A STUDY OF THE EFFECT OF PNEUMATIC DISTENSION OF THE URINARY BLADDER ON BLOOD PRESSURE

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Proportional rise in blood pressure was produced by pneumatic distension of urinary bladder. Rise varied through a range of 4.71 to 9.42 m.m. of Hg. No adaptation could be found during 1 min. period of maintained distension. The rise seemed to be influenced by basal level of it, depth of anaesthesia and bleeding of vesical mucosa.

Guttmann and Whitteridge (3) demonstrated that distension of bladder in patients with transverse lesions below mid-thoracic level gives rise to marked rise of B.P. (20-30 m.m. of Hg.). Watkins (6) has shown that the distension of urinary bladder in cats causes a rise in B. P. Mukherjee (4) found that rise of B. P. is never marked when vagi and carotid sinus nerves are intact in cats because of the buffer action of carotid sinus nerves and vagi. Cunningham, Guttmann, Whitteridge and Wyndham (2) have observed rise in venous pressure on distending the bladder upto 14 cms of H_2O and demonstrated increased peripheral resistance in splanchnic Area.

METHOD

Male and Female healthy dogs were used, they were anaesthetised by 50 mgm of Nembutal per Kg. body weight I.V. A rubber balloon was kept overstretched for 2 or 3 days prior to the experiment to remove the resilience of the rubber. Catheter with the balloon attached to its end was introduced into the bladder which was exposed by lower mid-line incision in the abdomen. Catheter was connected to a "T" piece, one end of which was connected to a sphygmomanometer bulb and the other end to the sphygmomanometer itself.

The balloon inside the bladder was distended with pressure of 40 m.m., 60 m.m., 80 mm., 100 m.m., and 120 m.m., of Hg. Femoral B.P. was recorded by traditional direct method

RESULTS

When the bladder was distended, there was usually a small rise of B.P. Some times there was no change and sometimes a fall was registered. Results of 15 individual experiments are shown in Table I.

It is evident from Table I that when intravesical pressure was as high as 120 m.m., of Hg. B.P. rise was never more than 20 m.m. of Hg. which corresponded to 13.2% of Normal. Rise in B.P. commenced after a lag of 2-10 seconds.

TABLE I

No. of Ex.	Normal B.P. in mm. of Hg.	Distension of 40 mm. of Hg.		Distension of 60 mm. of Hg.		Distension of 80 mm. of Hg.		Distension of 100 mm. of Hg.		Distension of 120. mm of Hg.	
		L.P. in Secs.	Change of B.P. in mm. of Hg.	L.P. in Secs.	Change of B.P. in mm. of Hg.	L.P. in Secs.	Change of B.P. in mm. of Hg.	L.P. in Secs.	Change of B.P. in mm. of Hg.	L.P. in Secs.	Change of B.P. in mm. of Hg.
1.	110	8	+6	8	+8	9	+10	9	+10	9	+12
2.	150	4	−14	5	−16	6	+18	6	−18	6	−18
3.	130	—	—	5	+10	5	+12	—	—	—	—
4.	120	8	+8	4	+6	8	+6	9	+12	6	+16
5.	210	9	−2	8	−4	10	+2	10	+8	10	+6
6.	200	5	−2	6	−4	6	−2	7	+2	6	+1
7.	170	5	−1	4	+2	5	+2	4	+4	6	+10
8.	150	6	+2	8	+2	8	+6	7	+6	7	+10
9.	150	4	+6	3	+12	5	+14	5	+10	3	+20
10.	160	3	+6	2	+6	3	+6	3	+10	4	+10
11.	170	3	+4	0	0	3	−2	4	+4	4	+6
12.	160	¾	—	8	+8	8	+12	8	+14	8	+16
13.	240	3	−2	3	−4	3	−8	3	−8	3	−2
14.	200	5	−2	0	0	0	0	0	0	5	+2
15.	180	8	+1	8	+2	8	+2	8	−10	—	—
16.	170	2	−2	3	−4	3	−8	3	−8	3	−6

When the filling was discontinued and bladder kept distended (which in our case was possible only for 1 min.), the blood pressure remained at a level which varied with intravesical pressure and it came down only after release of pressure. This is evident from Fig. 1.

