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VAGAL CONTROL OF THERMAL PANTING IN MAMMALS AND BIRDS

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SUMMARY

1. The role of the vagus nerves during normal respiration and thermal polypnoea was examined in four species of mammals and four species of birds by lung/air sac inflation and cervical vagotomy.

2. The Hering–Breuer inflation reflex could be elicited in all of the mammals during both normal respiration and panting; it was present in the fowl and pigeon at normal body temperature only.

3. Section of one vagus during hyperthermia reduced respiratory frequency in most animals and increased depth. Bilateral vagotomy had little further effect in the panting rabbit, lamb and pigeon, but abolished rapid respiration in the rat, guinea-pig, fowl, duck and quail.

4. Vagotomized lambs and pigeons started panting in response to increasing body temperature, although the rate attained was slower than in intact animals. Guinea-pig, rat, fowl and quail did not pant under these conditions.

5. Appropriate afferent vagal stimulation in the vagotomized fowl maintained normal respiration or thermal panting.

6. It is concluded that panting can, in some mammals and birds, be controlled by central mechanisms alone, while in others it depends on extrinsic stimuli mediated by way of the vagus nerves.

INTRODUCTION

Although pulmonary gas exchange is the primary function of the respiratory system, rapid ventilation of the non-respiratory surfaces represents an important heat-loss mechanism in many mammals and birds. Thus the occurrence of thermal polypnoea has been described, *inter alia*, in cats, dogs, sheep and cattle (Bligh, 1966) and in both wild and domestic birds (Salt, 1964). In both of these classes, the anterior hypothalamus and preoptic areas are known to be concerned in the co-ordination of the panting response (Magoun, Harrison, Brobeck & Ranson, 1938; Andersson,

Grant & Larsson, 1956; Akerman, Andersson, Fabricius & Svensson, 1960), which, in mammals, is generally thought to be mediated through the pneumotaxic centre in the anterior pons (Wang & Ngai, 1964). The contribution of extrinsic factors to the control of this type of respiration has, however, been relatively little studied, and since the functions of the pneumotaxic centre are considered by some authors (Wang & Ngai, 1964) as being in many ways analogous to those of vagal afferents, investigations of the role of the latter in the panting of different species would seem important.

Following the early work of Richet (1898), Anrep & Hammouda (1932) showed with dogs that not only did double vagotomy fail to affect panting but also that the Hering–Breuer inflation reflex was absent when panting had become fully established. Hiestand & Randall (1942), working with the rabbit and fowl, concluded that the vagi are unnecessary for mammalian panting but that at least one intact vagus is needed for panting in birds. Saalfeld (1936) had shown, however, that bilateral vagotomy does not affect thermal polypnoea in the pigeon, and this was later confirmed by Sinha (1959). The present work was designed as a first step towards further elucidation of the mechanisms governing polypnoeic breathing, with the immediate purpose of clarifying the role of vagal afferents in the panting response of selected mammals and birds.

METHODS

Acute experiments were performed on the following animals: five laboratory-bred guinea-pigs (body weight, 340–590 g), three rats (random bred strains, Ash/CSE, 280–300 g), two Kent lambs (6 and 10 days old, 2·5 and 5·2 kg), one laboratory-bred rabbit (3·5 kg), sixteen White Leghorn-type domestic fowls (*Gallus domesticus*, 0·8–1·8 kg), eight pigeons (*Columba livia*, 290–460 g), five Japanese quail (*Coturnix coturnix japonica*, 100–130 g), and two Aylesbury ducks (*Anas domesticus*, 2·8 and 3·7 kg).

