THE EFFECTS OF ACUTE OESOPHAGEAL DISTENSION ON ARTERIAL BLOOD PRESSURE, E.C.G. AND RESPIRATION IN DOG

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Summary : Anginal pain is a common clinical finding during cardiospasm or during pneumatic dilatation of oesophagus to relieve cardiospasm. Thus the present work was taken up to find out any relationship between the oesophagus and the cardiovascular system during experimental distension of the oesophagus in dogs. Pneumatic distension of oesophagus was done in anaesthetised dogs and its effects on arterial blood pressure, E.C.G. and respiration were studied.

A fall in arterial blood pressure was observed in almost all dogs during distension. E.C.G. changes like a reduction in the 'R' wave voltage, absence of 'R' wave and a shift in the isoelectric line were observed also during distension. Moreover, oesophageal distension produced a transient apnoea in almost all animals and they seemed to be in a respiratory distress. When the distension was continued, the arterial blood pressure instead of remaining low, came back to the control or even higher level inspite of the presence of distension. Bilateral vagotomy did not abolish the hypotensive effect and E.C.G. changes but abolished the increase of blood pressure during the later part of distension. The cardiovascular changes were considered to be of a reflex nature and besides the vagi, the sympathetics seem to play an important role in this.

INTRODUCTION

A clinical correlation of gastro-intestinal disturbances with cardiac disorders has been observed by various workers (1, 3, 9, 10). Anginal pain and pain from gastro-intestinal tract are so alike that it is sometimes difficult to differentiate. An attack of angina pectoris following a heavy meal and relief of anginal attack after eructation of gas are also not uncommon clinical findings. Morrison *et al.* (6) have also reported that pneumatic dilatation of oesophagus is often associated with anginal pain and vasomotor disturbances in man.

Experimental studies to investigate the relationship of the oesophagus and the heart are however so meagre that it is not possible to work out any possible relationship between the two so as to explain the finding of anginal pain associated with oesophageal distension.

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Thus the present study was taken up with an aim to find out any relationship between oesophagus and the heart. The respiration of the animals was also recorded with the view of explaining the cardiovascular changes during oesophageal distension.

MATERIAL AND METHOD

Dogs starved overnight were used. They were mostly young animals weighing between 4–12 *kg.*

Intravenous nembutal was administered and their body temperature was maintained at 37°C by placing the animals on a table with thermostatic control. They were placed in supine position with all the Limbs strapped to the table so as not to allow any change of position during the course of experiments.

A stiff polythene tubing carrying a toy ballon at one end was passed carefully into the oesophagus (mid portion) through the mouth and the tube was held in position by securedly tying it to a stand. The other end of the tube was connected via a 'T' tube to a pressure pump on one hand and the recorder on the other through the pressure transducer which recorded the intra oesophageal pressure. A Beckman type RM Dynograph recorder was used for recording all the parameters by incorporation of suitable Statham's transducers.

Arterial blood pressure recording was done by cannulating the common carotid artery in some and the femoral artery in others.

The oesophagus was distended at two grades of pressures namely, P_1 (45 to 50 mm. Hg) and P_2 (60 to 75 mm. Hg) and its effects on the arterial blood pressure, E.C.G. (Lead II) and respiration were recorded by the dynograph. The effects of distension of oesophagus on the above parameters were again recorded after sectioning both the vagi in the neck thus eliminating any reflex mediated through the vagi during distension. All results were statistically analysed.

RESULTS

The changes in systolic and diastolic arterial blood pressure and pulse pressure as recorded from the common carotid artery and the femoral artery during the experiment are shown in Table I. Table II shows these pressure changes in the same animals after they were bilaterally vagotomised.

Table III depicts the time factors for change in arterial blood pressure before and after vagotomy both at P_1 and P_2 of oesophageal distension.

TABLE I : Effect of distension of oesophagus on arterial blood pressure.

