones which responded to scrotal skin temperature. A preliminary report of this work has already been published (Taylor, 1980).

METHODS

Preparation. Male Sprague-Dawley rats (250–300g) were used, anaesthetized with Urethane (ethyl carbamate; 1·25–1·5g/kg I.P.) supplemented as necessary to maintain areflexia. The scrotum was clipped and a depilatory cream (Buto; Biometica Ltd.) was applied for one minute, after which the skin was rinsed with cool water to minimize any inflammation which might occur. The rat was then placed in a head holder on a stereotaxic carriage (Lister & Woodget, 1972) and the body temperature controlled at 38 °C by a rectal probe connected to a servo-controlled heating pad. A dental drill was used to perform craniotomies which allowed access to the thalamic and hypothalamic nuclei and the nucleus raphe magnus. The exposed cortical surface was covered by an oil pool as described elsewhere (Hellon & Taylor, 1982).

Brain lesioning procedure. The lesioning electrode was a 250 μm diameter tungsten wire sharpened by hand and varnished, with a 250 μm exposed tip. At the beginning of the experiment a lesioning electrode was lowered into the brain stem stereotaxically and cemented in place. The co-ordinates corresponding to the nucleus raphe magnus, with the head orientated according to Albe-Fessard, Stutinsky & Libouban (1966) were: 3 mm posterior, mid line, −0·5 mm deep. Sometimes an array of up to six lesioning electrodes was implanted, depending on the extent of the lesion required. A current of 250 μA for 20 s was found sufficient to produce discrete lesions in the brain stem.

Skin temperature stimulus. The scrotal skin was in contact with a water-perfused brass thermode (30 × 30 × 5 mm) with a thermistor bead cemented to the thermode at the interface between the skin and the thermode. The thermistor was connected to a Wheatstone Bridge calibrated over the range of 20–45 °C.To increase thermal conductivity an oil film was introduced between the scrotum and the thermode. The temperature of the thermode was controlled by mixing the inflow from two thermostatically controlled water-baths at 45 and 10 °C. When searching for thermoresponsive cells cyclic temperature changes were applied to the skin. These were provided by interrupting the outflow from the cold bath by a rotary pump, modified to occlude the tube for $\frac{2}{3}$ of each cycle. The temperature was cycled between 30 and 42 °C every 10 s. Once a thermoresponsive neurone had been identified, a series of static temperature plateaux were applied to the scrotum by turning off the rotary pump and adjusting the water flow manually.

Experimental procedure. Glass micropipettes filled with 2% Pontamine Sky Blue solution in sodium acetate 0·5 mol/l were used (4–10 MΩ impedance). The electrode was lowered into either

the thalamus or hypothalamus whilst the scrotal temperature was oscillating. When a thermoresponsive neurone was encountered the firing rate of the unit was determined at several steady scrotal skin temperatures. Once the response properties of a neurone were known, the lesion was made and the neurone re-tested. Finally a dye mark was made at the recording site and the rat perfused with 10% formyl saline. All lesion and recording sites were examined histologically.

