Selective vagotomy of the canine oesophagus— a model for the treatment of hiatal hernia

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Edwards, M. H. (1976). *Thorax*, **31**, 185–189. **Selective vagotomy of the canine oeso-phagus—a model for the treatment of hiatal hernia.** Hiatal herniation caused by contraction of the longitudinal muscle of the oesophagus has been prevented by dis-connecting the local vagal nerve supply while preserving the vagal connections to more distant organs. A selective oesophageal vagotomy above the lung hilum may prove an effective adjunct to orthodox hiatal hernia repair in man.

Repair of hiatal hernia as a treatment for oeso-phageal reflux remains unsatisfactory in up to 22% of cases (Mustard, 1970), usually because of a recurrence of the hernia.

The results are even less favourable in the presence of oesophageal shortening (Skinner and Belsey, 1967). Botha (1962) and Johnson (1968) have pointed out that shortening may be caused by vertical traction from the longitudinal muscle of the oesophagus. Davidson (1972) and Mullard (1972) have described myotomies of this muscle as an adjunct to orthodox hiatal hernia repair, so as to obtain more permanent reduction of the hernia. Such techniques, however, are theoreti-cally prone to oesophageal perforation, sub-mucosal ischaemia, and diverticulum formation. Weakening of the longitudinal muscle by discon-necting its motor nerve supply, the vagus, appeared to be potentially safer and to offer prospects of a more radical effect.

A logical approach to this problem, not pre-viously reported, was to assess the weakening effect of dissociating different sections of the vagus from the oesophagus while preserving the vagal connections with more distant organs.

The dog was chosen as the experimental model because the vagal bundles over the oesophagus are easily identifiable and the longitudinal muscle is well developed, which would make for clarity of dissection and for large differences in experi-mental response.

MATERIAL AND METHODS

Fasting adult mongrel dogs of mean weight 20·7 kg were anaesthetized using 200 mg thiopen-tone sodium intravenously, intubated with a cuffed endotracheal tube, and ventilated with a Bird Mark 2 ventilator using halothane 1·5% and oxygen at 2 l/min.

A mean of 3000 ml of 0·9% sodium chloride was administered intravenously to replace blood and fluid losses. Each dog was killed at the end of the experiment.

One side of the whole intrathoracic oesophagus was exposed by reflecting a large chest wall flap consisting of one side of the sternum and the fourth to eighth ribs, and removing the ipsilateral lung. Kymographs were attached to the oeso-phagus at three points—at the level of the hiatus, at the lower border of the hilum, and at the upper border of the aortic arch. The kymographs were connected by a system of pulleys to record longi-tudinal movements (Fig. 1).

The pressures in the lower oesophagus and stomach were measured by means of a continu-ously infused 3-lumen oesophageal tube connected to three Model P23 De Statham transducers, and recorded on a Devices M4 4-channel recorder. The fourth channel was adapted to record heart rate from a Devices ECG unit and a Devices Ratemeter type 2750.

Through a neck incision, both vagi were ex-posed at the level of the cricoid cartilage and divided, and their distal ends were then attached to bipolar silver electrodes. A train of square wave pulses of frequency 20 Hz, at an amplitude of 4·5 volts, was applied to the electrodes for periods of 5 seconds. This pulse train had been shown in preliminary experiments to produce
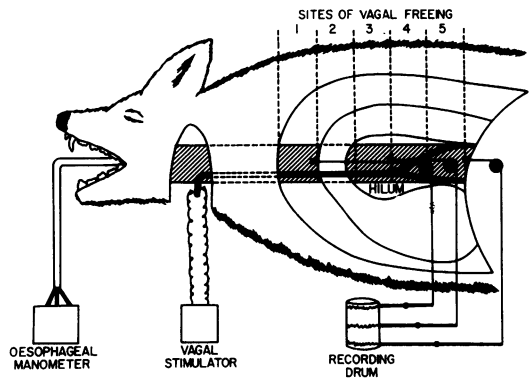
SITES OF VAGAL FREEING