The mammals were anaesthetized with pentobarbitone sodium (35–40 mg/kg body wt., intraperitoneally) and the birds with phenobarbitone sodium (175–200 mg/kg intramuscularly). Panting was facilitated by inserting a tracheal cannula as low as convenient in the neck (but avoiding damage to the cervical air sacs of the birds). Occasionally tracheal secretions caused blockage of the cannula, and in these cases atropine sulphate (2 mg/kg) was given intramuscularly. Respiratory movements were recorded on a kymograph by means of a Sherrington stethograph constructed from a Brodie Universal Lever (Palmer, Ltd.). Frequency of respiration (f, in intact animals; f_v after bilateral vagotomy) was determined from the trace by counting, and a qualitative estimation of changes in depth of breathing obtained from the amplitude of the record. Inflation of the lungs/air sacs was performed in all species except quail with a simple pressure bottle connected by a three-way valve to the tracheal cannula. A mercury manometer was calibrated to indicate drops in pressure which corresponded to removals of 5 ml. aliquots of air from the bottle. It was the purpose of these experiments to make only qualitative observations on the presence or absence of the Hering–Breuer inflation reflex and no attempt was made to measure intra-tracheal or eosophageal pressure. In all cases the smallest air volume which gave a consistent effect was used during normothermia, and several larger volumes were used during thermal panting.

The vagi were carefully prepared with loose ligatures and section performed by scissors with the smallest mechanical disturbance possible. Deep-body temperatures were taken throughout the experiments in the rectum (T_r) or cloaca (T_c) using a mercury-in-glass thermometer calibrated in $\frac{1}{5}$° C and inserted to a depth of 8 cm in the larger animals, and 4 cm in rat, guinea-pig and quail. The increase in body temperature generally required to initiate polypnoea in anaesthetized animals was induced by means of infra-red irradiation (250 W lamp). During the heating of intact animals, T_r 42·0° C and T_c 46·0° C were not exceeded in an attempt to ensure recovery, so that the same animal could be used for studies with progressive hyperthermia after vagotomy. In several cases respiratory frequency was still increasing at these temperatures (Figs. 2 and 3).

Electrical stimulation of the vagi was performed in the fowl using platinum electrodes and a Palmer Stimulator (H. 47) giving positive-going rectangular pulses of 1 msec duration.

Most experiments consisted of the following stages: (i) testing for the presence of the Hering–Breuer inflation reflex in the intact, normothermic animal; (ii) heating to T_r or T_c some 3–4° C higher than normal, by which time panting, or a much enhanced f had been established; (iii) re-testing for the inflation reflex in the intact hyperthermic animal; (iv) observing the effect of unilateral cervical vagatomy (both right and left vagi were examined in this respect) during near maximum f; (v) after establishment of a rhythmic and consistent respiratory pattern, observing the effect of section of the second vagus; (vi) examining the effect of lung/air sac inflation in the bilaterally vagotomized, hyperthermic preparation; (vii) cooling to the original control temperature (T_r 37–38° C; T_c 41–42° C); (viii) confirming the abolition of the inflation reflex by bilateral vagotomy in the normothermic animal; (ix) re-heating of the vagotomized preparations to T_r or T_c some 4–5° C above normal to observe the part played by the vagi in the respiratory response to progressive hyperthermia.

In addition, further experiments were performed: (i) with fowls and pigeons lying supine, on their left sides, or supported in the normal, upright position by a wire through the fused dorsal spinous process of the synsacrum, to examine the effect of posture on the results in birds (King & Payne, 1964); (ii) on fowls and pigeons vagotomized before initial heating to compare the results with those from the more severely stressed preparations which had already undergone progressive hyperthermia, vagotomy and subsequent cooling.

RESULTS

For the purpose of these experiments thermal polypnoea was defined simply as 'the rapid, shallow respiratory movements observed during heat exposure' and no attempt was made to correlate respiratory frequency with rectal or cloacal temperature. In all animals there occurred certain characteristic accessory movements, namely, those of the nostrils in mammals and of the hyoid apparatus ('gular flutter') in birds, which, in these species, appeared synchronized with the movements of the chest wall. Tracheotomy had no effect on these motor responses, although the 'sham panting' which resulted was often followed by a more copious flow of saliva, possibly owing to the absence of moving air in the buccal cavity.

