EFFECT OF DISTENSION OF URINARY BLADDER ON BLOOD PRESSURE AND RESPIRATION

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Summary: Effect of distension of urinary bladder on blood pressure and respiration has been studied in 45 dogs. There is rise of blood pressure and inconsistent change in respiration after graded distension. Abrupt distension and distension of exteriorised bladder showed more pronounced results. The effect is still enhanced when both vagi are cut. The rise of blood pressure on distension of bladder is seen even after ganglion blockade and reserpinisation.

Key words: Abrupt distension, graded distension, bilateral vagotomy, ganglion blockade, reserpinisation

The effect of abrupt and graded distension of urinary bladder on blood pressure and respiration in anaesthetised dogs, with intact vagus, after bilateral vagotomy, chemical sympathectomy and after administration of ganglion blocking agents is reported in the present work. These effects have been observed in situ as well as in the exteriorised preparation.

MATERIALS AND METHODS

Forty five healthy mongrel dogs of either sex weighing between 3.5 and 16 *kg* were anaesthetised with i.v. chloralose at 80 *mgm/kg*. The carotid blood pressure was recorded on a moving kymograph with mercury manometer. The respiration was recorded by Marey's tambour connected to a tracheal cannula.

The bladder was exposed through a mid-line incision in the abdomen above the pubic symphysis. An incision was made in the urinary bladder, and a rubber tube with the balloon attached to its end was introduced into the bladder. The other end of this tube was connected to sphygmomanometer. The urinary bladder was left *in situ* and the incision was closed. In 3 dogs the bladder was exteriorised out of the pelvis. The balloon inside the bladder was distended with varying pressures and changes in blood pressure and respiration were observed. Graded distension of bladder to pressure of 20, 40, 60, 80, 100, 120 and 140 *mm. Hg* and abrupt distension to 80, 100, 120, 140 and 160 *mm Hg* was done in 27 dogs. In 7 dogs, prolonged distension at a mean intravesical pressure of 128.5 *mm Hg* was done for 15 min.

Bilateral cervical vagotomy was done in 12 dogs. The blood pressure of the animal was allowed to come down to normal after vagotomy, before performing the actual experiments. In

8 dogs, in addition to bilateral vagotomy, ganglion blockade was achieved by the i.v. administration of pentolenium tartarate (Ansolysin, May & Baker) at the dose range of 2-3 *mg/kg*. The blockade was tested by stimulation of peripheral end of cut vagus and also by blocking common carotid arteries, showing no change in the blood pressure. Chemical sympathectomy was done in 4 dogs by reserpinisation with two intramuscular injections of serpasil (Ciba), 0.5 *mg/kg* at 12 *hr* and 48 *hr* respectively, before the commencement of the experiments.

RESULTS

The table shows the following results:-

Graded distension of *in situ* urinary bladder to 20 to 140/*mm Hg* has shown a rise of blood pressure varying from 3.2 to 8.3 *mm Hg*. The rise was more pronounced when the distending pressure was between 100 to 140 *mm Hg* (Fig. 1). Graded distension of exteriorised urinary bladder showed a more significant rise of blood pressure which ranged between 7.6 and 11 *mm Hg*. After bilateral vagotomy blood pressure rise on graded distension occured more frequently when compared with the controls before vagotomy. The rise was also more marked and statistically significant. The rise of blood pressure was observed even after ganglion blockade or reserpinisation in the dogs.

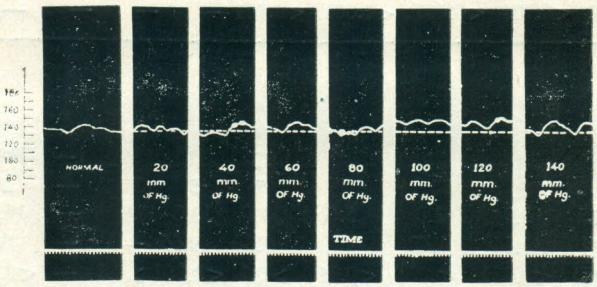

Fig. 1: Showing the effect of graded distension of urinary bladder on blood pressure (upper tracing); dotted line indicates the pretest level. Lower tracing — Time 1 div. = 1 sec.

Abrupt distension of bladder to 80 *mm Hg* or more, in general, showed greater rise of blood pressure than when distended in a graded fashion (Fig. 2). The results were statistically significant. Adaptation of receptors was observed during abrupt distension of bladder to a mean value of 128.5 *mm Hg* maintained for 15 minutes. The blood pressure reached its peak in 1-2 min followed by a gradual fall in 7-8 *mm* and got stabilised below the pretest level even though the bladder distension continued (Fig. 3).

Respiratory changes observed on raising the urinary bladder pressure did not show any consistent findings.

TABLE: Showing effect of distension of urinary bladder on blood pressure.

