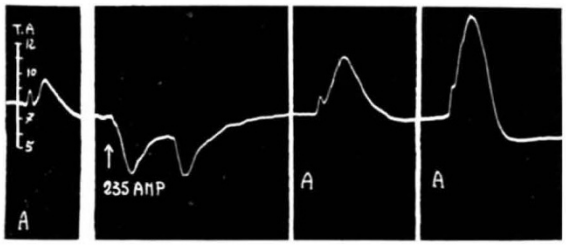


Fig. 1. Blood pressure record. *A*, Application of adrenaline on the fronto-parietal cortex of a rabbit. After intravenous injection of 235 *ANP* (50 mgm./kgm.) one can observe a considerable increase of the hypertensive reaction

regulator acids exert their stimulation. So far three possibilities have been examined :

(*a*) The potentialization of the central action of adrenaline by a stimulation of the cerebral cortex induced by the drug. (*b*) Direct action on the hypothalamus and hypophysis or on the level of catecholamines and serotonin, which are found in great quantities in this region. (*c*) Regularization by 235 *ANP* of the metabolic activity of the hypothalamic region could explain some of the powerful actions of the drug.

Pharmacological and biochemical studies are now being carried out with the view of explaining the mechanism of action of 235 *ANP* and other esters of plant-growth regulator acids, which seem to act as metabolic regulators at the level of the hypophyseal–hypothalamic region.

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## HÆMATOLOGY

### Glutathione Stability Test in Hæmoglobin *E* – Thalassæmia Disease

Hæmoglobin *E* – thalassæmia disease represents a double heterozygous state with reference to genes for hæmoglobin *E* and thalassæmia. Studies on glutathione stability which have recently provided valuable information regarding the cause of hæmolysis as it occurs in drug-induced hæmolytic anæmia and in hereditary non-spherocytic hæmolytic anæmia have not so far been done in hæmoglobin *E* – thalassæmia disease. In the present communication, 20 cases of hæmoglobin *E* – thalassæmia disease were investigated with reference to the behaviour of their erythrocytically reduced glutathione-level when incubated *in vitro* with acetylphenylhydrazine. The relevant features of these 20 cases are given in Table 1.

Table 1. Relevant Features of Twenty Cases of Hæmoglobin *E* – Thalassæmia Disease

| | Mean | Range |
|---|---|---|
| Age (yr.) | 5·8 | 1–28 |
| Hæmoglobin (gm. per cent) | 6·44 | 3·4– 9·28 |
| Hæmoglobin *F* (per cent) | 23·8 | 13·0–50·0 |
| Reticulocytes (per cent) | 8·1 | 2·1–17·0 |

The method employed was essentially the same as originally evolved by Beutler[1]. In order to increase the sensitivity of the test, 4 mgm. of glucose was added to 1 ml. of blood as suggested by Szeinberg *et al.*[2] and later adopted by Beutler[3]. With this method, the range of reduced glutathione destruction in the normal Bengalee population varied from 1 to 10 mgm. per 100 ml. of erythrocytes. Two apparently normal persons, however, showed unstable glutathione-level, indicating that a certain proportion of Indians has such glutathione instability similar to the state of instability reported first in people of negroid origin[5,6] and later in those of Syrian, Iranian and Jewish[2] extraction. In contrast to the normal findings, the pattern in hæmoglobin *E* – thalassæmia disease was different (Table 2). In 12 out of 20 cases, the glutathione destruction exceeded 30 mgm. per 100 ml. of erythrocytes and in 9 the post-incubation level was lower than 30 mgm. In all the patients, both sensitive and non-sensitive, glucose-6-phosphate dehydrogenase activity of red cells was, however, found to be high.

Table 2. Results of Glutathione Stability Test in Normals and in Hæmoglobin *E* – Thalassæmia Disease

| | No. of subjects | Reduced glutathione-level in mgm. per 100 ml. of erythrocytes | | | |
|---|---|---|---|---|---|
| | | Range | | Mean | *S.E.* |
| Non-sensitive normals | 20 | Before | 47–95 | 65·05 | 2·1483 |
| | | After | 40–89 | 61·60 | 2·6173 |
| Non-sensitive hæmoglobin *E* – thalassæmia | 8 | Before | 19·6–92 | 54·48 | 8·0291 |
| | | After | 17·6–92 | 51·70 | 7·4065 |
| Sensitive hæmoglobin *E* – thalassæmia | 12 | Before | 41·5–82 | 63·275 | 1·3039 |
| | | After | 0–50 | 27·5 | 5·2448 |

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### Blood Groups of Selected Aboriginal and Indigenous Populations

The Laboratory of Physical Anthropology at Yale is investigating the distribution of the following genetic traits in various populations of Primates, including man : the blood groups, the serum transferrins (β-globulins), haptoglobins, and hæmoglobin