(The figures indicate the percentage change in pressure over the resting value)

Distension pressure	*Parameters*	*Left common carotid artery (No. of expts : P_1 :- 7 & P_2 :- 4)*			*Left femoral artery (No. of expts: P_1 :- 5 & P_2 :- 7)*		
		Systolic pr.	*Diastolic pr.*	*Pulse pr.*	*Systolic pr.*	*Diastolic pr.*	*Pulse pr.*
P_1[45 to 50	Mean	–15.06	–16.55	–8.37	–13.66	–15.24	–4.28
mm. Hg.]	S.D. (±)	4.82	8.97	25.97	6.65	9.23	10.65
	S.E.	1.82	3.39	9.81	2.97	4.12	4.76
	't' value	8.27	4.88	0.85*	4.59	3.69	2.23
P_2[60 to 75	Mean	–14.4	–11.45	–25.02	–17.52	–20.62	–14.42
mm. Hg]	S.D. (±)	2.54	1.85	11.85	8.45	15.52	14.93
	S.E.	1.27	0.92	5.92	3.19	5.86	5.64
	't' value	11.33	12.44	4.22	5.49	3.51	2.55

*Statistically insignificant

TABLE II : Effect of distension of oesophagus on arterial blood pressure after bilateral vagotomy.

(The figures indicate the percentage changes in pressure over the resting value)

Distension pressure	*Parameters*	*Left common carotid artery pressure (No. of expts : P_1:- 2 & P_2 :- 5)*			*Left femoral artery pressure (No. of expts. : P_1 :- 1 & P_2 :- 3)*		
		Systolic pr.	*Diastolic pr.*	*Pulse pr.*	*Systolic pr.*	*Diastolic pr.*	*Pulse pr.*
P_1 [45 to 50	Mean	–14.5	–9.95	–23.35			
mm. Hg]	S.D. (±)	3.0	0.85	6.65	–7.3	–12.5	+3.4
	S.E.	2.12	0.6	4.70	(Only one expt. was		done)
	't' value	6.83	16.58	4.96			
P_2 [60 to 75	Mean	–12.72	–8.24	–24.34	–24.0	–28.53	–9.5
mm. Hg.]	S.D. (±)	5.63	6.85	26.17	4.86	7.05	13.48
	S.E.	2.51	3.07	11.7	2.8	4.07	7.78
	't' value	5.06	2.68	2.08	8.57	7.0	1.22

*Statistically insignificant

TABLE III : Time course of events for change in arterial blood pressure before and after bilateral vagotomy.

(time in seconds)

A. Before vagotomy

Parameters	At P_1 (45 to 50 mm. Hg.) (No. of experiments – 11)			At P_2 (60 to 75 mm. Hg.) (No. of experiments – 9)		
	Latent period for fall	Time of maximum fall	Total duration of fall	Latent period for fall	Time of maximum fall	Total duration of fall
Mean	3.0	6.5	14.89	3.55	7.05	12.21
S.D. (±)	0.592	1.378	7.717	0.464	1.323	5.114
S.E.	0.178	0.415	2.572	0.155	0.444	1.933
't' value	16.85	15.66	5.78	22.9	5.28	6.31

B. After vagotomy

Parameters	At P_1 (No. of expts – 5)			At P_2 (No. of expts – 7)		
	Latent period for fall	Time of maximum fall	Total duration of fall	Latent period for fall	Time of maximum fall	Total duration of fall
Mean	3.1	8.62	18.38	2.78	6.85	15.58
S.D. (±)	0.548	4.563	12.216	0.567	2.982	12.002
S.E.	0.245	2.041	6.108	0.214	1.127	5.000
't value	12.65	4.22	3.0	12.9	6.07	3.11

Table IV contains the findings on respiration rates and amplitude and changes in QRS voltage of E.C.G. during distension before and after vagotomy. Though in most of the animals there was an increase in respiratory rate and amplitude still the results were inconsistent and statistically insignificant. In almost all animals, there was a dicrease in the 'QRS' voltage on E.C.G. but the changes were not significant.

TABLE IV : Effects of distension of Oesophagus on respiration and 'QRS' voltage of E.C.G. before and after bilateral vagotomy.

(The figures indicate the percentage changes over the resting value).

A. Before vagotomy

Parameters	At P_1 (45 to 50 mm. Hg.) (No. of expts. – 7)			At P_2 (60 to 75 mm. Hg.) (No. of expts. – 14)		
	Resp. rate	Resp. amplitude	'QRS' voltage	Resp. rate	Resp. amplitude	'QRS' voltage
Mean	+14.14	+38.77	—7.02	+41.18	+163.44	-8.678
S.D. (±)	37.55	37.15	12.68	39.9	251.35	19.83
S.E.	14.11	14.04	4.79	10.66	67.17	5.29
't' value	1.00	2.76*	1.46	3.86*	2.43*	1.64

B. After vagotomy

Parameters	At P_1 (No. of expts. – 3)			At P_2 (No. of expts. – 8)		
	Resp. rate	Resp. amplitude	'QRS' voltage	Resp. rate	Resp. amplitude	'QRS' voltage
Mean	–21.13	+120.8	–10.96	+17.98	+ 67.12	–3.97
S.D. (±)	35.53	152.43	23.65	43.27	152.00	21.9
S.E.	20.51	88.0	13.65	15.29	53.74	7.74
't' value	1.03	1.37	0.8	1.17	1.24	0.57

*Statistically significant result.