FIG. 1. *Diagram of the experimental model.*

RESULTS

Stimulation of a cervical vagus led to a tetanic contraction of the thoracic oesophagus with displacement of the oesophagogastric junction vigorously into the chest, producing a hiatal hernia. There was no measurable displacement of the upper end of the oesophagus at the cricoid cartilage, and no other muscles were seen to contract. The mean rise of the gastro-oesophageal junction was 2·2 cm when one vagus was stimulated, and 2·4 cm when both vagi were stimulated. The response was unaffected by L-hyoscine and was abolished by D-tubocurarine, suggesting that only striated muscle was involved (Fig. 2).
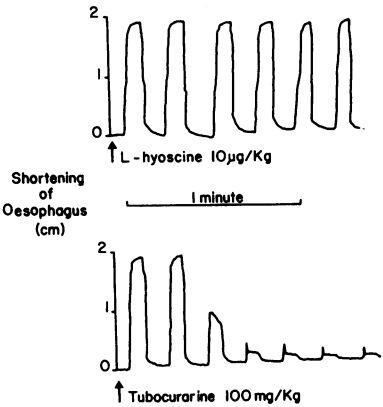
maximum shortening of the oesophagus without damage to the vagi.

The cervical vagi were stimulated, singly and together, and the resulting movements of the three oesophageal points were recorded by means of the kymographs. A mean of three recordings of 5-second stimulations taken at 10-second intervals was used in analysis.

Fifteen dogs were divided at random into three groups of five. In the five control dogs, the right side of the oesophagus was exposed by reflecting a right chest flap, and groups of recordings were made without interference with the intrathoracic vagi at 20-minute intervals for 100 minutes. In five dogs, with the right side of the oesophagus exposed, the right vagus was progressively freed in five stages from the hiatus to the level of the first rib at 20-minute intervals, and recordings were made after each freeing. The five stages comprised hiatus to midsubhilum, midsubhilum to lower hilum, lower hilum to upper hilum, upper hilum to upper border of the aortic arch, and aortic arch to the first rib (Fig. 1). In the last five dogs, similar recordings were made while the left vagus was progressively freed from the left side of the oesophagus, exposed through the left chest.

FIG. 2. *Effects of L-hyoscine and tubocurarine on contraction of longitudinal muscle of the oesophagus on stimulating the vagus in the neck.*

In the control animals, repeated stimulation of the vagi at 20-minute intervals for 100 minutes showed no significant change in the shortening of the oesophagus (Table I). Therefore it was assumed in the test animals that changes in the shortening of the oesophagus were due to the interference with the intrathoracic vagi, and not to general deterioration in the condition of the animals.

TABLE I
MEAN DISPLACEMENT OF OESOPHAGEAL KYMOGRAPHS (cm) ON STIMULATING THE VAGI IN THE NECK IN FIVE CONTROL DOGS AT 0 MINUTES AND AFTER 100 MINUTES

Oesophageal Kymographs	Left or Right Vagus stimulated			Both Vagi stimulated		
	Aortic Arch	Hilum	Hiatus	Aortic Arch	Hilum	Hiatus
Time 0 min	−0·12	0·89	2·04	−0·14	1·25	2·46
100 min	−0·06	0·89	2·24	−0·05	1·15	2·75

TABLE II

EFFECTS ON GASTRO-OESOPHAGEAL JUNCTION OF STIMULATING THE VAGI IN THE NECK BEFORE AND AFTER COMPLETE FREEING OF ONE VAGUS FROM THE INTRATHORACIC OESOPHAGUS

Left or Right Vagus freed (10 dogs)	Mean Elevation of Gastro-oesophageal Junction (cm)		
	Before Freeing	After Freeing	% Loss of Elevation
Ipsilateral vagus stimulated in neck	2·13	0·25	88·2
Opposite vagus stimulated in neck	2·63	1·99	24·4
Both vagi stimulated in neck	2·91	1·73	40·8

EFFECTS OF COMPLETE VAGAL FREEING In the test groups, after complete intrathoracic freeing of the stimulated vagus, there was an 88% loss of displacement of the oesophageal junction compared with the displacement before freeing (see Table II). Loss of displacement was seen when either the right or the left vagus had been freed (right 80%, left 97%). The very high loss on the left side occurred when the recurrent laryngeal nerve was separated from the oesophagus. In three dogs, the recurrent laryngeal nerve was unintentionally cut during this part of the dissection.