Results of lung/air sac inflation. Figure 1 illustrates some of the effects of lung and air sac inflation during normal respiration and thermal polypnoea. Typical Hering–Breuer inflation reflexes could be elicited in all four species of mammals, both at control body temperature and during hyperthermia. The effect of lung inflation (using volumes of 10–30 ml.) in the

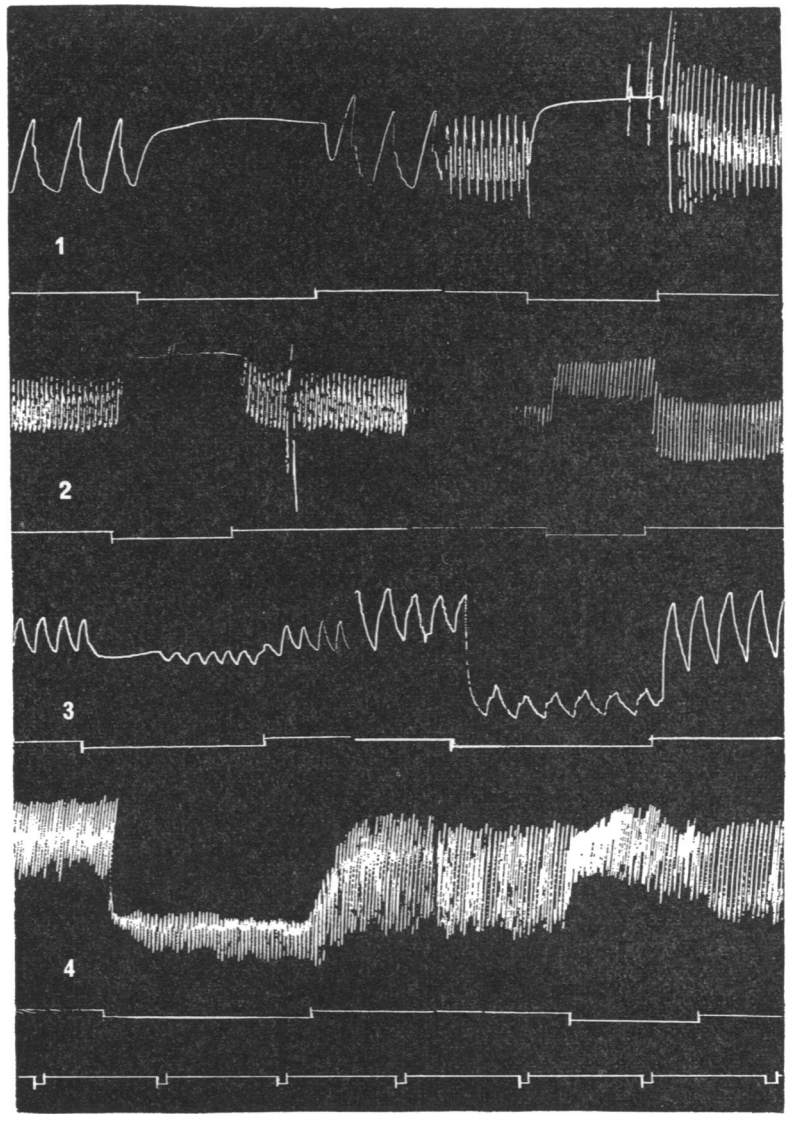

Fig. 1. Effects of lung/air sac inflation. (1) Intact rabbit; inflation with 30 ml. air during normal respiration (left) and thermal polypnoea (right). (2) Lamb; inflation with 40 ml. during panting when intact, and with 60 ml. after unilateral vagotomy. (3) Pigeon; inflation with 15 ml. during normal respiration when intact, and with 30 ml. after bilateral vagotomy. (4) Intact pigeon; inflation with 30 ml. during panting caused reduction in frequency in one bird; 15 ml. caused acceleration in another. Time marker, 5 sec. There is no special significance in the direction of displacement of the records. The arrangement of levers was such that inflation caused elevation of the trace in supine animals (all mammals and pigeon at (4), right), depression in upright animals (pigeons at (3), and (4), left).

guinea-pig and rat did not change, irrespective of the values of T_r or f, but in both rabbit and lamb a given minimum effective volume (25–35 ml.) which consistently produced apnoea during normal breathing, was often less effective during thermal polypnoea. Thus, in the rabbit, 30 ml. air caused apnoea which was indefinitely prolonged (at least 40 sec) at 37·0° C, but during panting at T_r 39·8° C the same volume inhibited respiratory efforts for only 3–5 sec, after which an intermittent gasping pattern set in. Greater inflation volumes prolonged the period of apnoea, but even 70 ml. did not cause a long-lasting cessation of respiration once panting had become fully established. Double vagotomy abolished the inflation reflex in all mammals, while in one lamb, section of the left vagus alone appeared to suffice.