A typical record of the effects of oesophageal distension is shown in Fig. 1. There was bradycardia and hypotension during the distension. In some animals the voltage of 'R' wave of E.C.G. was decreased during distension (Fig. 2.). In some the 'R' waves were dropped (Fig. 3). Similar E.C.G. changes were also seen during distension even after bilateral vagotomy (Fig. 4).

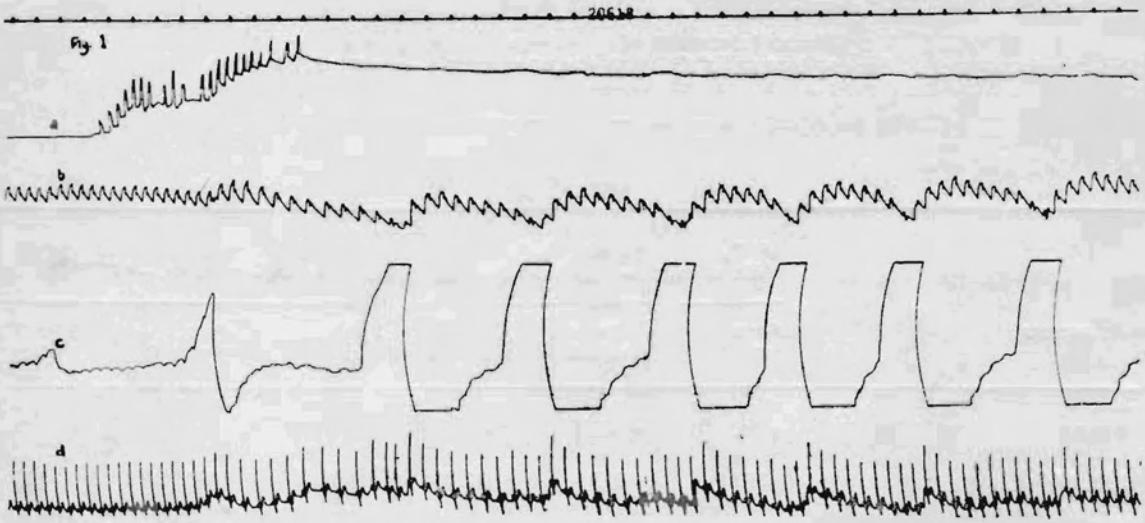

Fig. 1 : Effects of distension of oesophagus before vagotomy.
(a) Oesophageal distension pressure at P_1.
(b) Arterial blood pressure (shows a fall during distension)
(c) Respiratory movements (shows a transient apnoea followed by hyperpnoea during distension)
(d) E.C.G. (shows a reduction in heart rate and shifts in the isoelectric line during distension which correspond to the respiratory movements).