After stimulation of the opposite vagus following complete freeing of the ipsilateral vagus, there was a mean loss of displacement of 24%. This suggests that unilateral freeing causes interference with the neuromuscular connections of the opposite side of the oesophagus. However, stimulation of both vagi, when one was completely freed, produced a loss of displacement amounting to only 40% compared with stimulation before freeing, indicating that maximum loss of displacement would probably be achieved by freeing both vagi.

EFFECTS OF PROGRESSIVE VAGAL FREEING Freeing of the vagus from the oesophagus in five stages from hiatus to first rib led to a progressive loss of shortening of the oesophagus on stimulating the cervical vagi. First the lower part of the oesophagus would cease to contract while the more proximal parts would continue to do so and, indeed, would shorten more than at the start of the procedure. As the dissection continued, more and more of the intrathoracic oesophagus became inert, until after the final freeing hardly any contraction was seen at all. This applied to the circular as well as the longitudinal muscle. The cumulative effect of this striking phenomenon is best demonstrated diagrammatically (Fig. 3). Each non-contracting part lengthened to a maximum corresponding to the resting length. It was concluded that this feature indicated denervation.

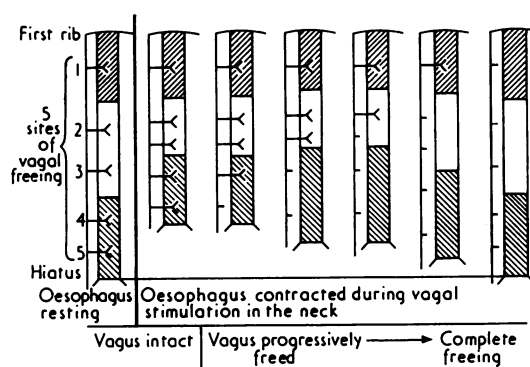Statistical analysis of this effect was carried

FIG. 3. *Diagrammatic summary of effects on longitudinal muscle of the oesophagus of progressive vagal freeing.*

out to confirm this appearance and to determine precisely which parts of the vagus innervated which parts of the oesophagus. To do this, an index was obtained of the effect of denervation on the freed side alone. It was called the X_1 effect and was calculated by subtracting the displacement of the kymographs on stimulation of the vagus being freed from the displacement on stimulation of the vagus of the opposite side. Mean values of X_1 as the denervations progressed are seen in Table III. In summary, freeing the lowest section of the vagus (section 5) had no effect, but freeing section 4 (midsubhilum to lower border of hilum) had a highly significant weakening effect on the oesophagus below the hilum. Freeing section 3 (behind the hilum) produced no significant further effect, but freeing section 2 (upper border of hilum to upper border of aortic arch) significantly affected the oesophagus between the lower hilum and the aortic arch. Freeing section 1 (above the aortic arch) affected the oesophagus at a similar supra-aortic level. It is quite likely that freeing section 3, but not 4, or even freeing section 2, but not 3 or 4, would have affected the hilum-to-hiatus segment of oesophagus, but because freeing was always upward, this evidence was not available. However, the

TABLE III

MEAN VALUES OF X_1, DISPLACEMENT (cm) OF THE OESOPHAGUS ON STIMULATION OF THE CERVICAL VAGUS ON THE UNFREED SIDE, MINUS THE DISPLACEMENT ON THE SIDE BEING FREED

Site of freeing Left or Right Vagus	Aortic Arch Kymograph	Intervening Oesophagus	Hilum Kymograph	Intervening Oesophagus	Hiatus Kymograph
No freeing	+0·13	−0·02	+0·12	−0·39	+0·51
Change					−0·03NS
Section 5	+0·07	−0·04	+0·02	+0·46	+0·48
Change				+0·65**	
Sections 4+5	+0·02	−0·51	−0·48	+1·20	+0·72
Change			+0·16NS		
Sections 3+4+5	−0·20	−0·45	−0·65	+1·56	+0·92
Change		+0·95***			
Sections 2+3+4+5	−0·44	+0·50	+0·06	+1·43	+1·50
Change	+0·27*				
Sections 1+2+3+4+5	−0·17	+0·66	+0·49	+1·10	+1·59

*P < 0·05; **P < 0·01; ***P < 0·001.

oesophagus between the hilum and the aortic arch was unaffected by freeing vagal fibres below the upper border of the hilum.