In the normothermic pigeon and fowl genuine reflexes were demonstrated, although these took the form of a pause in respiratory movements lasting from 2 to 5 sec with inflation volumes (15–25 ml. and 20–35 ml. respectively) rather larger than those required in mammals of similar body weight. Prolonged apnoea was never produced in birds and there was no evidence of functional reflexes during panting (maximum inflation volumes 50 ml. for pigeons, 120 ml. for fowls), except in two pigeons where some reduction in frequency occurred during inflation with 30 ml. or more. One further pigeon always accelerated its respiratory frequency during inflation (Fig. 1), but this effect unlike the genuine inflation reflexes observed, was not abolished by vagotomy.

No inflation reflexes could be elicited in the duck under any conditions, using volumes from 30 ml. to as high as 200 ml.

Effects of vagotomy on normal breathing. These results include two sets of observations. The first (indirect evidence), from experiments performed on all species except rabbit and duck, involved comparison between the type of respiration seen during a preliminary control period and that observed when normal body temperature had been regained following double vagotomy during hyperthermia. These observations were made while studying the influence of vagotomy on the respiratory response to hyperthermia, and this section of the results is therefore incorporated in Table 2. Direct evidence was also obtained in fowls and pigeons where double vagotomy was performed before initial exposure to heat.

Bilateral vagotomy produced in all animals the well known response of decreased rate and increased depth of breathing. These effects were especially severe in guinea-pig, fowl and quail, while in pigeon there was often little or no detectable alteration in the amplitude of respiratory movements. These results were confirmed by the direct observations.

Effects of vagotomy during hyperthermia. Table 1 presents a summary of experiments in which vagotomy was performed during fully established

thermal polypnoea. Section of either vagus generally resulted in a small reduction in frequency of breathing movements and typically a temporary increase in depth. The pigeons were exceptional in that this procedure was followed by a reduction in amplitude which was maintained indefinitely. An acceleration of breathing rate occurred in the rabbit.

Section of the second vagus resulted in immediate elimination of panting in guinea-pig, fowl, quail and duck. A substantial increase in depth usually occurred concurrently with abrupt cessation of rapid respiratory movements. Each of the ducks died within minutes of double vagotomy during

TABLE 1. Changes in respiratory frequency in relation to vagotomy during hyperthermia. Mean values, with range and percentage change in parentheses

			Respiratory frequency (breaths/min)				
Species	No. of animals	Body temp. (°C) T_r or T_c	Both vagi intact (I)	One vagus severed (II)	Change from (I) to (II)	Both vagi severed (III)	Change from (I) to (III)
Rat	3	40·1 (39·8–40·4)	152 (145–162)	112 (95–134)	−40 (26%)	52 (50–58)	−100 (66%)
Guinea-pig	4	41·0 (40·0–42·0)	161 (145–170)	152 (120–170)	−9 (5·6%)	32 (26–45)	−129 (80%)
Rabbit	1	39·0	372	432	+60 (16%)	436	+64 (17%)
Lamb	2	41·2 (40·4–42·0)	235 (195–275)	238 (195–280)	+3 (1·3%)	233 (191–275)	−2 (0·9%)
Fowl	5	44·2 (44·0–44·8)	159 (140–216)	141 (125–198)	−18 (11%)	7 (4–12)	−152 (96%)
Quail	3	45·3 (45·0–46·0)	542 (500–610)	534 (500–585)	−8 (1·5%)	20 (15–25)	−522 (96%)
Duck	2	44·0	147 (140–155)	119 (108–130)	−28 (19%)	4*	−143 (97%)
Pigeon	6	45·1 (44·4–45·4)	495 (380–576)	439 (380–535)	−20 (4·4%)	405 (362–515)	−54 (12%)

* One duck died before measurement of f_v was made.

hyperthermia, and the other three species exhibited severe respiratory distress, often with dysrhythmia and gasping. Cooling of these animals to normal body temperature caused a further decline in f_v and increase in amplitude. Two vagotomized guinea-pigs died soon after cooling to T_r 38·0° C.

