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CANINE OESOPHAGEAL MECHANORECEPTORS

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SUMMARY

1. The properties of slowly adapting oesophageal mechanoreceptors were determined in anaesthetized dogs in which the oesophagus had been isolated surgically. Changes in oesophageal pressure resulted in reproducible changes in oesophageal volume.

2. Action potentials were recorded from thirty-three oesophageal afferent nerve fibres in the cervical vagus. All the receptors were located in the thoracic oesophagus. The conduction velocities of the afferent fibres ranged from 9·3 to 27·7 m/s (mean, 17·2; S.D., 4·1). The receptors were of the 'in series' type.

3. In the pressure range of 0–1 mmHg, all units were tonically active with irregular firing rates that ranged from 0·2 to 13·0 Hz. Lung inflation altered the discharge rate in a variable manner depending upon the degree of distension of the oesophagus.

4. The adapted discharge rate of eight units reached a maximum of 23–44 Hz (mean, 35; S.D., 8) at an oesophageal pressure of 8–15 mmHg (mean, 11; S.D., 3). The relationship between pressure and discharge rate was linear over a narrow pressure range.

5. Ramps of similar gradient produced higher discharge rates in units whose afferent fibres had higher conduction velocities. In some units a large increase in firing rate occurred over a narrow pressure range and became more pronounced with ramps of increasing gradient. This increase in firing rate was called a 'burst'. Six out of thirty-three units showed a 'burst' response. The conduction velocities of these six units ranged from 18·7 to 23·5 m/s.

6. Slowly adapting oesophageal mechanoreceptors could be subdivided functionally into two types. Their discharge pattern was dominated by a narrow response range. These properties may be significant in an organ that is normally empty and has a low residual volume.

INTRODUCTION

The dog is the major species besides man that suffers a naturally occurring disorder of oesophageal motility. The canine motility disorder, megaoesophagus, has some features in common with the human disorder achalasia and both can cause significant morbidity. The aim of the present study was to describe the characteristics of oesophageal mechanoreceptors in a species prone to a motility disorder.

A prerequisite of any study which describes oesophageal mechanoreceptor function is a reproducible means of controlling oesophageal dimensions. Mechanoreceptor

function has been examined in the cat and the sheep using a balloon to produce oesophageal distension (Mei, 1970; Falempin, Mei & Rousseau, 1978). An isolated segment of the lower thoracic oesophagus has been used in a similar study in the ferret (Andrews & Lang, 1982). In the dog, surgical isolation of the whole oesophagus allowed pressure to be controlled and volume to be measured. The responses of slowly adapting mechanoreceptors to reproducible deformations of the oesophagus were determined. A preliminary report on this work has been presented (Satchell, 1982).

METHODS

Animal preparation

Thirteen greyhounds weighing 19–32 kg were anaesthetized with sodium pentobarbitone at a dose of 30 mg/kg. The animals were intubated and ventilated with a Bird Mark 8 respirator using a 50 % oxygen–air mixture. Systemic blood pressure was measured with a right carotid artery cannula