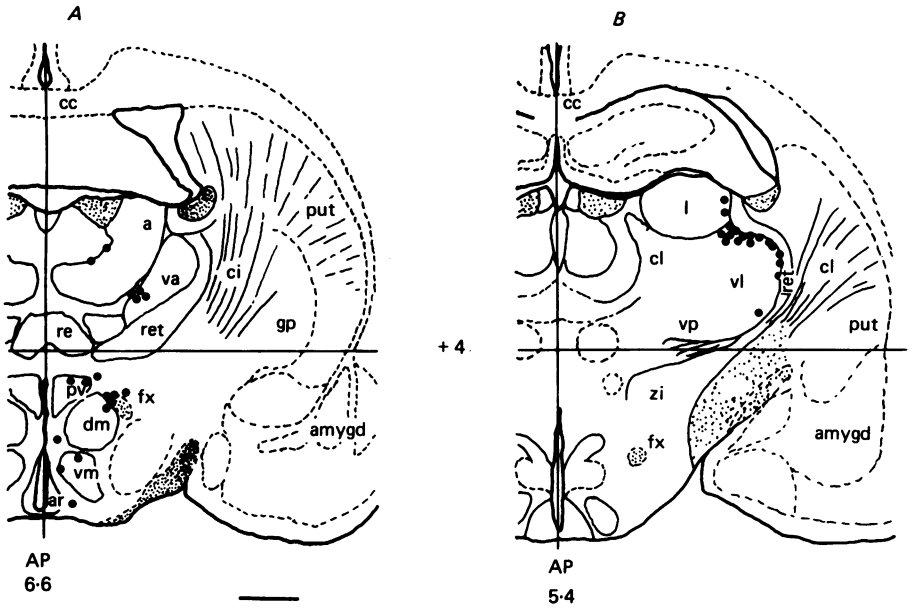

Fig. 1. The location of the thirty-six recording sites for the raphe lesion experiments superimposed on diagrams of standard sections 6·6 mm (*A*) and 5·4 mm (*B*) rostral to the ear bar; taken from Albe-Fessard *et al.* (1966). The scale bar represents 1 mm. Abbreviations are: a, n. anterior thalami; amygd, amygdala; ar, arcuate nucleus; cc, corpus callosum; ci, Internal capsule; cl, n. centralis lateralis thalami; dm, n. dorsomedialis hypothalami; fx, fornix; gp, globus pallidus; l, n. lateralis thalami; put, putamen; pv, n. paraventricularis hypothalami; re, n. reuniens; ret, n. reticularis thalami; va, n. ventralis anterior thalami; vl, n. ventralis lateralis thalami; vm, n. ventromedialis hypothalami; vp, n. ventralis posterior thalami.

RESULTS

Thirty-six neurones responsive to scrotal skin temperatures have been studied of which half were in the ventrobasal thalamus and the rest in other thalamic nuclei or the hypothalamus. The positions of all the recording sites are shown in Fig. 1. The ventrobasal neurones were located at the lateral edge of the rostral ventrobasal thalamus (vp in Fig. 1) where it merges with the nucleus ventralis lateralis (Fig. 1*B*). A further six thalamic neurones were observed more rostrally within the anterior and ventral anterior nuclei of the thalamus (Fig. 1*A*). The hypothalamic neurones were located in and around the paraventricular, dorsomedial and ventromedial nuclei of the hypothalamus (Fig. 1*A*). All of these recording sites correspond to those reported by other workers in the thalamus (Hellon & Misra, 1973; Schingnitz & Werner, 1980) and hypothalamus (Nakayama, Ishikawa & Tsurutani, 1979).

All lesion sites were examined histologically and Fig. 2 shows the extent of one of the smallest lesions which destroyed the nucleus raphe magnus. The lesion is discrete

