

LETTER TO EDITOR

BLOOD PRESSURE OSCILLATIONS OF CEREBELLAR ORIGIN

Sir,

Blood pressure oscillations are of two types : one caused by respiration, known as Traube Hering waves and the second which are independent of respiration known as Mayer waves. The latter type at times appear spontaneously under normal circumstances (6), but are usually seen in certain abnormal states. Asphyxia, intracranial hypertension (3), haemorrhage (2), metabolic acidosis (4) and high levels of circulating acetylcholine or angiotensin (10) are among the conditions known to cause Mayer waves. These are regularly occurring slow waves of different forms unrelated to respiratory activity.

In the previous study where cerebellum was explored in relation to gastrointestinal motility (12), this interesting phenomenon of blood pressure waves was observed frequently. In those chloralose anaesthetised cats (50 mg/kg i/v), Mayer waves appeared spontaneously in quite a few animals. Four observations were made where these existing waves got intensified on electrical stimulation of nodule ($5\text{ V}, 5\text{ msec}, 100/\text{sec}$) (Fig. 1). Both amplitude and frequency

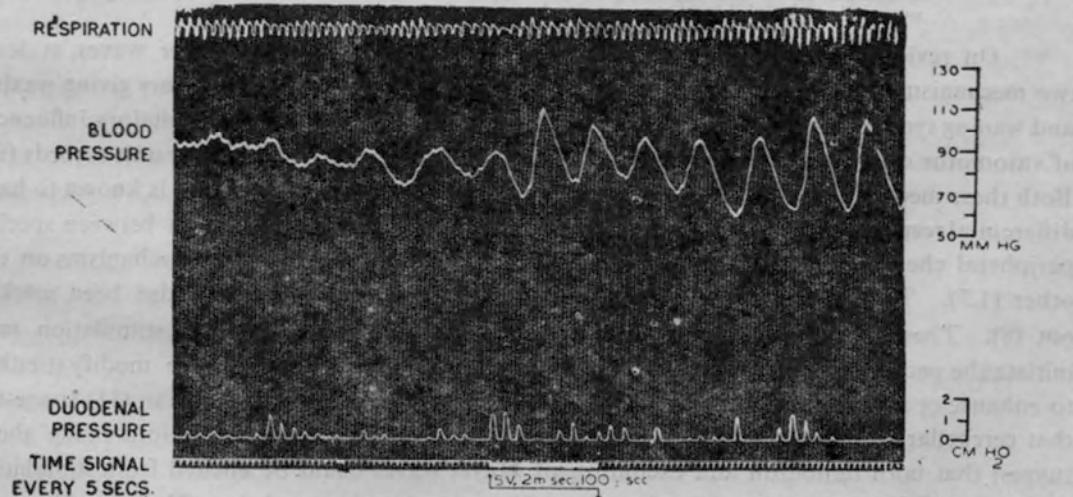


Fig. 1: A kymographic record showing intensification of already existing Mayer waves on electrical stimulation of uvula. Traube-Hering waves synchronous with respiration and superimposed on Mayer waves became inconspicuous after stimulation. Duodenal motility and respiration showed no change.

showed an increase. Peak to peak fluctuation was 40 mm Hg and frequency $1.5-2/\text{min}$ as compared to 15 mm Hg and $0.7-1/\text{min}$ before stimulation. Even the Traube Hering waves super-

imposed on Mayer waves became less conspicuous during post stimulatory phase. These waves lasted for several minutes (1/2 hr in one case) after cerebellar stimulation. This type of response was also observed in two cats where uvula and fastigial nucleus were stimulated. In another cat stimulation of uvula led to inhibition of Mayer waves (Fig 2.) This was concomitant with slight rise in blood pressure. The post stimulatory average blood pressure showed persistent rise but without these waves. As is apparent from the figures, electrical stimulation of these cerebellar regions did not show changes in other parameters like GI motility and respiration.