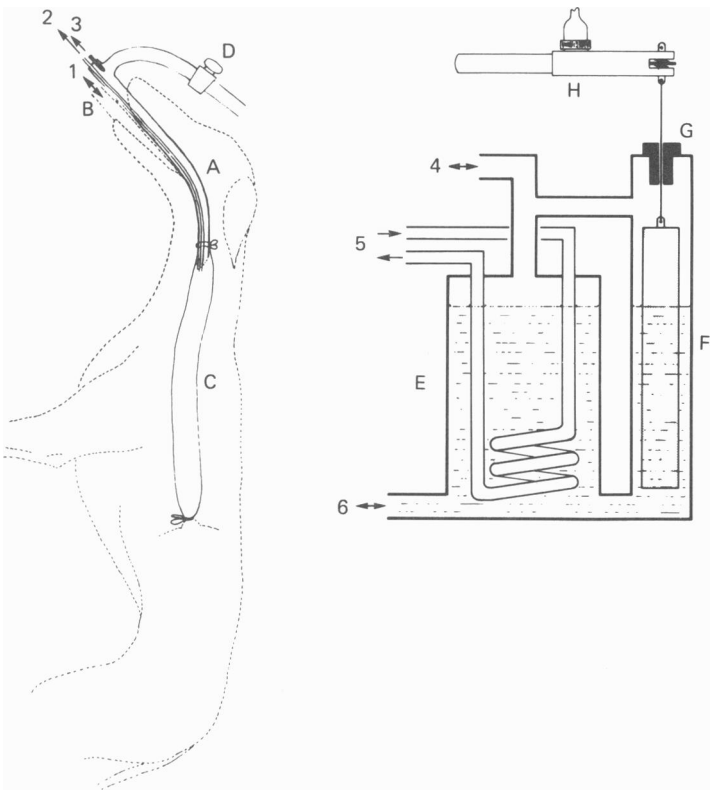

Fig. 1. Apparatus used to control oesophageal dimensions and measure oesophageal volume. Surgical isolation of the oesophagus (C) in a dog lying in the right lateral position. The oesophageal access tube (A) is connected via a tap (D) to the reservoir bottle. The animal is intubated (B). The reservoir bottle (E) is connected in parallel with the Perspex cylinder in which the uniform aluminium rod is suspended (F). The rod is supported by a stainless-steel wire passing through a Teflon plug (G); the wire is attached to a strain gauge (H). 1, 50 % oxygen–air mixture; 2, oesophageal pressure; 3, access tube temperature; 4, controlled pressure source; 5, warm water; 6, oesophageal perfusion fluid.

(Sanborn 267B Pressure Transducer) and rectal temperature was monitored (Grant Thermistor Thermometer). Tracheal air flow was measured with a pneumotachograph (Mercury Electronics) and the efficiency of ventilation was checked with a Radiometer ABL2 (Acid Base Laboratory).

Oesophageal isolation

The cranial end of the oesophagus was approached through a mid-line neck incision and the right lateral wall of the oesophagus was exposed without disturbing the vagus nerve, its branches or blood vessels. Gentle blunt dissection allowed spaces to be formed anterior and posterior to the upper oesophagus. After an identical exposure of the left side a binding tape was passed around the oesophagus but not tied. A left thoracotomy between the eighth and ninth ribs allowed binding tape to be passed around the supradiaphragmatic oesophagus without including the dorsal or ventral vagal trunks. The lower oesophagus was tied off 1 cm caudal to the origin of the dorsal vagal trunk (2–3 cm cranial to the diaphragmatic insertion). An oesophageal access tube, made from a no. 11 Portex endotracheal tube, was passed into the upper oesophagus through the mouth and the binding tape tightly tied (Fig. 1). The oesophagus was washed and connected to a perfusion bottle filled with warmed Hartman solution. The bottle was pressurized with oxygen, and any pressure level between 0 and 60 mmHg could be maintained for controlled periods of time. Step pressure changes and ramp pressure changes could also be produced. The temperature of the perfusion fluid was measured and controlled with a heating coil. Oesophageal pressure was measured with a catheter whose tip lay in the distal opening of the access tube (Sanborn 267B Pressure Transducer). The reference point for zero pressure was the mid line of the cervical oesophagus with the dog in the right lateral position. The volume of fluid moving into and out of the oesophagus was measured indirectly by monitoring the level of fluid in the perfusion bottle with a fluid level detector (Fig. 1). Changes in the fluid level altered the buoyancy of an aluminium rod and this was detected as a change in weight by a force displacement transducer (Grass FT10C).

Nerve preparation and recording

Stimulating electrodes were placed on the right and left vagal trunks 3–4 cm above the upper sternal margin. Both sets of electrodes were connected to stimulators (Digitimer Isolated Stimulator: Model DS2) which delivered 0·1 ms square-wave pulses and whose rate was controlled (Frederick Haer Co.: Pulsar 6i). Single-fibre and few-fibre strands of the left vagal trunk at the level of the carotid bifurcation were prepared on a black Perspex platform using a dissecting microscope and jewellers' forceps. The mineral oil pool was heated by a copper heat exchanger and was oxygenated. Nerve activity was recorded with silver–silver chloride electrodes, amplified and stored on magnetic tape (Electrodata 6400 FM Tape Recorder). The band width of the recording system was 5 Hz–10 kHz (−3 dB). Oesophageal pressure, volume and tracheal air flow were also recorded on magnetic tape. These variables as well as blood pressure and heart rate were continuously displayed on a Grass Model 7 Polygraph.

Experimental procedures and analysis

The conduction velocities of the afferent nerve fibres were measured by the techniques of Paintal (1953) and Iggo (1958). The receptor site was located by digital manipulation of the thoracic oesophagus. Discharge rate was determined by replaying the recording into a digital window (PDP 11/40 computer). The accuracy of the window was checked by replaying all recordings at slow speed and was greater than 90 %. The instantaneous discharge frequency was determined from the previous interspike interval; the dynamic range of the instantaneous frequency device was 2–300 Hz (the highest frequency encountered was about 100 Hz). The instantaneous rate was filtered to give mean discharge frequency (half-amplitude point, 3 Hz). The steady-state characteristics were determined by allowing oesophageal pressure and volume to become constant at a number of different levels and recording nerve activity for two respiratory cycles. Oesophageal volume became constant 15–40 s after an increase in pressure. The firing rate, 60–75 s after a pressure increase, was 103–115 % of the firing rate after 2 min and was used as a measure of the adapted firing rate.