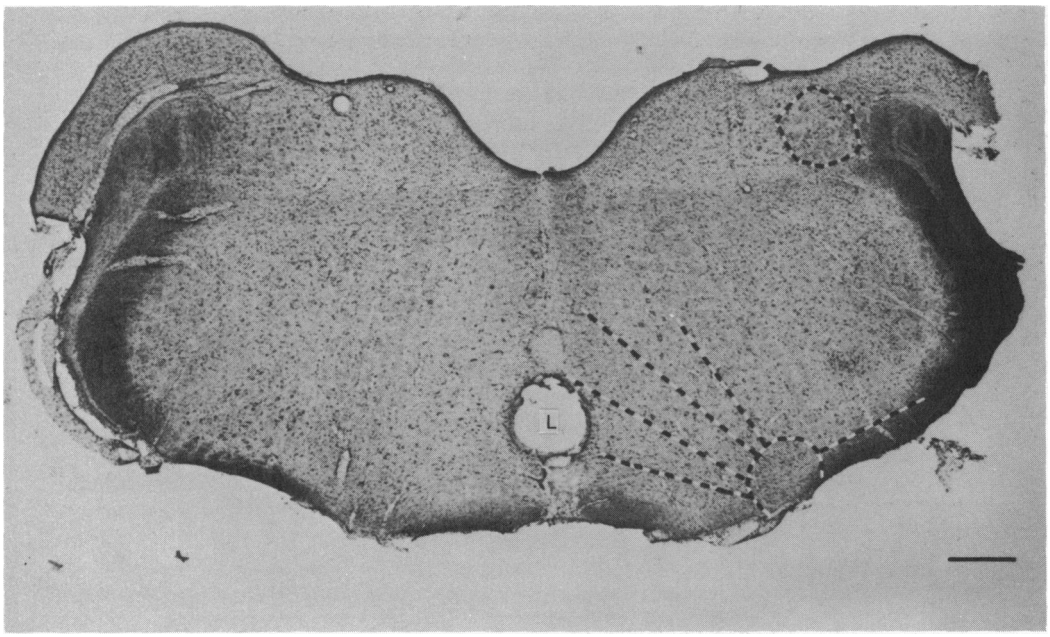

Fig. 2. Photograph of a 60 μm section of brain stem stained with cresyl fast violet, showing the extent of a lesion (L) which incorporates the nucleus raphe magnus. The dotted lines and areas enclosed by them coincide with the pattern of the ascending fibres observed by Zemlan *et al.* (1978), the ventral cluster of fibres corresponds with those that will join the lateral lemniscus. The dotted lines irradiating from the ventral cluster indicate the less concentrated terminal and pre-terminal degeneration observed in the medial lemniscus and reticular nuclei. The dorsal cluster of fibres coincides with the lateral vestibular nucleus. For further details see text. The scale bar represents 0·5 mm.

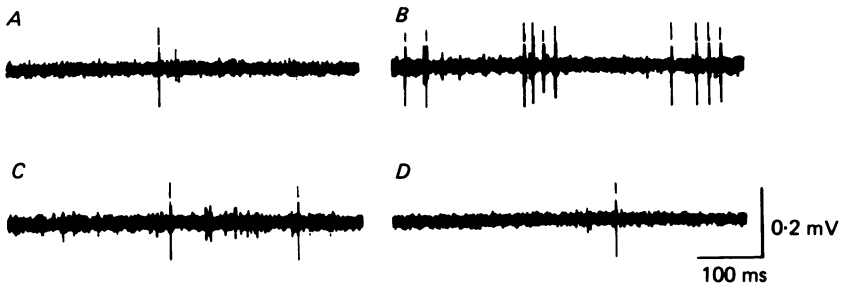

Fig. 3. The response of a thalamic neurone at two different scrotal skin temperatures, before (*A*, *B*) and after (*C*, *D*) a lesion of the nucleus raphe magnus. The scrotal temperature in *A* and *C* is 37 °C, and in *B* and *D*, 41 °C. The lesion abolished the temperature response of the neurone.

and does not interfere with the ascending lemniscal pathways from the ventrolateral quadrant of the spinal cord identified by Zemlan *et al.* (1978), shown by the dotted lines on the photograph.

Thalamic recordings

Fig. 3 shows the effect of lesioning the nucleus raphe magnus on the response of a scrotal temperature sensitive neurone in the ventrobasal thalamus. Before the lesion, the firing rate of the neurone increased abruptly from a very low rate (0·5 Hz) at 37 °C (Fig. 3*A*) to 20 Hz when the scrotum was warmed to temperatures greater than 40 °C (Fig. 3*B*). This warm reactive response was typical of the majority of neurones observed in this study. The lesion shown in Fig. 2 completely abolished the scrotal temperature response of the neurone as shown in Fig. 3*C* and *D*. After the lesion, the firing rate of the neurone at a scrotal skin temperature of 41 °C (Fig. 3*D*) was similar to that observed at a scrotal skin temperature of 37 °C both before (Fig. 3*A*) and after (Fig. 3*C*) the lesion.