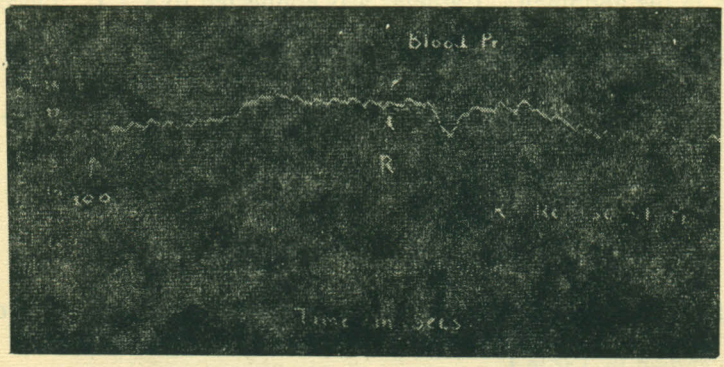

Fig. 1. Record of B.P. showing the effect of maintainance of intravesical pressure of 100 m.m. of Hg.

The extent of rise of B.P. was related to the degree of distension of bladder but it was also observed that even when intravesical pressure was high many times, vaso-pressor

responses were absent and sometimes even reversed. Table 2 gives an idea about the relationship of intravesical pressure and rise of B.P.

TABLE 2

Intravesical pre. in m.m. of Hg.	No. showing rise of B.P.	Arith. mean rise of B.P. in m.m. of Hg.	Standard Deviation	Standard error
40	7	4.71	2.564	0.9690
60	9	5.55	1.871	0.6236
80	10	6.00	3.662	1.149
100	11	7.27	2.549	0.7685
120	12	9.42	6.140	1.773

The results of the above table are shown graphically below. The graph shows linear relationship between distension and rise of B. P. within limits. Above 100 m.m. of Hg. intravesical pressure, there is great rise of B.P.

In some cases we observed the fall of B.P. instead of rise and in such cases it was found that original normal B.P. of dogs was higher than 170 m.m. of Hg, except in one case in which it was 150 m.m. of Hg. This is evident from Fig. 2.

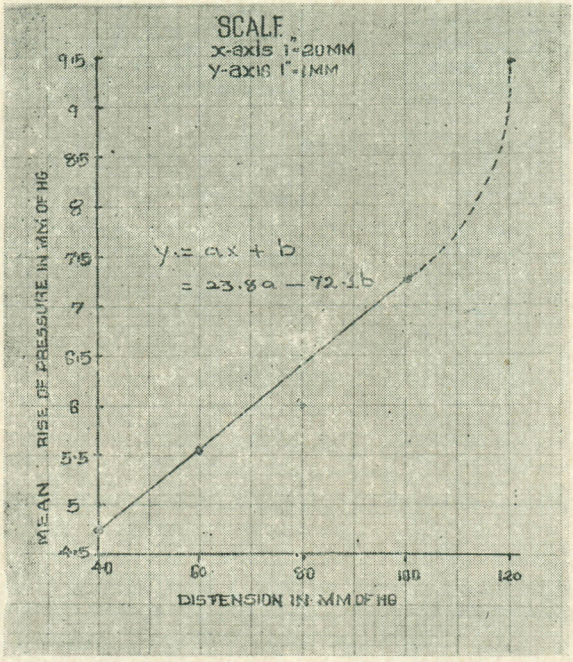

Fig. 2.

In some cases it was observed that rise of B.P. was preceeded by fall, and the fall was preceeded by a rise in some other cases as shown by following Fig. 3 & Fig. 4 respectively.

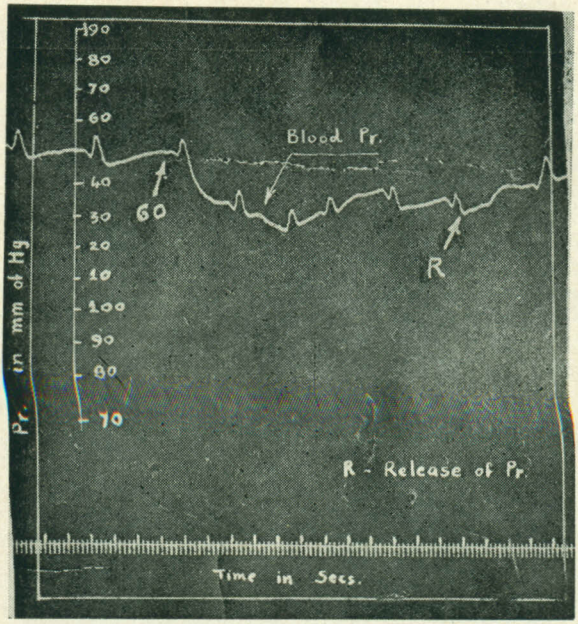

Fig. 3. showing the fall of B.P. Intravesical pressure raised to 60 m.m. of Hg.

In one case B.P. was recorded after distension during deep and light anaesthesia and it was observed that during deep anaesthesia there was fall of B.P. and during light anaesthesia there was rise of B.P.