Means were expressed with standard deviations which were all corrected for small numbers. When comparisons were made, a non-parametric statistical method (Mann–Whitney test) was used and a value of $P < 0.05$ was accepted as indicating significance.

RESULTS

Oesophageal isolation

Isolation did not alter the contractile properties of the oesophageal musculature. Pressure changes measured with a balloon placed in the mid-thoracic oesophagus were not altered when the upper tie was tightened around the access tube (Fig. 2). The oesophageal electromyogram was also unaffected. When oesophageal volume was

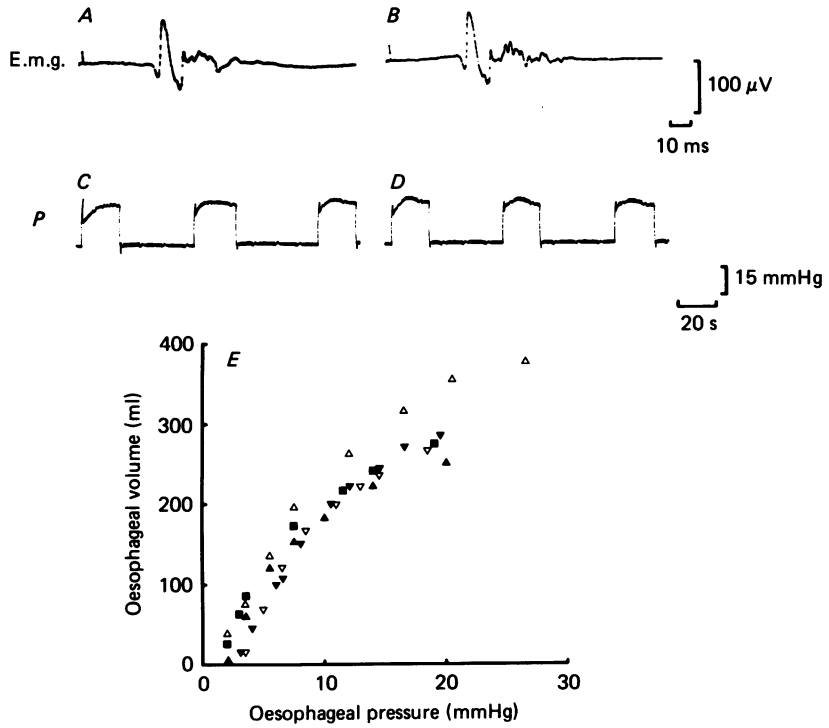

Fig. 2. Isolation produced no demonstrable change in oesophageal motor performance. *A* and *B*, bipolar electromyogram recording in the lower thoracic oesophagus before and after tightening the upper oesophageal tie. The response to supramaximal stimulation of the vagus (20 V; 0·1 ms; 20 Hz) was not changed. *C* and *D*, oesophageal pressure recorded by balloon before and after tightening the upper tie. The pressure change to supramaximal stimulation of the vagus (5 V; 0·2 ms; 20 Hz; 200 pulses) was not altered. In *E* there was only a small variation in oesophageal compliance in five different animals.

increased by a large amount (300 ml), there were small changes in central venous pressure (−2 mmHg), systemic pressure (−5 mmHg) and heart rate (< 2 beats/min).

Oesophageal compliance, measured in five animals under steady-state conditions, had a mean value of 32 ml/mmHg at a pressure of 3 mmHg and this decreased at higher pressures (Fig. 2). At any level of constant pressure, oesophageal volume varied by less than 20 ml over a period of 8 h in a particular animal. The maximum rate of volume change that could be achieved was 17 ml/s which was produced by pressure gradients of greater than 1·3 mmHg/s.

Oesophageal afferent fibre discharge

There were forty-nine afferent units that responded with sustained nerve activity to oesophageal distension (Fig. 3). The receptor sites of thirty-three units were located and only the characteristics of these units are described. When receptors were being located the oesophagus was unpressurized and it was useful to group the receptor sites into four zones. There were fourteen receptors in the lower thoracic oesophagus caudal