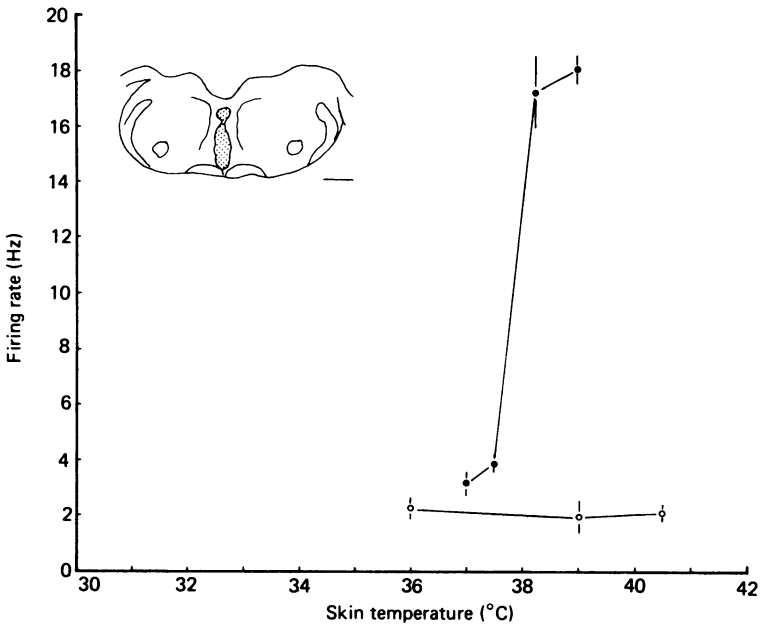

Fig. 4. The effect of a brain stem lesion on the response of a thalamic cell to steady scrotal skin temperatures. Filled circles show the response before the lesion, open circles show the response after the lesion. The insert shows the extent of the lesion (stippled area), scale bar represents 1 mm. Error bars show ± S.D. The lesion abolishes the response of the neurone.

The effect of destroying the nucleus raphe magnus on the response of another neurone in the ventrobasal thalamus is shown in Fig. 4. The extent of the lesion is shown by the stippled area on the insert. The abrupt increase in firing rate evident when the scrotal skin is warmed from 38 to 38·5 °C (filled circles) was completely abolished when the nucleus raphe magnus was lesioned. In contrast, lesions which spared the nucleus raphe magnus had no discernible effect on the responses of thalamic neurones to different scrotal skin temperatures.

Fig. 5 shows the effect of a lesion just lateral to the mid line which interrupted the medial lemniscus ipsilateral to the thalamic recording site. The nucleus raphe magnus remained intact and there was no apparent change in the response of the neurone to scrotal skin temperature changes. Similar lesions contralateral to the thalamic recording site had no effect on the scrotal temperature-responsive neurones. The activity of thalamic scrotal temperature-responsive neurones was only affected by those lesions which could be shown histologically to have damaged or destroyed the nucleus raphe magnus.

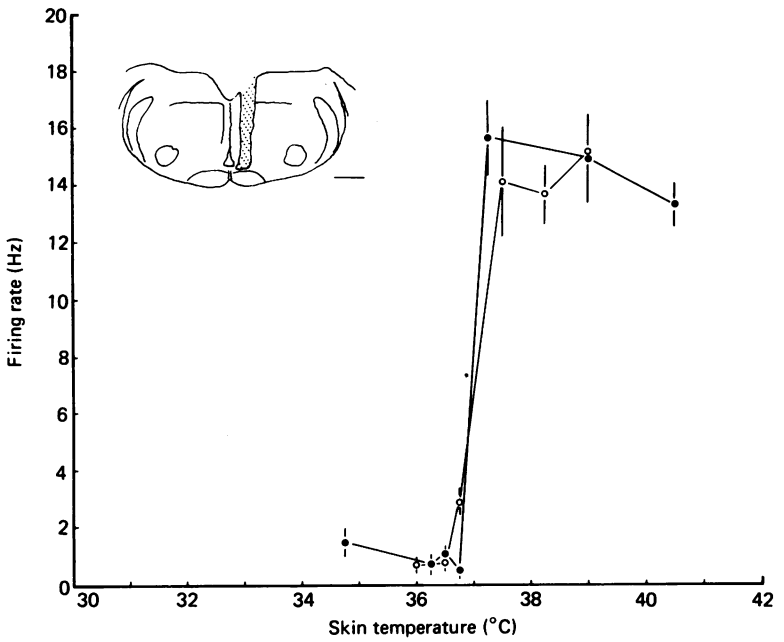

Fig. 5. The response of a neurone in the thalamus to scrotal skin temperature, before and after a lesion which spared the nucleus raphe magnus. The lesion (stippled area on insert) interrupts the medial lemniscus ipsilateral to the recording electrode. Other details as in the previous figure.