Bilateral vagotomy in the hyperthermic rat also substantially reduced the rate of breathing and enhanced the depth. The result, however, was intermediate between the profound collapse seen in the above four species and the effect in lamb, rabbit and pigeon where there was little reduction in rate of panting. Lamb and rabbit exhibited a considerable increase in depth of breathing, but pigeons were again exceptional in showing a reduced amplitude after vagotomy, and this was maintained for the period of observation (about 30 min).

TABLE 2. Effect of hyperthermia on respiratory frequency before and after vagotomy. Mean values, with range and percentage change in parentheses. Frequencies during hyperthermia represent maximum attained at body temperature 3–4° C above normal

		Both vagi intact Respiratory frequency (breaths/min)				After bilateral vagotomy Respiratory frequency (breaths/min)				
Species	No. of animals	During normothermia (I)	During hyperthermia (II)	Change from (I) to (II)	No. of animals	During normothermia (III)	Change from (I) to (III)	During hyperthermia (IV)	Change from (I) to (IV)	Change from (III) to (IV)
Rat	3	84	152	+68	3	41	−43	54	−30	+13
		(78–96)	(130–166)	(81%)		(36–46)	(51%)	(50–58)	(36%)	(32%)
Guinea-pig	4	48	168	+120	2	10	−38	20	−28	+10
		(40–53)	(147–176)	(250%)		(7–13)	(79%)	(18–22)	(58%)	(100%)
Rabbit	1	32	455*	+423	0	—	—	—	—	—
		—	—	(1,322%)		—	—	—	—	—
Lamb	2	36	235	+199	2	18	−18	234	+198	+216
		(33–39)	(195–275)	(553%)		(15–21)	(50%)	(200–268)	(550%)	(1,200%)
Fowl	8	34	164	+130	6	5	−29	9	−25	+4
		(29–41)	(149–188)	(382%)		(3–8)	(85%)	(7–13)	(74%)	(80%)
Quail	4	56	542	+486	3	6	−50	17	−39	+11
		(49–60)	(500–610)	(868%)		(4–9)	(89%)	(14–22)	(70%)	(183%)
Duck	2	38	147	+109	0	—	—	—	—	—
		(36–40)	(140–155)	(287%)		—	—	—	—	—
Pigeon	7	85	500	+415	5	46	−39	365	+280	+319
		(70–88)	(413–576)	(488%)		(42–50)	(46%)	(323–501)	(329%)	(693%)

* Rabbit's body temperature was 39·2° C.

Cooling of these last three species resulted in a rather abrupt cessation of panting at a temperature 0·6–1·4° C higher than that at which this occurred in the same animals with intact vagi.

Effects of progressive hyperthermia after vagotomy. Table 2 summarizes the changes in respiratory frequency during increasing body temperature, both with intact vagi and after bilateral vagotomy. The influence of vagotomy supported the results of the previous section and the contrast

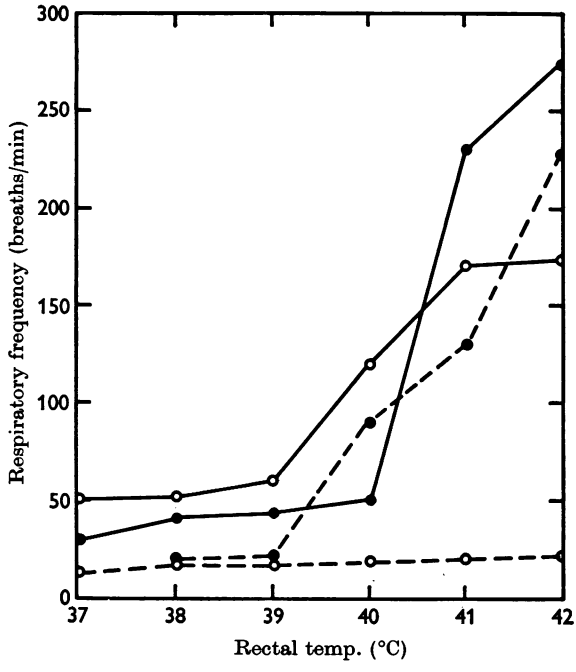

Fig. 2. Changes in respiratory frequency in lamb and guinea-pig during progressive hyperthermia, before and after bilateral vagotomy. (One representative of each species.) ●——● intact lamb. ● - - - ● vagotomized lamb. ○——○ intact guinea-pig. ○ - - - ○ vagotomized guinea-pig.

between species is illustrated in Figs. 2 and 3. A profound influence was seen in guinea-pig, fowl and quail where the slow breathing rate of the bilaterally vagotomized preparations increased relatively little, even after a 4–5° C increase in body temperature, compared to that which occurred with vagi intact. One fowl (No. 4), however, was exceptional in exhibiting a sudden increase in f_v from 10/min to 85/min as T_c rose from 44 to 45° C. Results from this bird are not included in Table 2.

