A STUDY OF THE PRESSOR MECHANISM OF THE INTESTINAL STRETCH RECEPTORS IN DOGS¹,²

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The pressor mechanism of intestinal stretch receptors has been studied in dogs. Moderate distension of intestine in chloralose anaesthetised dogs causes rise of arterial blood pressure which is more marked with proximal segments. The heart is slowed and cardiac output diminished. There is different effect on different vascular beds. Cutaneous vasoconstriction is associated with dilatation of the blood vessels of the abdominal viscera and limb muscles. The cutaneous vasoconstriction apparently assures maintenance of adequate blood pressure and blood flow to the active organs of digestion.

Irving, McSwiney and Suffold (1937), Downman, McSwiney and Vass (1948) and Garry (1957) have shown that intestines have a sensory nerve supply anatomically and physiologically distinct from that of the adjacent mesentery. Vasomotor reflexes have been demonstrated in man by the distension of the duodenum by a balloon (Carmichael, Doupe, Harper and McSwiney, 1938). Downman and McSwiney (1946) have demonstrated further that visceral stimulation, particularly pinching the intestine, caused an increase of blood pressure and reflex movements of hindlegs in spinal animals and the reflex arc is completed within the spinal cord. Similar pressor responses from distension of other parts of the intestine in dogs have been reported by Kahali and Gupta (1958, 1958b, 1962a).

This work has been undertaken to investigate the mechanism of this pressor response.

METHODS

Male and female dogs varying in body weight (from 10 to 16 kg.) were used. Chloralose (60 to 70 mg./kg. in 2% solution at 40°C intravenously) under preliminary light ether anaesthesia was used in all experiments (Kahali and Gupta, 1958a).

The abdomen was opened by a 3" median incision. The portion of the gut under investigation was distended with air in a rubber balloon attached to a catheter introduced into the lumen of the gut by a linear cut in the anti-mesentery border and the opening was repaired by a purse string suture. A T glass tube was attached to the catheter. One end of the T was connected with a mercury manometer to measure

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The pressure and the other end to a hand pump used to inflate the intraluminal balloon with air at a predetermined pressure level. Three levels of pressure 10 mm., 20 mm., and 40 mm. of mercury were used in most of the cases while in the case of the duodenum in some cases the pressure up to 120 mm. Hg. was used. In between the distension periods an interval of 15 minutes was given.

The blood pressure was recorded from the left carotid artery by a mercury manometer. The same kymographic record, run on a medium speed, was used for counting the heart rate.

The variation in the blood flow through the intestine and spleen was recorded by the intestine and spleen plethysmograph connected to the Marry's tambour. The variation in the blood flow through the limb with and after removal of the skin was recorded by the technique of Kahali and Gupta (1962).

The cardiac output was estimated by the direct Fick's method.

The animal was given rest for half an hour after the operative procedures before any experimental distension of the intestine was done.

RESULTS AND DISCUSSION

When the intestine was distended there was usually a gradual rise of 5 to 20% of the initial blood pressure. The rise in blood pressure was often of a fluctuating pattern. In certain cases there was a slight initial fall followed by rise of blood pressure. When the gut was emptied the blood pressure quickly returned to the original level. When the filling of the intestine was repeated with emptying and periods of rest the pressor responses remained almost the same.

The rise of blood pressure was more marked with the distension of the upper part of the intestine than that of the distal parts.

The extent of rise of blood pressure is usually related to the degree of distending pressure of the gut. The maximum rise of blood pressure was obtained at 40 to 60 mm. Hg. pressure. If the distending pressure was further increased, the blood pressure either did not show any change or showed a tendency to fall. At 100 and 120 mm. Hg. pressure a definite fall was noted.

The heart showed slowing of 10 to 30% of the initial rate. The electrocardiogram showed diminution in heart rate, increase in P, R interval and increase in amplitude of P, R and T waves. After injecting atropine (0.05 to 0.1 mg./kg. body weight) distension of the gut produced no cardiac slowing. The rise of blood pressure was more marked after atropine as the rise of blood pressure due to vascular effect was not counteracted by cardiac slowing. The cardiac output showed diminution.

The blood flow through the spleen and the intestine showed increase. The increase was more marked with 40 mm. Hg. pressure. The effect on the limb
blood flow was variable. Usually it showed diminished blood flow. After removing
the skin, the limbs always showed increased blood flow, showing that the gut
distension caused vasodilatation of the muscle vessels but vasoconstriction of
the skin vessels. The effect on blood pressure depends on the algebrical summation
of the effects of (i) vasoconstriction of the skin blood vessels causing rise of blood
pressure and (ii) diminished cardiac output and of vasodilatation in various
organs and muscles causing fall of blood pressure. The cutaneous vasoconstriction
is apparently very intense as it more than counteracts the hypotensive effects of fall
of cardiac output and muscular and visceral vasodilatation.

To our surprise, in some of the recent experiments done during cold weather,
the distension of duodenum produced fall of blood pressure with increase of spleen
volume. This was possibly due to intense previously existing cutaneous vaso-
constriction to prevent heat loss. There was no further cutaneous vasoconstriction
to counteract the fall of blood pressure due to visceral and muscular vasodilatation
due to duodenal distension. This is supported by the observations of Best and
Taylor (1961) that stimulation of the wall of a large vein or distension of the
duodenum causes reflex constriction of the cutaneous vessels and that a mechanism
also exists in the experimental animal whereby afferent impulses arising within an
organ, although leading to generalised vasoconstriction result locally in dilatation and
increased blood flow. The physiological role of splanchnic dilatation during
physiological distension caused by ingestion of food is obvious. The cutaneous
vasoconstriction apparently assures maintenance of adequate blood pressure and
blood flow to the active organs of digestion. The cardiac slowing is due to
stimulation of baroceptors.

REFERENCES
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