The nucleus raphe magnus was lesioned in twelve rats and in each the temperature response of the thalamic neurones was completely abolished. The smallest lesion which abolished the activity of the neurone is shown in Fig. 6*A* (right). Twelve lesions were made which spared the nucleus raphe magnus, four were extensive and spared only the mid line, the remainder were more discrete. The total area encompassed by these lesions, which had no effect on the response of the neurone being studied, is also shown in Fig. 6*A* (left). Only those lesions which damaged the nucleus raphe magnus had any effect on the response of the neurone being tested.

Hypothalamic recordings

Twelve of the neurones studied were situated in and around the hypothalamic nuclei (Fig. 1*A*). The nucleus raphe magnus was lesioned on six occasions. The results obtained were identical to those observed for thalamic neurones. The smallest lesion

which abolished the skin temperature response of the hypothalamic neurones is shown in Fig. 6*B* and coincides with the nucleus raphe magnus. In six experiments the lesions spared the nucleus raphe magnus. Three of the lesions were large and bilateral whereas the others lesioned only the medial or lateral lemnisci. Only those lesions which destroyed the nucleus raphe magnus affected the firing rate of the temperature responsive neurones.

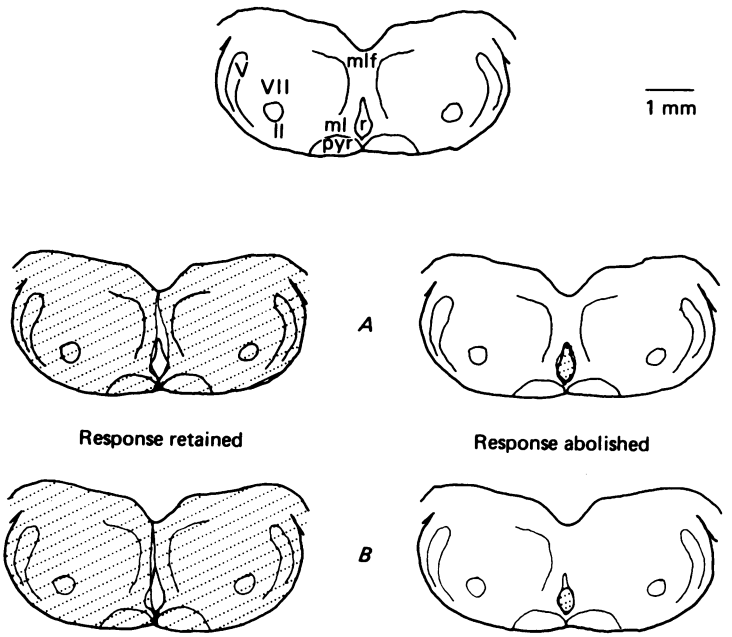

Fig. 6. The cumulative extent of the lesions in the raphe lesion experiments. The shaded areas show the lesions employed in *A*, the eighteen ventrobasal recordings; *B*, the eighteen recordings from the other thalamic and hypothalamic nuclei. The diagrams on the right show the smallest lesion in each series which abolished the response of the diencephalic neurone. The diagrams on the left show the cumulative extent of twelve (*A*) and six (*B*) lesions which had no effect on the response of the cell under investigation. Abbreviations used in the top diagram: v, nucleus of the spinal trigeminal nerve; vii, nucleus of the facial nerve; r, nucleus raphe magnus; pyr, pyramidal tract; ml, medial lemniscus; ll, lateral lemniscus.

DISCUSSION

It has been believed for many years that the nerve fibres subserving cutaneous pain and temperature sensation in man pass from the dorsal horn of the spinal cord into the contralateral anterolateral quadrant and then ascend, presumably to the thalamus, via the lateral lemniscus (Foerster & Gagel, 1932; Ranson & Clarke, 1947). Electrical stimulation of the anterolateral quadrant of the monkey spinal cord antidromically activates temperature responsive neurones in the lumbar dorsal horn (Handwerker, Iggo, Ogawa & Ramsey, 1974). Although the equivalent experiments have not been performed in rats, the results of the spinal cord lesion experiments (Taylor, 1979) indicate that the thermoafferent fibres from the rat scrotum pass up the anterolateral quadrants. However, neither the spinal cord lesion nor the

stimulation experiments can be taken as evidence for a direct spinothalamic projection.

