Mechanism of Bronchial Constriction in Asthma. R. S. Jones (Institute of Child Health, Liverpool). The lability index was measured in 24 normal subjects aged 20–35 years and found to be between 4 and 21% with a mean of 12%. The lability index measures the tendency of the bronchi to dilate and constrict, using the FEV1 as an index of airway resistance. Figures less than 20% are regarded as normal. On another day each subject was given 100 mg. of propranolol by mouth, 40 minutes before a repeat measurement of lability. There was a significant increase in lability for the group as a whole (range 6–42%; mean 18%; p < 0·01). When the criteria for defining asthma in terms of lability were applied, 8 subjects had moved into the asthmatic range. The difference in lability for this group, with and without propranolol, was highly significant (p < 0·01). In the group formed by the remaining 16 subjects, there was no significant difference. The pattern of bronchoconstriction after exercise in the group of 8 was exactly similar to that found in asthma.

In the normal subject at rest, muscle cell receptor activity causing relaxation (R) must exceed receptor activity causing constriction (C), since the bronchioles are almost fully dilated and stable. No constriction occurs after β-blockade at rest, so R must still exceed C despite the smaller value of R.

On exercise, when constriction occurs after blockade, C must exceed R. Hence, enhanced activity (C) of undefined receptors must occur on exercise. In the absence of blockade, this activity results in minimal or no bronchoconstriction because it is opposed by the intact adrenergic mechanism.

The fact that 40% of asthmatics develop constriction at rest after propranolol indicates that they are dependent upon β-receptor activity for the prevention of constriction to a degree which the normal subject is not dependent. β-receptor activity is probably enhanced in the asthmatic therefore, but it may not be sufficient to maintain full dilatation at rest. β-receptor activity in these is presumably opposed by constrictor receptors activated by histamine or 'H'-like substances.

Post-exercise bronchoconstriction in asthma may not be due to an abnormal mechanism during exercise, but to the normal constrictor mechanism on exercise operating on a bronchus which is less stable than normal due to histamine or 'H'-like substances.

The phenomenon of abnormal lability, which is the determinant of clinical asthma, may therefore depend upon two mechanisms: (1) constriction due to activation of receptors by substances released after an allergic reaction, and (2) an inherently less stable bronchial tree which renders the individual vulnerable should an allergic reaction occur.

Muramidase (Lysozyme) Excretion in Children. T. M. Barratt and R. Crawford (Department of Immunology, Institute of Child Health, London). (Introduced by J. Lloyd). Lysozyme is a low molecular weight protein (14,000) that is synthesized by granulocytes and liberated.