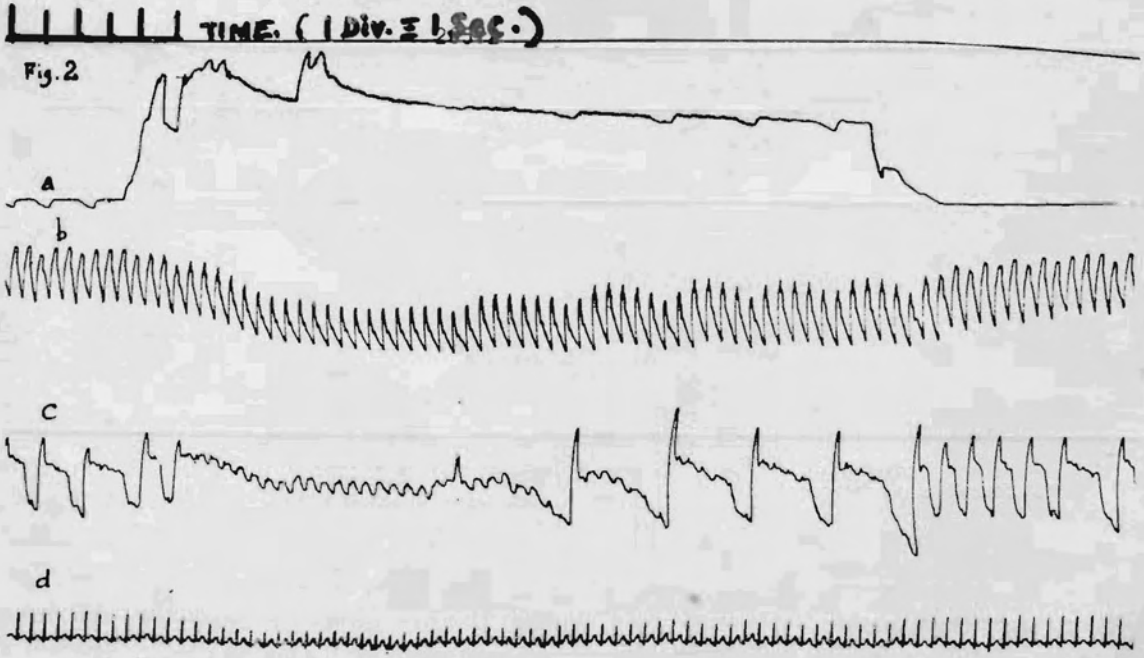

Fig. 2 : Effects of distension of oesophagus before vagotomy.
(a) Oesophageal distension pressure at P_2.
(b) Arterial blood pressure (shows a fall at first and then a rise to control level after release of distension).
(c) Respiratory movements (shows a transient apnoea followed by hyperpnoea during distension).
(d) E.C.G. (shows a reduction in the voltage of 'R' wave during distension).

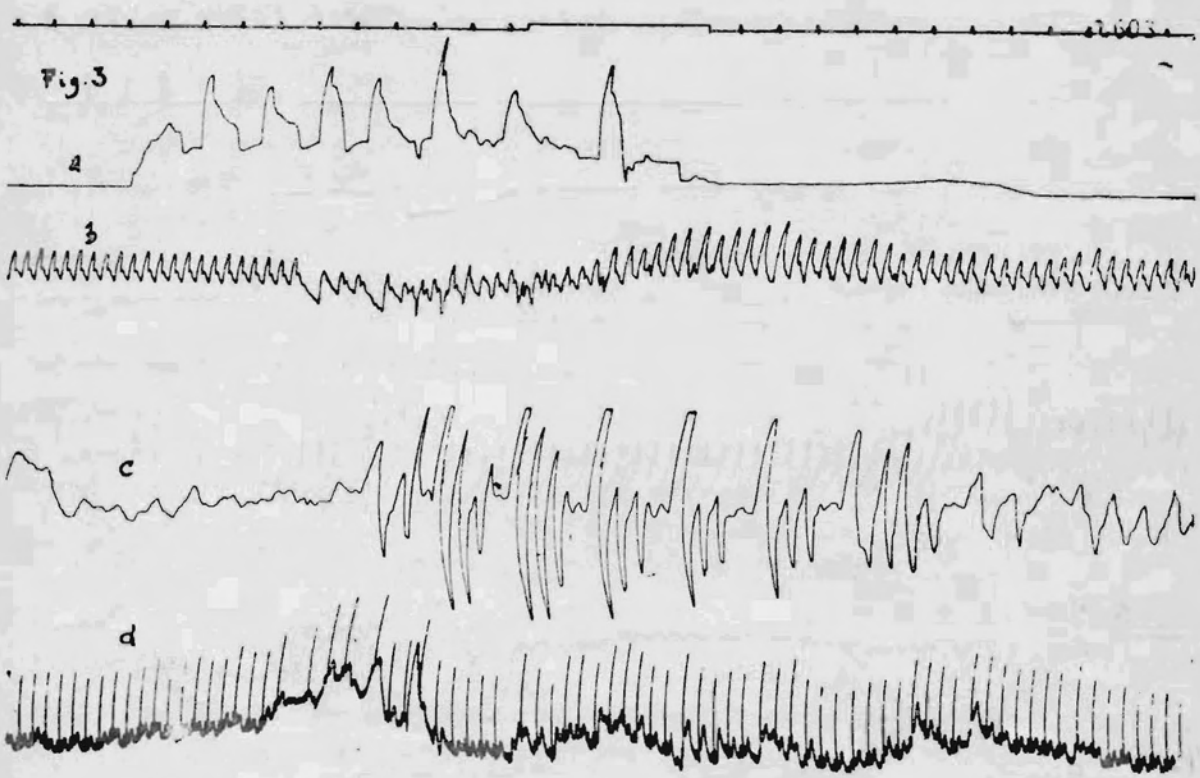

Fig. 3 : Effects of distension of oesophagus before vagotomy.