	Intravesical pressure (mm of Hg).											
	Graded distention							*Abrupt distention*				
	20	40	60	80	100	120	140	80	100	120	140	160
Normal												
No. of Expts.	27	27	27	27	27	23	8	2	5	9	6	4
Rise of B.P. (no.)	10	12	12	14	14	14	6	2	5	7	6	3
Mean rise	3.2	4.5	4.7	4.9	7.0	7.4	8.3	7.0	6.8	13.1	13.0	25.3
S.D. ±	2.15	4.19	3.23	3.39	5.54	6.13	6.25	1.41	5.76	7.10	9.18	14.06
Exteriorised												
No. of Expts.	3	3	3	3	3	—	—	1	1	—	1	—
Rise of B.P. (no.)	2	3	3	3	3	—	—	1	—	—	1	—
Mean rise	8.6	9.0	7.6	9.6	11.0	—	—	7.0	—	—	5.0	—
S.D. ±	2.24	3.0	5.2	3.16	3.60	—	—	—	—	—	—	—
p Value	<0.001	<0.001	<0.001	<0.001	<0.001	—	—	—	—	—	<0.01	—
Bilateral Vagotomy												
No. of Expts.	14	14	14	14	12	11	6	—	3	3	1	4
Rise of B.P. (no.)	8	9	10	10	7	5	2	—	2	2	1	4
Mean rise	4.25	3.3	3.8	5.6	9.0	13.0	8.0	—	16.0	16.0	6.0	17.5
S.D. ±	2.34	1.14	1.48	4.0	6.08	9.65	—	—	8.48	—	—	12.69
p Value	<0.001	<0.001	<.001	<0.01	>0.1	<0.001	>0.1	—	<0.01	<0.01	<0.01	<0.01
Reserpinised												
No. of Expts.	4	4	4	4	4	2	—	1	2	2	1	—
Rise of B.P. (no.)	2	1	3	2	3	1	—	—	2	2	1	—
Mean rise	2.0	4.0	3.3	5.0	3.3	2.0	—	—	5.0	14.0	22.0	—
S.D. ±	2.83	—	1.14	1.41	1.14	—	—	—	1.41	2.82	—	—
p Value	>0.1	>0.1	<0.01	>0.1	<0.001	<0.001	—	—	>0.10	>0.10	<0.01	—
Ganglion Blockade												
No. of Expts.	8	8	8	8	5	4	2	1	2	2	—	—
Rise of B.P. (no.)	4	3	4	2	2	4	2	1	2	1	—	—
Mean rise	3.0	3.0	4.5	8.0	4.0	6.0	5.0	4.0	7.0	8.0	—	—
S.D. ±	2.0	1.0	2.52	2.83	2.83	3.65	1.41	—	4.24	—	—	—
p Value	>0.1	<0.001	>0.1	<0.001	<0.001	<0.01	<0.01	>0.1	>0.1	<0.01	—	—

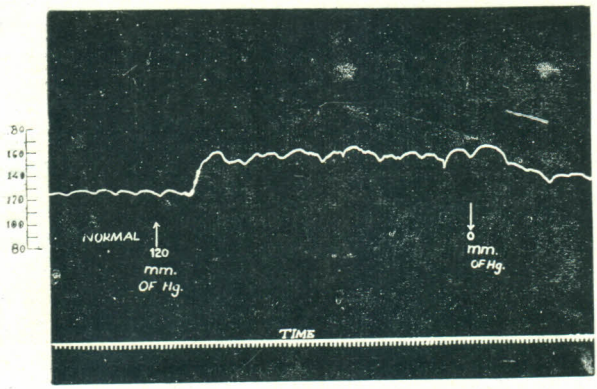

Fig. 2: Showing effect of abrupt distension of bladder on blood pressure (upper tracing), ↑ indicates the point where intravesical pressure was raised, ↓ indicates release of intravesical pressure. Lower tracing = Time 1 div. = 1 sec.

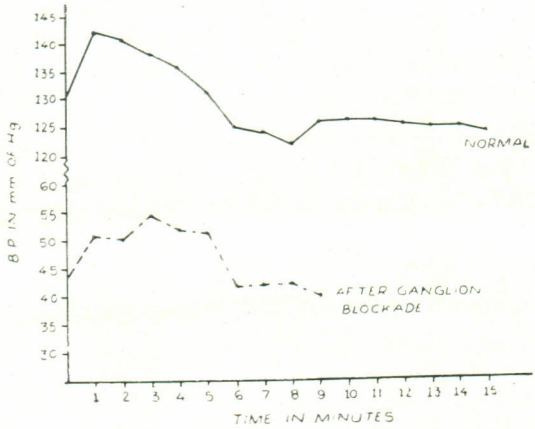

Fig. 3: Showing effect of abrupt distension of bladder to 128.5 *mm Hg* mean intravesical pressure maintained for 15 min (upper tracing). Lower tracing shows the effect of abrupt distension of bladder to 78.5 *mm Hg* mean intravesical pressure maintained for 9 min after ganglion blockade.