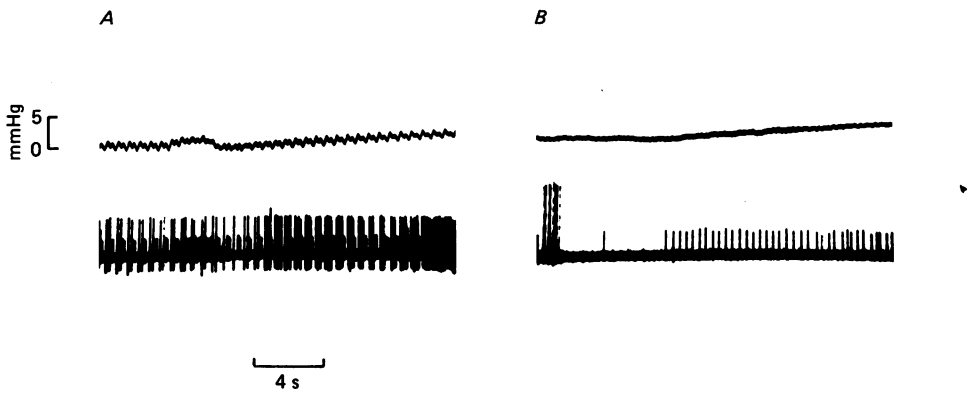

Fig. 3. Impulses in afferent vagal nerve fibres from oesophageal mechanoreceptors. Upper trace, oesophageal pressure; lower trace, afferent nerve discharge. In *A* the oesophageal unit (largest impulse) discharged in time with the cardiac pulsation visible in the pressure trace. Respiration produced a small increase in oesophageal pressure which resulted in an increase and then decrease in the firing rate of the oesophageal unit. The firing frequency increased in each burst after the pressure ramp started until it became continuous. In *B* the oesophageal unit (smaller impulse) discharged at a low frequency when oesophageal pressure was in the range of 0–1 mmHg. After the start of the pressure ramp the unit discharged regularly.

to the division of the left vagus into the ventral vagus and the 'crossing branch'. In the oesophagus adjacent to the lung hilum, adjacent to the aortic arch and cranial to the arch, there were seven, eight and four receptors respectively. Receptors were located in the oesophageal wall in the aortic arch region by gently separating the aorta and oesophagus and retracting the arch.

Spontaneous activity was present in all units in the pressure range of 0–1 mmHg. Four of thirty-three units discharged with a regular rhythm; these four units were located in the lower three zones and the rhythm was that of the arterial pulse. The spontaneous impulse frequency of all units during apnoea ranged from 0·2 to 13·0 Hz (mean, 2·7; S.D., 2·9). In the pressure range of 0–1 mmHg lung inflation produced variable changes in discharge rate. In twenty-six units, eleven increased, nine decreased and six did not alter their firing rate with lung inflation, which itself increased oesophageal pressure by 0–2 mmHg. In the pressure range of 8–10 mmHg the discharge rate of nine of twenty-four units decreased with lung inflation while an equal number showed no change.

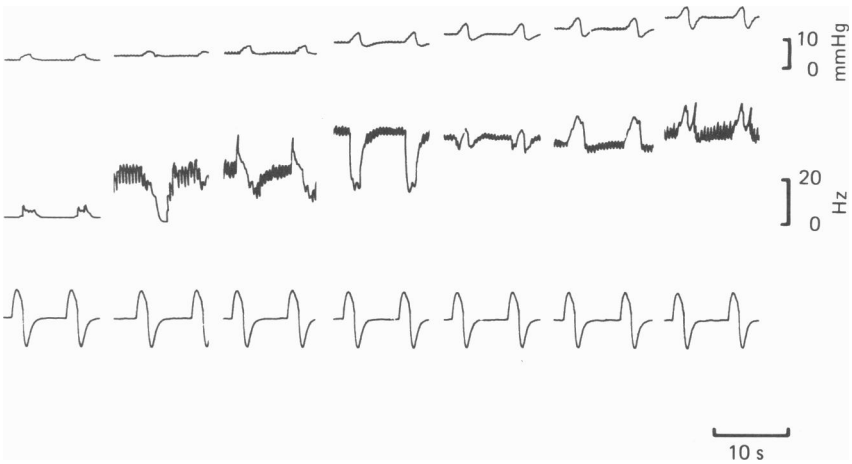

Fig. 4. Effect of increasing pressure on the discharge frequency of an oesophageal unit and the changes produced by lung inflation. Upper record: oesophageal pressure. Middle record: discharge rate of a unit located in the oesophageal wall close to the division of the left vagus into the 'crossing branch' and the ventral vagus. Lower record: tracheal air flow. The mean discharge frequency reached a maximum at an oesophageal pressure of 8 mmHg. Fluctuations in the discharge pattern due to lung inflation were prominent and varied in character as oesophageal pressure increased. Another secondary rhythm had the same frequency as the ripple on the pressure trace (2·2 Hz). The heart rate was 135 beats/min.