In one case respiration was recorded with B.P. and it was found that with every distension there was apnoea followed by hyperpnoea.

DISCUSSION

Distension of urinary bladder causes rise of B.P. This confirms similar observations of Mukherjee (4) in cats. He found maximum rise of 12 m.m. of Hg. which was 12% of normal. We found maximum rise of 20 m.m. of Hg. which was 13.2% of normal. Guttmann and Whittridge (3) found a rise of 20-30 m. m. of Hg. in paraplegic patients. Lag in our series was 2-10 secs as compared to that of Mukherjee (4) who found 2-40 secs. Relationship between intravesical pressure and rise in B. P. confirms the findings of Mukherjee (4) in cats, of Watkings (1938 in non-spinal cats, and of Guttmann and Whittridge (3) in chronic spinal man.

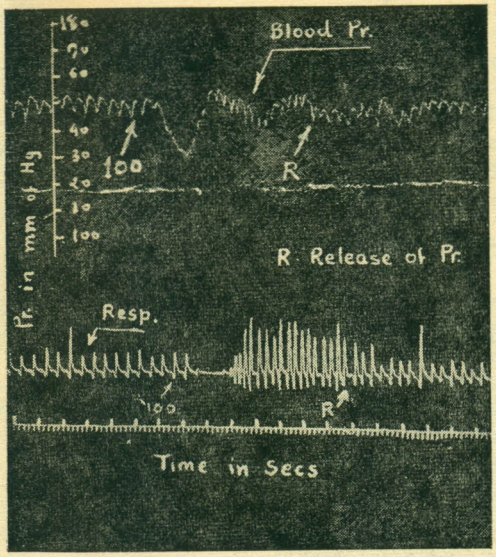

Fig. 4

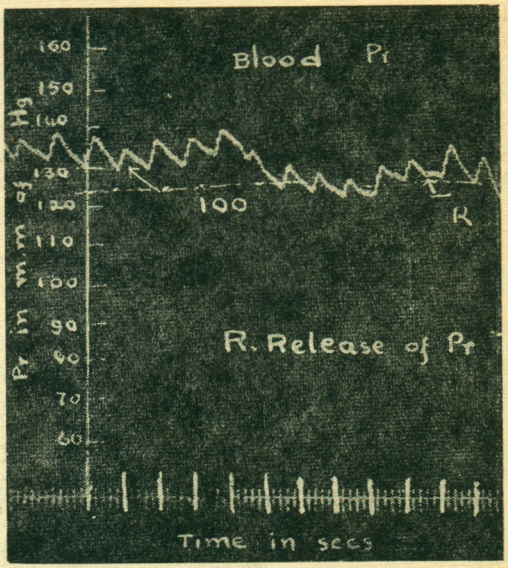

Fig. 5

No rise in B. P. was observed in some animals. It was explained by Mukherjee as due to haematoma in bladder when rapid rate of filling or very high head of pressure was used.

Fall of B. P. observed in few cases can be explained as : (i) in one dog in deep anaesthesia there was fall of B. P. and in light anaesthesia there was rise of B. P. Hence it may be effect of anaesthesia. (ii) Shukla, Agarwal and Kapoor (5) have obtained similar results on distension of duodenum. They explained it by assuming two types of receptors; pressor and depressor. We may explain our results on similar basis. (iii) As fall of B. P. was obtained in cases whose normal B. P. was higher than 179 m. m. of Hg, it is likely that receptors are not active at such high pressure. It has been proved that at very high and very low normal pressures, more distending force is required. Fall preceeding the rise can be explained by observation of Brodie and Russel (1) during distension of stomach where there was a rise preceeding the fall.

EFFECT OF ANAESTHESIA:—In our series normal B. P. of most of the dogs was high. This was because barbiturates depress the activity of vagus and sino-aortic mechanism.

By recording B. P. in light and deep anaesthesia in the same dog we found that fall during deep anaesthesia, was converted to rise during light anaesthesia on distension of bladder. This confirms that barbiturates in bigger dose can paralyse the presso-receptors but Nembutal differs in that it has less depressant activity compared to other barbiturates.

Even when maintaining intravesical pressure for ½ to 1 min, the B. P. did not fall indicating that presso-receptors do not adapt or adapt very slowly.

Because rise is very small in intact animal, these viscero-vascular reflexes are insignificant. Besides, such high rise of pressure is not common in normal individuals. But in certain abnormal conditions like spinal lesions this reflex may become an important reflex in making B. P. labile. At the same time these reflexes explain how organisms have mechanism to adjust their blood flow; when distension occurs beyond 50 m. m. of Hg, likelihood of stoppage of blood supply is prevented by rise in B. P.

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