(a) Oesophageal distension pressure at P_1.

(b) Arterial blood pressure (shows a fall during distension but a rise to above the control level after the release of distension).

(c) Respiratory movements (shows a transient apnoea followed by distressed breathing during distension)

(d) E.C.G. (shows absence of 'R' waves and shifting of isoelectric line during distension.

In all animals a transient apnoea followed by an increase in respiratory rate and amplitude was seen during distension. The animals seemed to be in respiratory distress and the respiration was irregular.

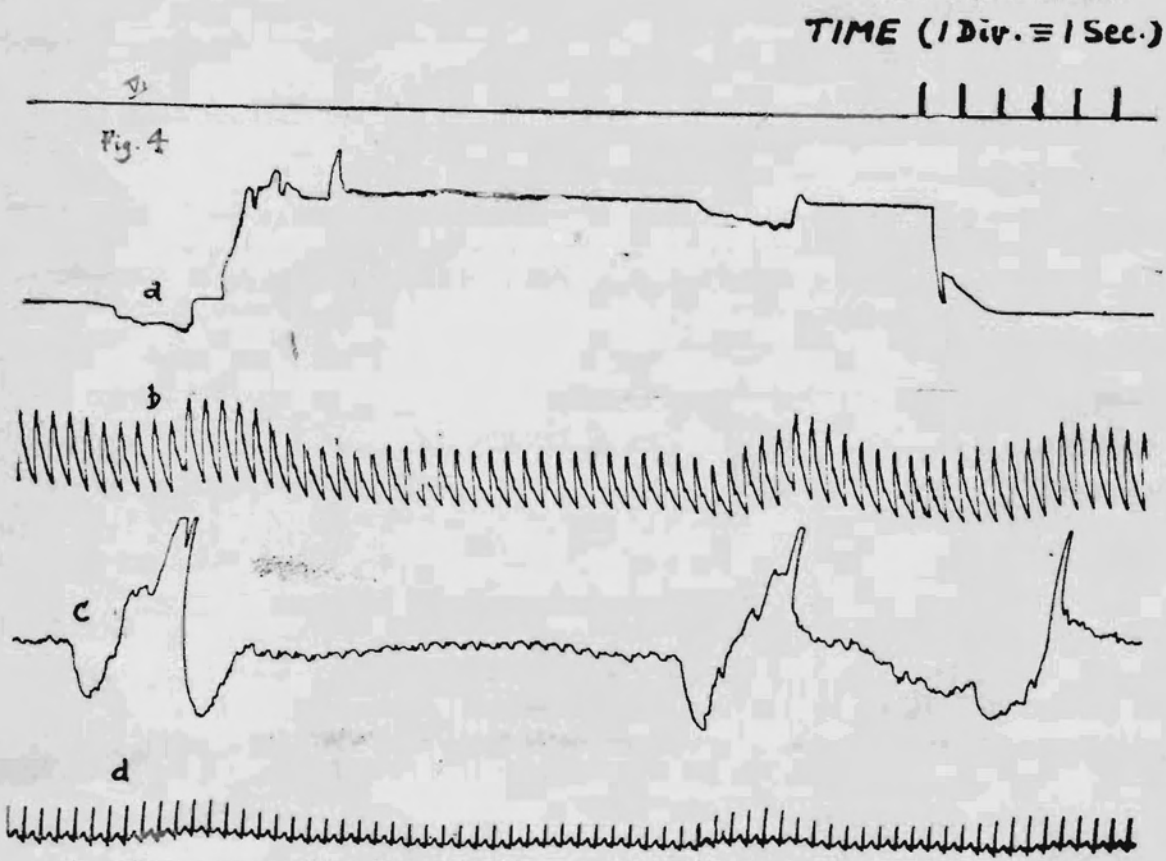

Fig. 4 : Effects of distension of oesophagus after bilateral vagotomy.