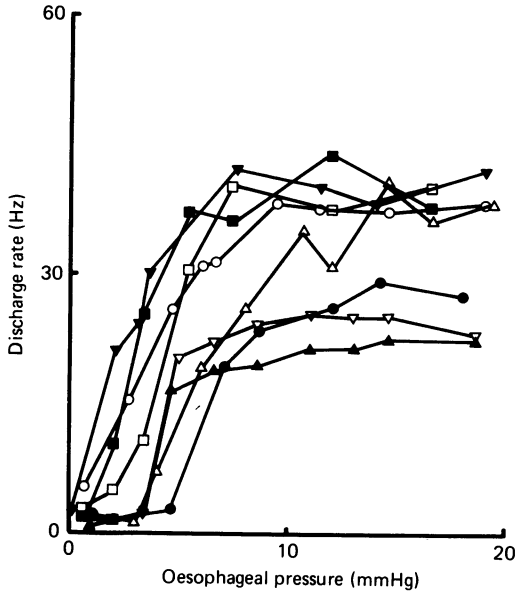

Fig. 5. The graph shows the mean adapted discharge rate of eight oesophageal units when pressure was held constant at different levels.

TABLE 1. Discharge characteristics of eight slowly adapting oesophageal mechanoreceptors under steady-state conditions

	Pressure at maximum firing rate (mmHg)	Maximum firing rate (Hz)	Maximum pressure (mmHg)	Firing rate at maximum pressure (Hz)	Volume change at maximum firing rate (ml)	Maximum volume change (ml)	'Linear' pressure range (mmHg)	'Linear' volume range (ml)
U2	9·5	38	19·0	38	—	—	0·5–6·5	—
U5	7·5	40	16·5	40	195	315	2·0–7·5	195
U6	12·0	44	16·5	38	265	315	0·5–5·5	265
U8	14·5	41	19·5	39	245	285	3·0–10·5	200
U10	14·5	29	18·0	28	—	—	4·5–8·5	—
U12	14·5	23	18·5	23	235	265	3·0–5·0	55
U13	11·0	26	18·5	23	200	265	3·5–5·0	55
U24	7·5	42	19	42	175	270	0–7·5	150
Mean	11	35	18	33	219	286	—	—
S.D.	3	8	1	8	34	24	—	—

Steady-state responses

Lung inflation produced variable changes in discharge rate at different levels of constant oesophageal pressure (Fig. 4). Rhythms related to the arterial pulse were observed in units located near and distant to the aorta. The relationship between the adapted discharge rate and oesophageal pressure for eight units is shown in Fig. 5.

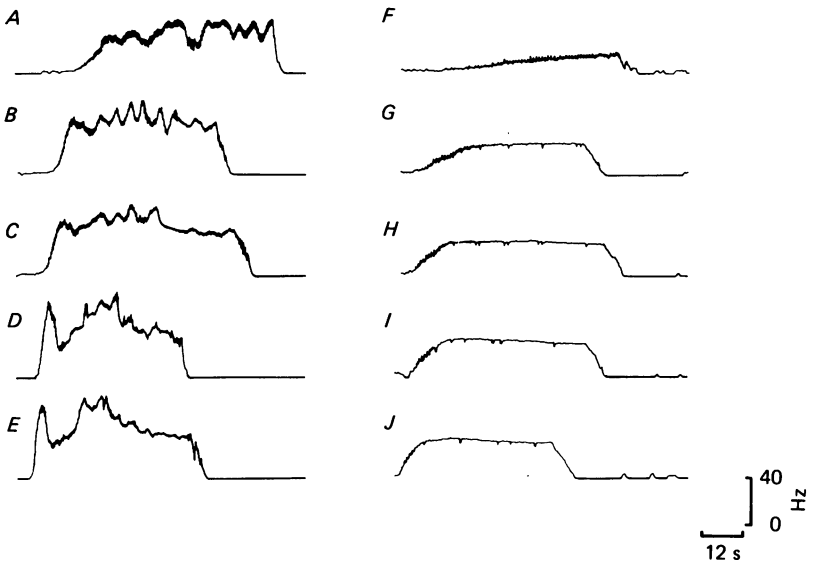

Fig. 6. The responses of two oesophageal mechanoreceptor units to pressure ramps of different gradient. Five different ramps (gradients (mmHg/s): $A = 0{\cdot}06$, $B = 0{\cdot}11$, $C = 0{\cdot}18$, $D = 0{\cdot}34$, $E = 0{\cdot}41$) produced significant changes in the discharge pattern of the unit. The discharge frequency fluctuated markedly when the rate of pressure change was low. As the rate of pressure change increased the 'burst' response became more obvious as did a second more sustained increase in discharge frequency. In another unit (gradients (mmHg/s): $F = 0{\cdot}07$, $G = 0{\cdot}19$, $H = 0{\cdot}28$, $I = 0{\cdot}42$, $J = 0{\cdot}64$) the discharge pattern altered relatively little despite large changes in the rate of pressure increase.