The respiratory response to heat in the lamb appeared little affected by vagotomy, while in the pigeon, although the maximum values for f_v were lower than those for f, panting, nevertheless, occurred at over 300/min in

all specimens examined. Again the rat showed a response intermediate between the two extremes of complete dependence and complete independence of thermal panting on afferent vagal drive.

In no animal did unilateral vagotomy have any lasting effect on this response.

Effects of posture and experimental stress. When experiments were conducted in the three positions described above, no effects attributable to body posture were observed in birds.

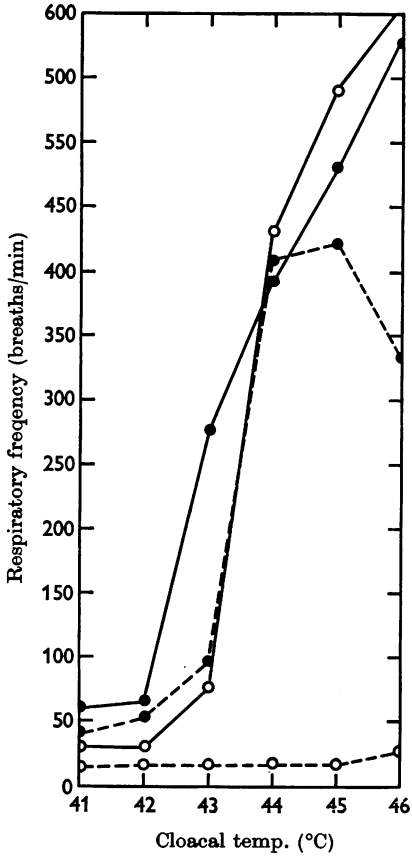

Fig. 3. Changes in respiratory frequency in pigeon and quail during progressive hyperthermia, before and after bilateral vagotomy. (One representative of each species.) ●——● intact pigeon. ● - - - ● vagotomized pigeon. ○——○ intact quail. ○ - - - ○ vagotomized quail.

Birds vagotomized before initial heating showed no marked differences in respiratory response to increasing T_c from those which had survived the full experimental procedure.

Electrical stimulation of the vagus in the fowl. Following the surprising results described in fowl No. 4, the role of the vagi in this species was further investigated by means of electrical stimulation of the central stump of the right vagus. A weak stimulus (2–3 V; 10 c/s) was found to protect the fowl against the severe respiratory collapse which normally followed double vagotomy, to support a respiratory frequency of 20–30/min at T_c 41–42° C, and to permit the initiation and maintenance of thermal panting in such preparations in response to increasing body temperature. Panting continued at a somewhat reduced rate in two specimens after the stimulus was then switched off, but in three others f_v fell immediately to between 6 and 11/min.

DISCUSSION

Inevitably, there is some difficulty in distinguishing between merely rapid breathing and the genuine thermal polypnoea defined by Richet (1898) simply as a facilitated form of shallow and fast respiration. Lim & Grodins (1955) chose to make their distinction at a respiratory frequency of 100/min in dogs and during the present experiments it was recognized that an arbitrary division of this sort would have to suffice, especially in a species like the rat which did not pant at a conspicuously rapid rate compared with control values, and where peripheral vasodilatation (Thompson & Stevenson, 1965) and salivary evaporation (Hainsworth, 1968) are the normal means of heat loss. Nevertheless, it was felt that an investigation of the role of the vagi during the rapid breathing of heat exposure would itself be valuable and would serve to underline the differences observed between various species.