Histological evidence for the sites of termination of anterolateral quadrant ascending fibres has been provided by Zemlan *et al.* (1978), who traced the degeneration following lesions of the anterolateral quadrants using a silver impregnation technique. The distribution of fibres at the level of the facial nerve is shown by the dotted lines in Fig. 2. Preterminal degeneration and degenerating fibres were seen in the nucleus raphe magnus, reticular nucleus gigantocellularis and the lateral vestibular nucleus. The majority of the fibres passed up the lateral lemniscus to terminate in the superior colliculi, central grey matter, nucleus parafascicularis and the lateral and rostral edges of the ventrobasal thalamus.

As stated in the Introduction it is clear that the fibres which convey the information from the scrotal thermoresponsive neurones in the rat dorsal horn pass up the anterolateral quadrant (Taylor, 1979). The fibres might pass directly to the ventrobasal thalamus or terminate in the brain stem. Skin temperature-responsive neurones have been identified in the medianus and dorsalis raphe nuclei which respond to scrotal temperature stimulation (Jahns, 1976) and in the nucleus raphe magnus which respond to changes in trunk skin temperature (Dickenson, 1977). Since the nucleus raphe magnus is known to receive a major input from the spinal cord (Brodal *et al.* 1960; Zemlan *et al.* 1978) and to project to the other raphe nuclei (Conrad, Leonard & Pfaff, 1974; Bobillier, Sequin, Petitjeau, Salvert, Touret & Jouvet, 1976), it appears possible that the nucleus raphe magnus might receive an input from the spinal cord and project, either directly or indirectly, to those higher centres known to receive an input from the scrotal skin thermosensors. If this hypothesis were true then the nucleus raphe magnus would contain neurones responsive to scrotal skin temperature, which in fact the following paper proves to be the case (Hellon & Taylor, 1982).

The results of the present study clearly demonstrate that the input to the thalamic and hypothalamic nuclei from the scrotal skin temperature sensors depends upon the integrity of the nucleus raphe magnus. It is possible that the raphe magnus lesions could have damaged collaterals from spinothalamic tract fibres, thereby producing a conduction block at the junction of the collateral and the ascending fibre (Barron & Matthews, 1935). However, the abolition or inhibition of the temperature response was observed for up to an hour following the lesion, with no recurrence of activity. Furthermore, lesions of the brain stem which spared only the raphe magnus had no effect on the response of either thalamic or hypothalamic neurones, so the ascending fibres must lie in the nucleus raphe magnus. This indicates that the nucleus raphe magnus does not receive a collateral input from the lemniscal pathways.

The possibility that the nucleus raphe magnus lesions may have had a general effect on the state of the preparation may be discounted since many thalamic neurones responsive to innocuous mechanical stimulation of the hind-quarters and scrotum retained their activity unchanged after the lesion.

The nucleus raphe magnus might also exert a modulatory role at either spinal or supra-spinal levels, as it is known to do in the ascending nociceptive pathways (Besson, Dickenson, LeBars & Oliveras, 1978). Stimulation of the nucleus raphe magnus has no effect on the spinal trigeminal neurones which respond to facial skin temperature (Dawson, Dickenson, Hellon & Woolf, 1981). There is evidence, however, that the scrotal skin temperature responsive neurones in the dorsal horn of the rat

spinal cord are subject to tonic descending control (Pierau, Neya, Yamasato & Ulrich, 1980). Cooling the thoracic spinal cord, which reversibly blocks descending pathways, decreases the intrinsic firing rate of 60% of the scrotal temperature-responsive neurones in the spinal cord but the activity of the remaining temperature-responsive neurones is usually increased. This modulation of activity might originate from the raphe nuclei. Such a possibility is not excluded by the results of this study.

