and the various morphological observations and growth data were recorded at each harvest.

The plants of the set receiving 0.006 *M* NaCl in addition to normal ARNON and HOAGLAND's solution showed good vegetative growth and dark green leaves as compared with the control set. There was no indication of any toxic effect on the vegetative growth of the plants. At first and second harvest, the leaves in sodium set showed more dry weight than the control (Table). This increase in dry weight is in agreement with the results of BROWNELL[11] and others[12,13].

A marked effect on pod development was, however, observed. Quite a large number of pegs failed to develop into pods, the tips of the gynophore turned brown and showed necrotic symptoms (Figures 1 and 2). Those pegs which developed into pods, were smaller and with necrotic lesions. It would appear that the presence of Na^+ ions around the pegs inhibits the development of the pod. It may be that the Na^+ ions inhibit the uptake of Ca^{++} ions by the pegs or the growing pods. That calcium inhibits the rate of Na^+ absorption is known, and an interaction between the 2 ions in pod formation appears plausible. The observation that the vegetative growth of the plants is not affected by Na^+ might indicate that, at the root level, sodium may not interfere with the uptake of calcium, but that the calcium uptake directly by the developing pods is inhibited by the Na^+ ions[14].

Fig. 1. Entire plants of peanut (IV harvest, 70 days) showing inhibition of pod development, but no toxic effect of NaCl, on the vegetative growth, as compared to the control plants.

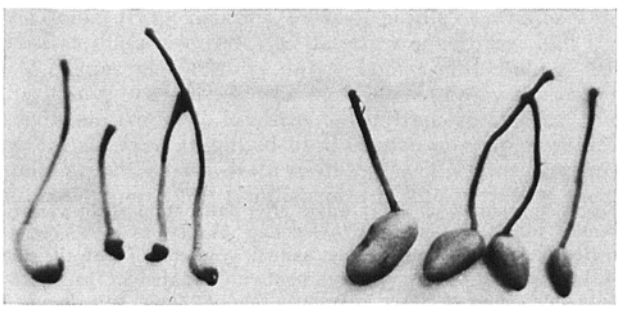

Fig. 2. Pods showing black necrotic lesions, in presence of NaCl, along with normal pods from control plants.

Zusammenfassung. Natrium Chloratum (0,006 *M*) hemmt das vegetative Wachstum der Erdnusspflanze nicht, wohl aber die Entwicklung der Hülsenfrüchte selber. Es wird angenommen, dass die Natrium-Ionen die Calciumaufnahme an der Wurzel nicht stören, dass sie aber von den sich entwickelnden Hülsen in der Nähe der Natrium-Ionen direkt gehemmt wird.

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Department of Botany, University of Allahabad (India), 22nd November 1966.

[12] P. M. HARMER and E. J. BENNE, Soil Sci. *60*, 137 (1945).
[13] J. J. LEHR, J. Sci. Fd Agric. *4*, 460 (1953).
[14] This research was supported by the grant from U.S. PL-480 Scheme No. FG-In-232, which is thankfully acknowledged.

The Role of the Cerebellum in Blood Pressure Regulation

In previous papers from this laboratory, the distribution of pressor and depressor neurons in the brain stem was studied by means of the transection method[1,2]. It was shown that the pressor response to peripheral nerve stimulation is reversed to blood pressure fall by cutting the brain at a critical level. The latter was found in the cat at an inter- to post-collicular plane, while in the rabbit it was placed somewhat more rostrally, viz. at a pre- to intercollicular level.

It was however recognized that intercollicular transections destroy also the connections of the anterior brain stem with the cerebellum[2]. Therefore the role of this structure in vasomotor reflexes requires clarification. It is known that stimulation of various parts of the cat's cerebellum induces blood pressure rises, the most sensitive region being the fastigial nucleus[3]. We have confirmed these observations by probing systematically the cerebellum of the conscious rabbit with bipolar concentric electrodes. From about 60 points, scattered through all parts of the cerebellum, only hypertensive responses could be evoked, but we have been unable to obtain any depressor reaction. This result is in apparent disagreement with the findings of ZANCHETTI and ZOCCOLINI[3] on the thalamic cat, where sometimes a blood pressure fall was encountered when the cerebellum was stimulated in the interval between 2 sham rages. On the other hand, exci-

[1] U. LEIBOWITZ, F. BERGMANN and A. D. KORCZYN, Archs int. Physiol. *71*, 662 (1963).
[2] F. BERGMANN and A. D. KORCZYN, Isr. J. med. Sci. *1*, 979 (1965).
[3] A. ZANCHETTI and A. ZOCCOLINI, J. Neurophysiol. *17*, 475 (1954).

tation of certain cerebellar areas, while not leading to any direct circulatory change, may inhibit the blood pressure rise evoked by carotid occlusion or by brain stem stimulation[4,5].

The role of the cerebellum in vasomotor regulation became evident after cerebellectomy. This procedure converted the hypertensive reactions of the rabbit, elicited by peripheral nerve stimulation, into blood pressure fall (Figure 1). The change was complete, i.e. depressor responses followed stimulation over the whole frequency range used, from 5–300 pulses/sec. It appears possible, therefore, that intercollicular transection in the rabbit causes reversal of pressor reactions by breaking the connections between hypothalamus and cerebellum. Partial reversals, described in earlier papers[1,2], may be due to incomplete destruction of these connections. It should be recalled that intercollicular cuts leave the links of more caudal parts of the brain stem with the cerebellum intact.