Vagal reflexes at normal body temperature. There have been many studies of Hering–Breuer inflation effects in mammals and the present work agrees with Widdicombe (1961) in finding strong reflexes in the rat, guinea-pig and rabbit, as well as in the lamb. The presence of such reflexes in birds has been disputed since Graham (1940) purported to demonstrate them in modified form in the fowl, where he considered their function to be chiefly that of promoting expiration. Sinha (1958) described similar effects in the pigeon, and the present results confirm these findings although it was not found possible to elicit definite inflation reflexes in the duck. Even with the fowl and pigeon, however, the respiratory arrest never exceeded a few seconds' duration, as Blankart (1960) also found after tracheal occlusion studies in the pigeon. Comparison with mammals is difficult without quantitative data on tidal volume or pressure swing, but the reflex inhibition does appear relatively weak in the fowl where inflation volumes were employed equal to the maximum inspired at each breath during forced breathing, or more than twice the volume of the lungs

(King & Payne, 1962). The unique anatomy of the avian respiratory system has led to several conflicting views of function (Salt & Zeuthen, 1960) and it has not been shown that normal volume changes in the bird's lung are sufficient to stimulate any stretch receptors that may be postulated. Results with the pigeon, fowl and duck appear to support the evidence of Fedde, Burger & Kitchell (1963) for the involvement of additional peripheral receptors, also innervated by branches of the thoracic vagus, in the maintenance of co-ordinated respiratory movements.

The effect of vagotomy on normal respiration was in most cases to reduce the rate and increase the depth of breathing movements. Oberholzer & Schlegel (1957) found vagal drive to be indispensable for the maintenance of respiration in the guinea-pig, and during the present experiments also, this species resembled the fowl and quail in exhibiting severe respiratory depression after section of both vagi. The pigeon survived vagotomy well and was the exception among all the species in that it frequently showed no change in amplitude of breathing movements after vagotomy. Otherwise, however, these findings support the widely held view that one of the vagal reflexes is concerned with inhibition of maximal inspiration which results in an increase of breathing rate by permitting expiration to occur earlier.

Vagal reflexes during hyperthermia. In each of the mammals the inflation reflex appeared intact during thermal polypnoea, but it was weaker in the hyperthermic rabbit and the lamb than at normal body temperature, as indicated by the duration of apnoea with any given inflation volume. There is the possibility, therefore, that had the reflex been tested in these species at still higher body temperatures and at maximum panting frequency, it would have been absent, as described by Hammouda (1933) in the dog. He also reported in several experiments an acceleration in respiratory frequency rather than an inhibition as a result of inflation during thermal polypnoea, an effect seen repeatedly in one pigeon and which, in contrast to the genuine Hering–Breuer reflexes, was not abolished by vagotomy. Generally, however, inflation during hyperthermia in birds had no detectable effect in either direction on panting frequency.

Vagotomy during thermal polypnoea revealed important species differences among both mammals and birds. Using the dog (Richet, 1898; Anrep & Hammouda, 1932) and cat (Uyeno, 1923), previous workers have shown that panting continued after section of the cervical vagi and this proved also to be the case in the rabbit and the lamb; in the hyperthermic rabbit there was actually an acceleration of rate following vagotomy, a phenomenon also described by Anrep & Hammouda (1932). In contrast to these four species, vagotomy in the hyperthermic rat and guinea-pig caused a profound reduction in breathing rate and in the latter

severe respiratory distress, a difference that was especially striking for species relatively closely related to the rabbit. Perhaps the difference in function here reflects a difference in normal activity, for the rabbit, like the dog is well adapted for sustained muscular effort which would be a large internal contribution to body heat; this possibly contrasts with the rat and guinea-pig which by characteristic habit might be expected to avoid rather than tolerate both extremes of ambient temperature and muscular exertion.

The demonstration that thermal polypnoea can be initiated and maintained in the vagotomized fowl by electrical stimulation suggests some form of mobilization of central mechanisms during hyperthermia which, in contrast to those of the pigeon, do not apparently function in the absence of afferent vagal drive. In the pigeon, the 'panting centre' described by Saalfeld (1936) in the anterior dorsal mid-brain must, in association with impulses from the anterior hypothalamus (Akerman *et al.* 1960), be conceived as capable of controlling polypnoea by way of the medullary respiratory centres without essential contributions from receptors in the thorax.

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