These units were located in all four zones of the thoracic oesophagus. The pressure range over which the relationship was approximately linear was narrow for some units. The linear responses occurred over different parts of the oesophageal pressure range and four of the eight units showed a 'threshold' effect (Fig. 5). The discharge rate reached a maximum value in the eight units and remained approximately constant despite continued oesophageal distension. A very similar relationship existed between oesophageal volume and the adapted discharge rate (Table 1).

Ramp pressure responses

The responses of mechanoreceptors to ramp pressure changes were examined during periods of apnoea because of the fluctuations in discharge rate produced by lung inflation. Pressure ramps did not produce reflex muscular activity. There was no oesophageal wall movement, pressure fluctuation or electromyographic activity during distension even with both vagus nerves intact. Pressure ramps with gradients

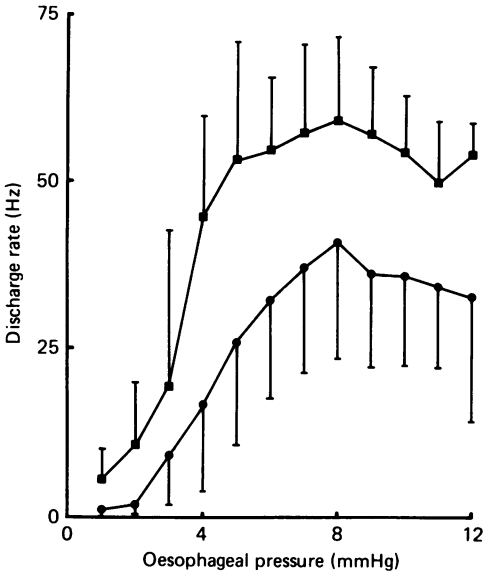

Fig. 7. The discharge pattern of oesophageal mechanoreceptor units during pressure ramps of similar gradient depended upon the conduction velocity of the afferent fibre. Seven units with velocities greater than 19 m/s (squares) had significantly higher discharge rates than ten units with conduction velocities less than 17 m/s (circles). Above pressures of 10 mmHg the difference was not significant. The bar represents one standard deviation from the mean.

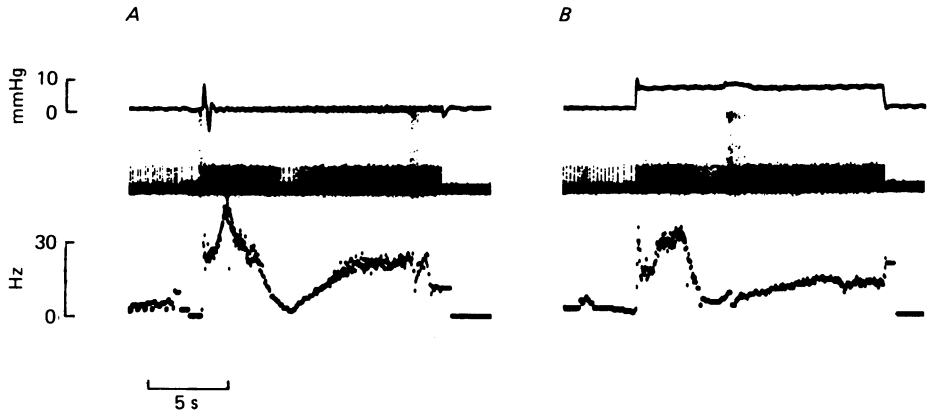

Fig. 8. The discharge pattern of an oesophageal mechanoreceptor unit during isotonic and isometric contraction of the oesophagus. Contraction was produced by stimulating the right vagus supramaximally (20 V; 0·1 ms; 20 Hz). In *A* and *B*: upper record, oesophageal pressure; middle record, afferent fibre discharge; lower record, discharge frequency. In *A* oesophageal volume decreased while pressure remained constant. Thirty seconds later (*B*) the discharge pattern during isometric contraction had the same biphasic shape (the frequencies before and during contraction were a little lower).

of less than 0·5 mmHg/s produced very similar changes in volume. The oesophagus appeared less compliant with steeper pressure gradients due to the flow restriction imposed by the perfusion tubing.