Since the input to the hypothalamus from trunk (Dickenson, 1977, 1978) and scrotal skin thermal sensors appears to pass through the nucleus raphe magnus, one might expect lesions of this area to affect thermoregulation. Lesions of the nucleus raphe magnus in pigeons abolish their day/night temperature rhythm, causing the animals to regulate their temperature at a new intermediate value (Necker & Wegner, 1980). In contrast, stimulating the nucleus raphe magnus in guinea pigs appears not to have any effect on heat production in the cold, although the stimulation excited hypothalamic neurones which responded to warming the skin, but inhibited hypothalamic neurones which responded to skin cooling (Brück & Hinckel, 1980). The projection from the raphe nuclei to the hypothalamus is probably mainly serotonergic (Dahlström & Fuxe, 1964; Conrad *et al.* 1974; Bobillier *et al.* 1976). Biochemical lesioning of the raphe nuclei by depleting rabbits (Lin, Pang, Chern & Chia, 1978) and rats (Lin, Chern & Chern, 1978) of serotonin with *p*-chlorophenylalanine causes a decrease in rectal temperature but also increases the metabolic response of the animals to cold. The authors explain the paradoxical effects of serotonin as being due to the action of *p*-chlorophenylalanine inhibiting serotonin synthesis both centrally and peripherally.

One may conclude that the nucleus raphe magnus plays a role in thermoregulation, the nature and importance of which is not understood. The results presented here and in the following paper (Hellon & Taylor, 1982) indicate that the nucleus forms an essential link in the ascending thermal pathway from the rat scrotum, which could be an important input to the thermoregulatory centres (Waites, 1976). This suggestion conforms with Dickenson's results in rats which showed that the nucleus raphe magnus receives an input from the cutaneous thermal sensors of the trunk and projects to the hypothalamus (Dickenson, 1978). It is consistent with the observation that extensive brain stem lesions which spare ony the nucleus raphe magnus have no discernible effect upon the temperature responsive cells in either the thalamus or hypothalamus.

I wish to express my thanks to Dr R. F. Hellon for his help and guidance throughout this study. My thanks also to Mr G. E. Goldsmith and Miss J. Bagnall for excellent technical assistance, and to Prof. Fr.-K. Pierau for his helpful comments on this manuscript. I was supported by a M.R.C. Research Studentship.

REFERENCES

ALBE-FESSARD, D., STUTINSKY, F. & LIBOUBAN, S. (1966). *Atlas stéréotaxique du diencephale du rat blanc.* Paris: C.N.R.S.

BARRON, D. H. & MATTHEWS, B. H. C. (1935). Intermittent conduction in the spinal cord. *J. Physiol.* **85**, 73–103.

BESSON, J.-M., DICKENSON, A. H., LEBARS, D. & OLIVERAS, J.-L. (1978). Opiate analgesia: the physiology and pharmacology of spinal pain systems. In *Advances in Pharmacology and Therapeutics*, vol. 5, ed. DUMONT, C. Oxford & New York: Pergamon Press.

BOBILLIER, P., SEQUIN, S., PETITJEAU, F., SALVERT, D., TOURET, M. & JOUVET, M. (1976). The raphe nuclei of the cat brain stem: a topographical atlas of their afferent projections as revealed by autoradiography. *Brain Res.* **113**, 449–486.

BRODAL, A., WALBERG, F. & TABER, E. (1960). The raphe nuclei of the brainstem in the cat. III. Afferent connections. *J. comp. Neurol.* **114**, 261–282.

BRÜCK, K. & HINCKEL, P. (1980). Thermoregulatory noradrenergic and serotonergic pathways to hypothalamic units. *J. Physiol.* **304**, 193–202.

CONRAD, L. C. A., LEONARD, C. M. & PFAFF, D. W. (1974). Connections of the median and dorsal raphe nuclei in the rat; an autoradiographic and degeneration study. *J. comp. Neurol.* **156**, 176–206.

DAHLSTRÖM, A. & FUXE, K. (1964). Evidence for existence of monoamine containing neurones in the central nervous system. I. Demonstration of monoamines in the cell bodies of brain stem neurones. *Acta physiol. scand.* **62**, suppl. 232, 1–55.

DAWSON, N. J., DICKENSON, A. H., HELLON, R. F. & WOOLF, C. J. (1981). Inhibitory controls on thermal neurones in the spinal trigeminal nucleus of cats and rats. *Brain Res.* **209**, 440–445.

DICKENSON, A. H. (1977). Specific responses of rat raphe neurones to skin temperature. *J. Physiol.* **273**, 277–294.