The effect of cerebellectomy on vasomotor reflexes is also in agreement with recent observations on the influence of barbiturates. The hypertensive reactions evoked by cerebellar stimulation of the conscious animal, were abolished by small subnarcotic doses of the drug (e.g. 5–10 mg/kg of pentobarbitone)[6]. On the other hand, we have reported that such small doses of the barbiturate produce reversal from pressor to depressor responses when peripheral nerves[6] or brain stem structures are stimulated[7]. From these 2 series of experiments, it may be concluded that small amounts of narcotic suppress the propagation of vasomotor impulses in which the cerebellum participates.

A further interesting phenomenon was observed when only the vermis was ablated. Meyer waves now became prominent or – if present before the operation – were markedly intensified (Figures 2a and b). The waves continued indefinitely, i.e. until death, and were usually so strong as to overshadow any other vasomotor response evoked by peripheral or cerebellar stimulation.

However, the Meyer waves disappeared completely after extirpation of the rest of the cerebellum (Figure 2c). It is suggested that the cerebellum contains 2 regions influencing these waves: One which enhances them or may even be responsible for their appearance, and a second area with inhibitory action. The main localization of the inhibitory elements appears to be in the vermis. The ability of many drugs to intensify the Meyer waves may thus be due – inter alia – to blockade of these inhibitory neurons.

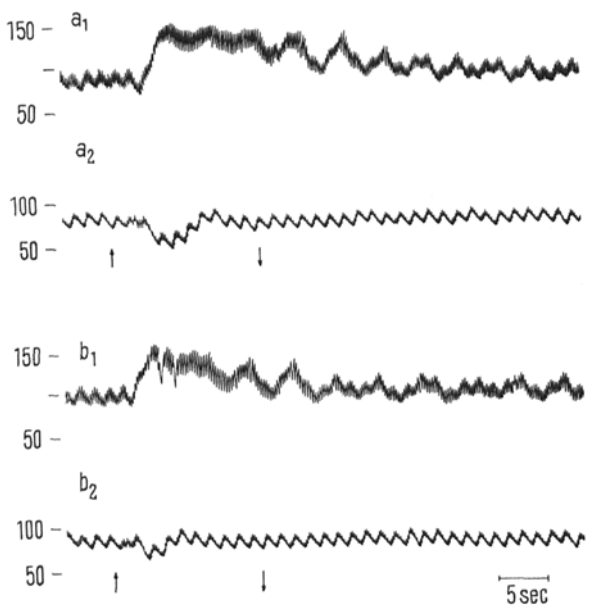

Fig. 1. Reversal of blood pressure response to peripheral nerve stimulation by extirpation of the cerebellum. Male rabbit, 3.4 kg. Continuous infusion of succinylcholine, 0.02 mg/kg/min, into left femoral vein; artificial ventilation. Blood pressure recorded from right carotis on an E & M physiograph by means of a Bourdon type photoelectric transducer. Right sciatic nerve stimulated via a constant current supply at 0.5 mA and 1 msec pulse duration for periods of 15 sec (as indicated by arrows). Blood pressure scale (in mm Hg) on left side; time marking (5 sec) in right lower corner. Rectal temperature was 37 °C. a_1, intact animal; stimulation at 20 pulses/sec. a_2, same, after extirpation of the cerebellum by suction. b_1 and b_2 same for 100 p/sec. Note stronger blood pressure fall in a_2 than in b_2.

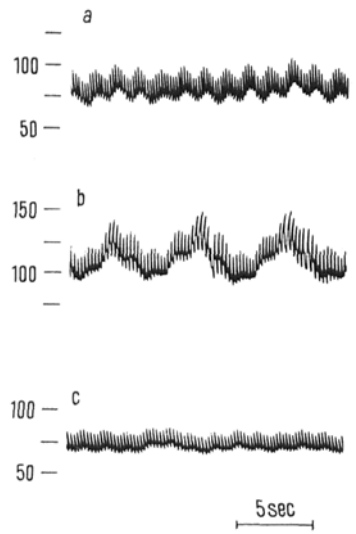

Fig. 2. Appearance of Meyer waves after extirpation of the vermis. Female rabbit, 2.3 kg. Succinylcholine infusion as in Figure 1. a, Hering-Traube waves of intact animal, synchronous with the rate of the respiratory pump (42/min). b, after extirpation of the vermis. Note appearance of Meyer waves at a rate of 12/min, superimposed on the Hering-Traube waves. c, after complete cerebellectomy. Note absence of Meyer waves, but presence of weak Hering-Traube waves. Average blood pressure in (a) is 80 mm, in (b) 120 mm and in (c) 75 mm. Elevation of blood pressure level after removal of the vermis was not a constant finding.

Résumé. La stimulation du cervelet du lapin produit seulement des réactions hypertensives. Après la cérebellectomie, la stimulation périphérique provoque – au lieu d'une élévation – une chute de la pression sanguine. Les ondes de Meyer s'intensifient après l'extirpation du vermis et disparaissent après la cérébellectomie totale.

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Department of Pharmacology, The Hebrew University, Hadassah Medical School Jerusalem (Israel),
6th December 1966.

[4] G. MORUZZI, J. Neurophysiol. *3*, 20 (1940).
[5] B. HOFFER, R. RATCHESON and R. S. SNIDER, Fedn Proc. Fedn Am. Socs exp. Biol. *25*, 701 (1966).
[6] J. GUTMAN, F. BERGMANN and M. CHAIMOVITZ, Archs int. Physiol. *69*, 509 (1961).
[7] J. GUTMAN, M. CHAIMOVITZ, Y. GINATH and F. BERGMANN, Archs int. Physiol. *70*, 33 (1962).