(a) Oesophageal distension pressure at P_2.
(b) Arterial blood pressure (shows fall during distension which remained low even after release of distension).
(c) Respiratory movements (show a transient apnoea followed by a distressed breathing).
(d) E.C.G. (shows a reduction in voltage of 'R' wave.

DISCUSSION

In almost all animals there was a fall in both systolic and diastolic blood pressures during either grades of distension. Recordings from the femoral and common carotid arteries were similar and there were no significant difference in these values. This is not in conformity with the observations of Mc. Laughlin *et al.* (5) who observed a difference in the pressures recorded from the common carotid and femoral arteries during their studies on gastric distension, which they explained to be due to mechanical obstruction of the femoral arteries due to compression of the abdominal aorta by the distended stomach. In the present work, had the fall in blood pressure been due to mechanical obstruction of the common carotids by the distended oesophagus, the fall in common carotid arterial

pressure would have been more pronounced than that in the femoral arteries and moreover the fall in common carotid arterial pressure would have been more pronounced at P_2 than at P_1. But the observations do not confirm this. Thus, any mechanical effect of the distended oesophagus on the arteries is ruled out. The hypotension may be explained on the basis of a possibility of the reduction in venous inflow to the heart due to obstruction of superior venacava by the distended oesophagus. Jakhanwal *et al.* (4) demonstrated that obstruction of inferior venacava was a factor for reduced blood pressure during distension of stomach in dogs. In the present work the superior venacaval pressure was not monitored. But it could be argued that, had the drop in blood pressure been due to a mechanical obstruction of the superior venacava, it would have been significantly more at P_2 than at P_1 of oesophageal distension. Our observations do not corroborate this. It would of course not be wise to rule out this mechanical factor without monitoring the superior venacaval pressure during oesophageal distension.

It was observed that oesophageal distension was associated with E.C.G. changes like reduction in votage of 'R' wave, decrease in heart rate due to absence of 'R' wave and change in the isoelectric line of E.C.G. The last effect was probably mechanical due to displacement of the heart due to the distended oesophagus. It may be mentioned here that in some animals, the oesophagus could be suddenly distended to the required pressure and this could be maintained uniformly but in others, to compensate for the stress relaxation in the oesophageal wall, the balloon had to be intermittently inflated to maintain the required distension pressure. It was observed that variations in the type of distension had no effect on the arterial blood pressure response. However, the differences in E.C.G. response may be due to different types of distersion. Similar changes in E.C.G. were reported by (1, 6) during distension of stomach and oesophagus in human beings. Such changes in the heart is likely to be associated with a fall in cardiac output resulting in hypotension. Moreover in the present work the hypotension and changes in cardiac activity occurred within a latent period of about three seconds in almost all animals. Associated with these, a transient apnoea was observed during distension in most of the animals. Association of hypotension with transient apnoea and the short latency of response would suggest the involvement of a reflex mechanism operating from the oesophagus. Iggo (2) showed that pressure sensitive receptors are present in the external muscle layer and serosa of oesophagus which are stimulated by distension.

It was further observed in the present wotk that, when the distension was maintained for a long period, the arterial blood pressure instead of remaining low came back to the normal level or even more inspite of the distension being continued. This might further be due to a reflex compensatoy phenomenon.

To study such a reflex mechanism, the vagi were cut in the neck and the effects of distension were again studied. It was observed that bilateral vagotomy did not abolish the hypotensive effect and E.C.G. changes during distension (Fig. 4). The latent period for the hypotension was similar to that recorded before vagotomy. But it was observed

that after bilateral vagotomy the compensatory rise of blood pressure even during or after distension was not seen.

Paintal (7) while recording electrical activity from the afferent fibers connected with gastric wall receptors in cats found that on distension of stomach, the frequency of discharge in the afferent fibers increased with increasing grades of distension. These fibers travel through the vagi. He proposed that these receptors might be concerned with hunger and satiety mechanism. He did not study the possibility of these fibers taking part in some reflex mechanism associated with cardiovascular changes. Satpathy *et al.* (8) while studying gastric distension in dogs observed an increase in peripheral vascular resistance during distension and this effect was abolished after the vagi were cut. But vagotomy had no effect on the hypotension produced during gastric distension. Thus it seems that sympathetics play a possible role as the afferent pathway in the hypotensive effect produced during distension of stomach or oesophagus. Further study is necessary to confirm this possible role of sympathetics. The question of applicability of these results to an unanaesthetised person is a matter of debate. However, study on unanaesthetised animals would throw more light on this problem.

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