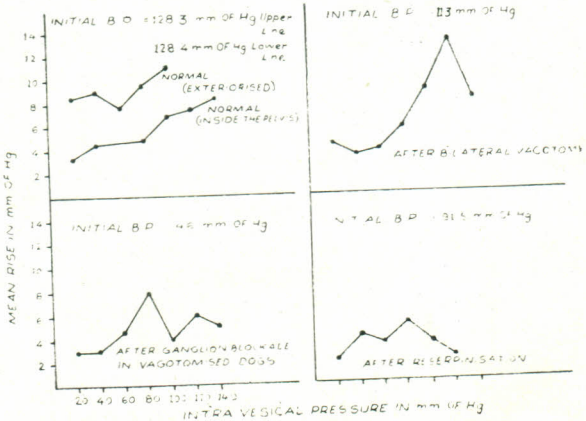

Fig. 4: Showing effect of graded distension of urinary bladder on blood pressure in normal, texeriorised, vagotomised, after ganglion blockade and reserpinised animals.

DISCUSSION

As early as 1899, Sherrington (8) demonstrated that the distension of hollow viscera like urater or bile duct resulted in reflex vascular responses. Reflex responses of nictitating membrane and the blood pressure to distension of bladder and rectum have been reported (9). Paraplegics (C_8 - T_5) have been known to exhibit blood pressure increase on distension of urinary bladder (3). Mukherjee (6) reported vasomotor response on distension of bladder in the cat. This response was more marked after bilateral vagotomy and carotid sinus denervation and was abolished after splanchnicotomy.

Present observations show a rise of blood pressure after a lag period of 30 to 60 seconds on distension of urinary bladder. The lag period varied with the degree of distension. At low distension pressure the lag period was more but as the distension pressure was raised above 100 *mm, Hg* the lag period shortened.

The rise of blood pressure has been observed in response to graded distension and abrupt distension of bladder *in situ* and when it was exteriorised. Comparing the results in these situations, it is evident that the rise of blood pressure is more marked on graded distension in exteriorised bladder. Abrupt distension shows still more rise of blood pressure ranging between 7 to 25 *mm Hg*. This rise of blood pressure in response to distension has been attributed to stimulation of proprioceptive end organs, situated in the bladder wall. Two factors may work in stimulation of these end organs; either the absolute rise of intravesical pressure or increase in the volume of bladder. In the *in situ* bladder inspite of increase of intravesical pressure the volume of the bladder is limited by the pelvis. In exteriorised bladder volume of the bladder increase with the rise of intravesical pressure. Since the results indicate a more marked rise in exteriorised bladder, it is probable that increase in the volume of the bladder plays a more important part in stimulation of the tension mechanoreceptors rather than absolute rise of intravesical pressure. The alteration of the intravesical pressure is minimum over a wide range of intravesical volume. Similar results are reported by other workers (5).

Mechanoreceptors of the bladder have been studied by Adamovich (1) and later by Koshino (5). They have reported that these tension mechanoreceptors are sensitive to abrupt stretch, adapting within 5-60 seconds. Electrical activity in the hypothalamus due to stimulation of these tension mechanoreceptors with the rise of intravesical tension has also been reported (2). The receptors are reported to be interposed in series with detrusor muscle and are more abundant in the bladder neck region (4).

Abrupt distension of bladder to a mean value of 128.5 *mm Hg* in the present work showed a quick rise of blood pressure. When this distension was maintained to that level for 15 min, the blood pressure showed a gradual fall in 7-8 min and later got stabilised below the pretest level even though the distension was continued. The tension mechanoreceptors showed the phenomenon of adaptation. Similar reports of adaptation of these receptors have been published by earlier workers (1,5).

The effect of graded distension after bilateral vagotomy, ganglion blockade and reserpinisation are showin in Fig. 4. The rise of blood pressure is more marked and statiscally highly significant following vagotomy. This may be due to removal of buffer mechanism of the vagus nerves resulting in considerable rise of blood pressure on graded distension following vagotomy agreeing with results of other workers (6,7).

The rise of blood pressure on distension of bladder is reported to be brought about by splanchnic vasoconstriction and active vasoconstriction in the renal circulation reflexly and this reflex response was abolished after bilateral splanchnicotomy (6). Ganglion blockade was done in the present study to decrease the total systemic vascular resistance and splanchnic blood flow thus reducing the blood pressure. Graded distension of urinary bladder after ganglion blockade and after reserpinisation showed a rise of blood pressure which is suggestive of some vasoconstrictor humoral substance released in the circulation. Cross circulation studies and catecholamine levels in the blood before and after distension of bladder may be able to throw some light on the mechanism of pressor response.

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