Pressure ramps of increasing gradient had relatively little effect on the discharge pattern of some units while significantly altering the pattern of others (Fig. 6). In

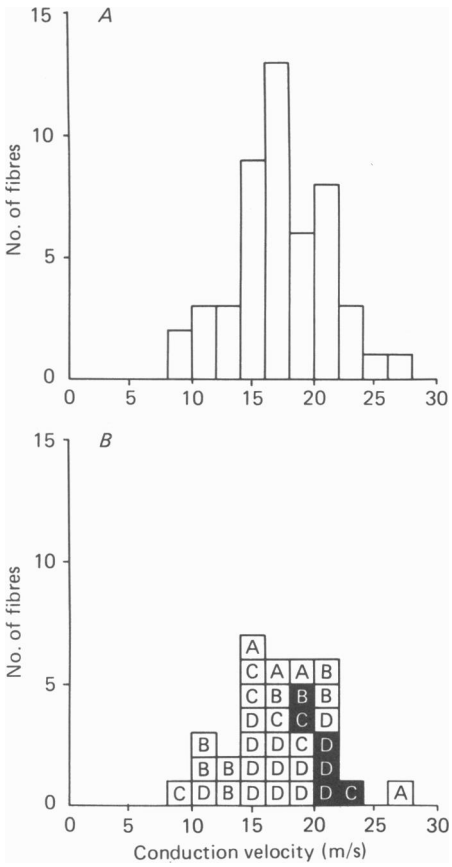

Fig. 9. Histograms of conduction velocities of single oesophageal mechanoreceptor units. *A*, velocities of forty-nine units that responded to oesophageal distension with sustained nerve activity. *B*, the velocities of thirty-three of these units whose receptor site was located in the oesophageal wall. Location sites: A, cranial to aortic arch; B, adjacent to arch; C, adjacent to lung hilum; D, caudal to division of left vagus into 'crossing branch' and the ventral vagus. More caudally located units did not have slower conduction velocities. Units with 'burst' responses (shaded squares) were located in lower three zones and had a narrow range of velocities.

this latter group, some units more than doubled their mean firing rate over a pressure range of about 1 mmHg. This marked increase in discharge rate was called a 'burst' response. A 'burst' became more distinct as the gradient of the pressure ramp increased but the pressure at which the response occurred remained constant. Six of thirty-three units produced a 'burst' at various pressures with most responses occurring soon after the start of a ramp. Three of the units that exhibited a 'burst'

also displayed a second more sustained increase in firing rate. Declining ramps of different gradients never produced a 'burst'. The receptor sites of the six units were spread evenly throughout the thoracic oesophagus.

The discharge patterns of seventeen units during pressure ramps of similar gradient are shown in Fig. 7. Part of the variation in the firing pattern could be related to the conduction velocity of the afferent fibre. Seven units with conduction velocities greater than 19 m/s (three of these showed a 'burst') had significantly higher firing rates over the pressure range of 1–10 mmHg than ten units with conduction velocities of less than 17 m/s ($P < 0.05$). Both groups reached a maximum discharge rate at the same pressure.

Oesophageal afferent fibre discharge during oesophageal contraction

Isometric contraction of the oesophagus was produced by stimulating the right vagus nerve supramaximally at 20 Hz for 5–30 s while keeping the tap between the oesophagus and the perfusion bottle closed. All units increased their discharge rate in the first second. The discharge rate remained approximately constant or declined at a variable rate to a frequency that was less than half of the peak rate but still higher than the resting rate. The discharge pattern did not change when the oesophagus was allowed to empty (Fig. 8). Under these conditions pressure changes occurred only at the onset of contraction and volume decreased by 30–50 ml.

Conduction velocity properties

The conduction velocities of forty-nine oesophageal afferent fibres ranged from 9·2 to 27·7 m/s (mean, 17·4; S.D., 4·1). Units whose receptors were located at opposite ends of the thoracic oesophagus had similar conduction velocities (Fig. 9). Six units with 'burst' responses had conduction velocities (range, 18·7–23·5 m/s; mean, 21·2; S.D., 1·8) which were significantly higher than the twenty-seven other units whose receptors were located (range, 9·3–27·7 m/s; mean, 16·3; S.D., 3·9) ($P < 0.01$).

DISCUSSION

In the present investigation distension of the oesophagus resulted in a slowly adapting discharge pattern in all receptors which reached a maximum at a low oesophageal pressure. The discharge rate was also increased by isometric contraction and this discharge pattern was not altered when the oesophagus was allowed to empty. Thus all receptors were of the same functional type, with the mechanoreceptor being 'in series' with striated muscle elements (Iggo, 1955; Leek, 1977). There were cardiorespiratory fluctuations in the discharge pattern which changed character at different levels of distension. The changes were due to a number of effects which included the narrow response range of the receptors, cardiorespiratory fluctuations in oesophageal pressure and compression from adjacent organs. These changes were observed in receptors located throughout the thoracic oesophagus which suggested that uniform alterations in wall tension were responsible. The secondary rhythms were observed in a uniformly distended oesophagus in an open-chested anaesthetized dog and thus are unlikely to occur under normal conditions. However, the potential for secondary rhythms must not be ignored, particularly in conscious animal studies

where the discharge pattern may be important for unit identification (Rousseau & Falempin, 1979).