PRESSURE MEASUREMENTS Table IV shows the values of the resting pressures in the oesophagus at the level of the lower sphincter during the procedures. There were no significant differences in the values in the control groups with the passage of time, nor in the test groups as the vagal freeing was performed.

Estimations of the sphincter pressures during vagal stimulation were not obtained since the violent contraction of the longitudinal muscle displaced the recording tube away from the sphincter area into the stomach.

CHANGES IN HEART RATE Recording of heart rate in three control dogs and six test dogs showed profound falls (mean 31/min) on vagal stimulation. In three of the test dogs, freeing of the vagi from the oesophagus did not affect this fall. However, in the three remaining test dogs the falls were abolished on freeing sections 4, 3, and 2 respectively, suggesting interference with cardiac vagal fibres.

CONTINUITY OF THE VAGAL NERVE In all test dogs, after freezing the vagi, bundles passing through the diaphragmatic hiatus appeared grossly intact. In two dogs, in which barium was instilled into the stomach after vagal freeing, the contrast medium had passed through the pyloric canal within 20 minutes; evidently there had been no serious damage to the vagal supply to the pylorus. The intragastric pressure recordings did not register any rises due to peristalsis in this preparation at any time, so no information is available concerning the vagal supply to the stomach.

DISCUSSION

It is widely agreed that an adequate length of intra-abdominal oesophagus is largely responsible for successful control of gastro-oesophageal reflux (Davidson, 1972; Orringer, Skinner, and Belsey, 1972; Hill, Skinner, and Woodward, 1974). The principles of fluid mechanics underlying this concept have been clearly described by Johnson (1968).

However, there is a physiological need in most mammals to cancel the competence at the gastro-oesophageal junction to allow the expulsion of vomit and gas. This is achieved in man (Botha,

TABLE IV

MEAN STATIC LOWER OESOPHAGEAL SPHINCTER PRESSURE AT BEGINNING AND END OF EACH EXPERIMENT IN CONTROL AND TEST GROUPS (in mm Hg)

	Control (5 dogs)	Left or Right Vagus freed (9 dogs)
Beginning	20	21·1
End	22·5	22·6

1962), in the cat (Torrance, 1958), and in the dog (Johnson, 1968) by sudden movement of the intra-abdominal oesophagus and the cardia into the chest. Whereupon the contraction of the abdominal muscles and the diaphragm succeed in forcing the gastric contents into the oesophagus.

In the dog (Johnson, 1968) and the cat (Torrance, 1958), the elevation of the gastro-oesophageal junction was thought to be caused by contraction of the striated longitudinal muscle of the oesophagus. Our findings support this view for the dog.

In the human cadaver, the effect can be imitated by pulling on the thoracic oesophagus (Johnson, 1968), but direct evidence of longitudinal muscle contraction in man is lacking. In the cat, the striated muscle extends very rarely below the lower border of the lung hilum, and a similar arrangement is seen in man (Arey and Tremaine, 1933). In the cat, the striated muscle almost exclusively controls oesophageal shortening during vomiting (Torrance, 1958). It is tempting to speculate that the same muscle may perform the same function in man.

Our results show that, in the dog, innervation from the vagus to the striated muscle is mainly segmental. By analogy, we suggest that the relevant vagal fibres in man might branch from the parent nerve above the level of the lung hilum.

A selective vagotomy localized to this area would avoid the achalasia-type of malfunction that has been described in experimental animals undergoing total subhilar vagotomies (Carveth *et al.*, 1962). However, these workers have shown that selective subhilar vagotomies in the dog produce only transient dysphagia. Our results also support their findings that the function of the lower oesophageal sphincter was unimpaired by the latter procedures.

Possibly the safer procedure of dissecting the relevant part of the oesophagus from the vagal plexus rather than freeing the vagi from the oesophagus would obviate the danger of damage to recurrent laryngeal and cardiac nerves reported here. In addition, this procedure would bring about bilateral rather than unilateral vagal denervation, a point supported by our results.

Clearly, the state of investigation so far does not justify attempting this procedure in man in the treatment of hiatal hernia. However, it is interesting to note that where vagal disconnection was performed, albeit as a method to control syncope following swallowing, the patient was symptom-free after operation (Sapru *et al.*, 1971).

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