DICKENSON, A. H. (1978). Serotonergic raphe neurones involved in an ascending thermal pathway. In *New trends in Thermal Physiology*, Proc. Satellite Symp. on Thermal Regulation, Lille, France, July 1977, XXVII, Int. Congr. of Physiol. Sci., ed. HOUDAS, Y. & GUIEU, J. D., pp. 50–52. Paris: Masson.

FOERSTER, O. & GAGEL, O. (1932). Die Vorderseitenstrangdurchschneidung beim Menschen. *Z. ges. Neurol. Psychiat.* **138**, 1–92.

HANDWERKER, H. O., IGGO, A., OGAWA, H. & RAMSEY, R. L. (1974). Input characteristics and rostral projection of dorsal horn neurones in the monkey. *J. Physiol.* **244**, 76–77*P*.

HELLON, R. F. & MISRA, N. K. (1973). Neurones in the ventrobasal thalamus responding to scrotal skin temperature. *J. Physiol.* **232**, 375–388.

HELLON, R. F. & TAYLOR, DAVID C. M. (1982). An analysis of a thermal afferent pathway in the rat. *J. Physiol.* **326**, 319–328.

JAHNS, R. (1976). Differential projections of cutaneous thermal inputs to single units of mid-brain raphe nuclei. *Brain Res.* **101**, 355–361.

LIN, M. T., CHERN, Y. F. & CHERN, S. I. (1978). The effects of brain monoamine depletion on *p*-chlorophenyl-alanine-induced hypothermia. *Experientia* **34**, 1595–1596.

LIN, M. T., PANG, I. H., CHERN, S. I. & CHIA, W. Y. (1978). Changes in serotonin contents in brain affect metabolic heat production of rabbits in cold. *Am. J. Physiol.* **235**, R41–47.

LISTER, W. C. & WOODGET, L. L. (1972). Precision stereotaxic equipment. *J. Physiol.* **222**, 130–132*P*.

NAKAYAMA, T., ISHIKAWA, Y. & TSURUTANI, T. (1979). Projection of scrotal thermal afferents to the preoptic and hypothalamic neurones in the rat. *Pflügers Arch.* **380**, 59–64.

NECKER, R. & WEGNER, L. (1981). Effects of nucleus raphe lesions and *p*-CPA on temperature regulation and day-night rhythm of body temperature in pigeons. In *Advances in Physiological Sciences: Contributions to Thermal Physiology*, ed. SZELÉNI, Z. & SZÉKELY, M. pp. 29–31. Budapest: Académiai Kladó; Oxford: Pergamon Press.

NORRSELL, U. (1979). Thermosensory defects after cervical spinal cord lesions in the cat. *Exp. Brain Res.* **35**, 479–494.

PIERAU, Fr.-K., NEYA, T., YAMASATO, T. & ULRICH, J. (1980). Generation and processing of peripheral temperature signals in mammals. *Int. J. Biomet.* **24**, 243–252.

RANSON, S. W. & CLARK, S. L. (1947). *The Anatomy of the Nervous System, its Development and Function*, 8th edn. Philadelphia and London: W. B. Saunders Company.

SCHINGNITZ, G. & WERNER, J. (1980). Responses of thalamic neurones to thermal stimulation of the limb, scrotum and tongue in the rat. *J. therm. Biol.* **5**, 53–62.

TAYLOR, DAVID C. M. (1979). The effect of spinal cord lesions on the temperature-responsive neurones in the ventrobasal thalamus of the rat. *J. Physiol.* **292**, 21*P*.

TAYLOR, DAVID C. M. (1980). The effects of raphe magnus lesions on temperature-responsive neurones in the diencephalon of the rat. *J. Physiol.* **305**, 42P.

WAITES, G. M. H. (1976). Temperature regulation and fertility in male and female mammals. *Israel J. med Sci.* **12**, 982–993.

ZEMLAN, F. P., LEONARD, C. M., KOW, L.-M. & PFAFF, D. W. (1978). Ascending tracts of the lateral columns of the rat spinal cord: A study using the silver impregnation and horseradish peroxidase techniques. *Expl Neurol.* **62**, 298–334.