The predominant characteristic of oesophageal mechanoreceptor discharge patterns was the narrow response range. This was observed for ramp or constant pressure distension. Maximum discharge rates occurred at lower pressures during ramp distensions which reflected a rate-sensitive property in a proportion of the receptors. While slowly adapting visceral mechanoreceptors are known to respond to the rate of distension (Paintal, 1963), it has not been demonstrated that this property varies considerably or that greater dynamic responsiveness is displayed by units whose afferent fibres have higher conduction velocities. All afferent fibres were of the small myelinated type and were thus similar to those innervating the oesophagus in the ferret and the sheep (Falempin *et al.* 1978; Andrews & Lang, 1982). In both previous studies unmyelinated afferent fibres have also been identified. Despite the use of appropriate methods (Iggo, 1958), unmyelinated oesophageal afferents were not detected in the dog, although unmyelinated nerve fibre activity was often recorded. In previous studies the lower oesophageal sphincter zone, a region richly innervated by unmyelinated nerve fibres, has not been excluded as in the present study (Falempin *et al.* 1978).

The discharge pattern of oesophageal units during distension was qualitatively similar to that of a bladder afferent unit although the saturation pressure for the bladder unit was much higher (Floyd, Hick & Morrison, 1976). In contrast, the rate of discharge of slowly adapting gastric tension receptors was not linearly related to intra-gastric pressure but did have a linear relationship with gastric volume (Iggo, 1955; Paintal, 1963). However, as gastric volume continued to increase the discharge rate rose disproportionately whereas the discharge rate of all oesophageal units reached a definite maximum. It is likely that slowly adapting visceral mechanoreceptors in different organs vary markedly in their responses to passive organ distension.

The narrow response range of oesophageal mechanoreceptors was of even greater significance if the 'burst' response constituted part of the normal physiological discharge pattern. The 'burst' may have reflected the effect of an unphysiological stimulus as has been inferred to have occurred in a number of studies on gastrointestinal mechanoreceptors (Andrews, Grundy & Scratcherd, 1980). In the present study the usual filling rate was less than 300 ml/min which was well within the normal ingestion rate of large dogs. It is possible that the 'burst' response was due to reflex vagal motor activity, although it is rare to observe this in anaesthetized animals, except in certain species (Andrews *et al.* 1980). Local muscle contraction has been suggested as a cause of irregularity in the discharge pattern of gastric mechanoreceptors (Paintal, 1963). In the present study, oesophageal electromyogram activity was not observed during passive distension, although this might not have represented the electrical state of the muscle adjacent to the receptor. The location of 'burst' receptors throughout the oesophagus and the narrow range of conduction velocities of these units suggested that the response was not an artifact. The present study has demonstrated that oesophageal mechanoreceptors, in addition to having different degrees of dynamic responsiveness, can also be functionally subdivided into different types.

Most oesophageal units exhibited a narrow pressure range over which there was

an approximately linear relationship between pressure and discharge rate. There was no linear relationship in units with 'burst' responses. While the upper limit of the response range could be determined accurately none of the receptors demonstrated a true 'threshold' as all were spontaneously active. At an oesophageal pressure of 3 mmHg all receptors had more than tripled their spontaneous firing rate. This contrasts with a threshold range of 5–15 mmHg for mechanoreceptors in the lower oesophagus of the ferret (Andrews & Lang, 1982) and a similar value for a bladder afferent unit in the cat (Floyd *et al.* 1976).

Thus in the dog the response range of oesophageal mechanoreceptors is narrow, particularly so in units with 'burst' responses, and encompasses only the lower end of the oesophageal pressure range. This may reflect the specialized nature of the dog's oesophagus. The canine oesophagus is structurally similar to that of the rabbit, the ferret and the sheep, with inner and outer layers of striated muscle (Vantrappen & Hellemens, 1974). The functional properties of the canine oesophagus and its afferent innervation, determined with bolus diversion experiments (Janssens, Valembois, Vantrappen, Hellemans & Pelemans, 1973; Janssens, Valembois, Hellemans, Vantrappen & Pelemans, 1974), suggest that an intraluminal bolus is required for primary and secondary oesophageal peristalsis in the cervical region, but not in the thoracic region, where a bolus improves the efficiency of peristalsis. The dog is the only species which is known to require an intact afferent innervation for efficient peristalsis (Janssens, 1978). Thus it is possible that the characteristics of oesophageal mechanoreceptors described in the present study are specific to the dog and that disturbance of these properties underlies the propensity of this species to develop disorders of oesophageal motility. However, the narrow response range and the almost switch-like performance of some receptors are suitable characteristics for the afferent innervation of an organ that has a low residual volume and which remains empty under normal conditions.

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