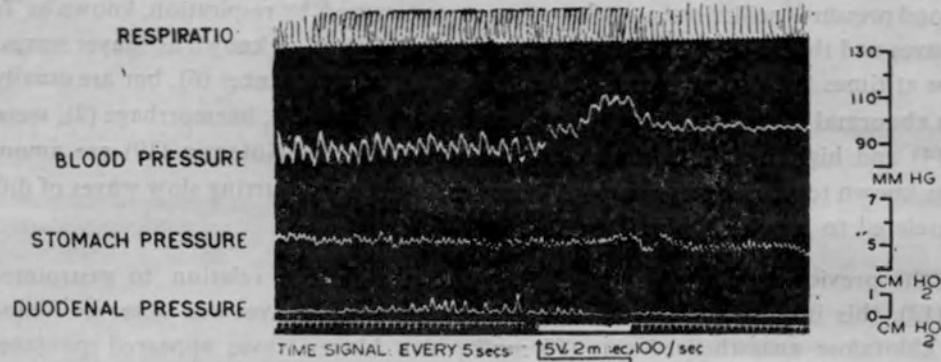


Fig. 2: Shows inhibition of Mayer waves and rise in mean blood pressure of 10 mm Hg on stimulation of nodule. Spontaneously appearing Mayer waves with frequency 9/min, peak to peak amplitude of 15 mm Hg completely disappeared after stimulation. Duodenal pressure waves also got inhibited in post stimulatory period.

On reviewing literature regarding mechanism of production of Mayer waves, at least two mechanisms have been suggested : periodic activity of chemo & baroreceptors giving waxing and waning synchronising input to the vasomotor centres (2,5) or the direct oscillatory influences of vasomotor centres on the pre-ganglionic sympathetic neurones, as shown by unit records (9). Both these mechanisms could be influenced by the cerebellum more so when it is known to have differential response pattern in cardiovascular dynamics, through its interaction between specific peripheral chemo and baroreceptors on one hand and central vasomotor mechanisms on the other (1,7). The pathways for these cerebello-medullary projections have also been worked out (8). Therefore, the observations made above, suggest that cerebellar stimulation may initiate the periodicity and synchronised activity of the vasomotor neurones or modify it either to enhance or inhibit these waves. In their ablation studies Ramu and Bergman (11) suggested that cerebellar vermis was inhibitory to Mayer waves but couple of observations made above suggest that both inhibition and excitation of Mayer waves could be elicited from stimulation of vermis and fastigial nucleus stimulation can also sometime trigger their production. However, more experiments are needed to explore various regions of cerebellum, in this context.

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REFERENCES

1. Achari, N.K., and C.B.B. Downman. Autonomic effector responses to stimulation of nucleus fastigius. *J. Physiol.*, **210** : 637-650, 1970.
2. Anderson, B., R.A. Kenney and E. Neil. The role of the chemoreceptors of the carotid and aortic regions in the production of Mayer waves. *Acta Physiol. Scand.*, **20** : 203-220, 1950.
3. Cushing, H. Concerning the definite regulatory mechanisms of the vasomotor centre which controls blood pressure during cerebral compression. *Bull. Johns Hopkins Hosp.*, **12** : 292, 1901.
4. Ferretti, R., N.S. Cherniack., G. Longobardo., O.R. Levine., E. Morkin, D. H. Singer and A.P. Fisherman. Systemic and Pulmonary vasomotor waves. *Am. J. Physiol.*, **209** : 37-50, 1965.
5. Guyton, A.C. and J.W. Harris. Pressoreceptor-autonomic oscillations: a probable cause of vasomotor waves. *Am. J. Physiol.*, **165** : 158-166, 1951.
6. Guyton, A.C. and J.H. Satterfield. Vasomotor waves possibly resulting from CNS ischaemic reflex oscillation. *Am. J. Physiol.*, **170** : 601-605, 1952.
7. Lisander, B. and J. Martner. Interaction between the fastigial pressor response and the baroreceptor reflex. *Acta Physiol. Scand.*, **83** : 505-514, 1971.
8. Miura, M. and D.J. Reis. The paramedian reticular nucleus; a site of inhibitory interaction between projection from fastigial nucleus and carotid sinus nerve acting on blood pressure. *J. Physiol.*, **216** : 441-460, 1971.
9. Polosa, C., P. Rosenberg, A. Mannard, N. Wolkow and I. Wyszogrodski. Oscillatory behaviour of the sympathetic system induced by picrotoxin. *Can. J. Physiol. & Pharmacol.*, **47** : 815-826, 1969.
10. Race, D. and M. Rosenbaum. Non-respiratory oscillations in systemic arterial pressure of dogs. *Circulation Res.*, **18** : 525-533, 1966.
11. Ramu, A. and F. Bergman. The role of the cerebellum in blood pressure regulation. *Experientia*, **23** : 383-384, 1967.
12. Tandon, O.P. Exploration of cerebellum in relation to gastrointestinal motility. M.D. Thesis, All India Institute of Medical Sciences, New Delhi, 1971.