to take place in supraoptic nucleus and median eminence in photosensitive birds exposed to prolonged daily photo-
periods4,10, and correlated to an increased gonadotropin release.

As dehydration implicates antidiuretic hormone release associated with depletion of neurosecretory material, a similar parallel can be drawn between the augmented phosphatase activity and the liberation of the octapeptide over strengthened by the fact that the breakdown of neurosecretory cells, This relation seems more-
that the release of neurosecretory material is made
through a holocrine or apocrine mechanism, the increased
systems of several species, including the rat x, x, suggest
present paper, provides a reliable biochemical basis for
these morphological findings.

**The Role of the Pentose-Phosphate Pathway in Adjustment of the Heart to a High Load and the Development of Myocardial Hypertrophy**

The compensatory hypertrophy represents an essential factor in adjustment of the heart to a prolonged increase of physiological load in deficiency, hypertension and intense physical work. The development of the compensa-
tory hypertrophy proceeds on the base of a significant increase of the synthesis of nucleic acids and proteins in the myocardium. This ensures not only a timely replace-
ment of worn myocardial structures but also a rapid augmentation of the mass of the myocardium1.

Apparently the activation of the biosynthesis of nucleic acids and proteins in the myocardium in its hyper-
function is reached by way of coordinated increase of activity of a number of fermentative systems. It may be suggested that an important role in realization of these processes must belong to pentose phosphate pathway as far as this metabolic process is the fundamental source of reduced NADP-H₂ and riboso-5-phosphate for the nucleic acids and protein synthesis.

In this connection, in the present work the change of activity of one of the most important fermenters of the pentose pathway, transketolase, in the hyperfunction of the heart has been studied. Simultaneously the study of action of the antagonist of vitamin B₁₂ - oxihtamine, on the transketolase activity, protein synthesis and hyper-
trophy of the heart in its hyperfunction has been carried out.

The compensatory hyperfunction of the heart was experimentally produced in rabbits by creation of stenosis of the aorta with the earlier described method3 ensuring a persistent narrowing of the transverse section of the aortic lumen 3 times. The activity of the transketolase was determined by the method of Bruns2 in control animals and in rabbits with hyperfunction of the heart 2 and 45 days following creation of stenosis.

As seen from Table I, 2 days following the onset of hyperfunction when the processes of biosynthesis of nuclear acids and proteins3,4 in the myocardium are sharply intensified, the activity of transketolase in this organ is increased by more than 60%. Following 45 days when the process of hypertrophy is essentially completed, the transketolase activity decreases, approaching the normal level.

Apparently, these changes are specifically connected with the compensatory hypertrophy of the heart. They take place only in the myocardium and are lacking in other tissues, for instance in blood.

The close correlation between the activity of transketolase and the intensity of development of the myocardial hypertrophy is revealed in the analysis of data relating to different animals at the initial stage of hyper-
function and hypertrophy of the heart. From the Figure it is seen that the higher the activity of transketolase in the myocardium of the animal, the more increased the relative weight of the left ventricle, i.e. the more intense the development of the process of hypertrophy.

This suggests that activation of the transketolase is apparently one of the links of the biochemical mechanism of hypertrophy of the myocardium in its compensatory hyperfunction.

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1 F. Z. MEERSON, Myocardium in Hyperfunction, Hypertrophy and Insufficiency of the Heart (Moscow 1965).
The increase of the synthesis of nucleic acids and proteins in the myocardium at the initial stage of hyperfunction underlying its hypertrophy certainly requires an increased inflow of precursors, in particular that of riboso-5-phosphate produced in the course of pentose-phosphate way of splitting of the carbohydrates. The majority of the riboso-5-phosphate necessary to the cell is formed from glucose-6-phosphate in the non-oxidative reactions of pentose-phosphate way catalyzed by transketolase and transaldolase. The evidence obtained of the increase of activity of transketolase in the hypertrophied myocardium together with the data presented by other authors on the low activity of transketolase as compared to other ferments of the pentose-phosphate way, suggest that transketolase limits the rate of formation of riboso-5-phosphate. In the normal myocardium, the capacity of this enzyme seems to ensure the current requirements of metabolic processes in riboso-5-phosphate, but it does not create any reserve sufficient for a considerable increase of production of this metabolite.

In this connection, the increase of activity of the transketolase at the initial stage of hyperfunction of the heart apparently represents an indispensable condition for the increase of production of riboso-5-phosphate and the intensity of nucleic acids and protein synthesis for the replacement of worn structures and a simultaneous increase of the absolute mass of the myocardium.

Such a concept of the role of the transketolase is in agreement with the results obtained in the experiment with the antagonist of vitamin B₁₂-oxythiamine. As shown by the data presented in Table II, injection of oxythiamine in doses of 100-400 mg/kg daily to rabbits with experimental aortic stenosis inhibits the activity of transketolase and at the same time abolishes the activation of protein synthesis, thus essentially slowing down the myocardial hypertrophy.

**Table I. Change of transketolase activity in compensatory hypertrophy of the heart**

<table>
<thead>
<tr>
<th>Examined index</th>
<th>Control 45 days</th>
<th>Compensatory hypertrophy 45 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative weight of the left ventricle in %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activity of transketolase in the myocardium, of sedoheptulose-7-phosphate/hg of fresh tissue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activity of transketolase in blood of S-7-P/h/ml of blood</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of animals</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table II. The action of oxythiamine on the activity of the transketolase, incorporation of S³⁵-methionine into the proteins of the myocardium and the relative weight of the left ventricle in rabbits with experimental aortic stenosis**

<table>
<thead>
<tr>
<th>Examined index</th>
<th>Without oxythiamine</th>
<th>With oxythiamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activity of transketolase, of S-7-P/g/h</td>
<td>4.40 ± 0.05</td>
<td>6.22 ± 0.35</td>
</tr>
<tr>
<td>Relative specific activity of protein</td>
<td>3.20 ± 0.13</td>
<td>2.65 ± 0.25</td>
</tr>
<tr>
<td>Relative weight of the left ventricle, %</td>
<td>0.154 ± 0.005</td>
<td>0.192 ± 0.007</td>
</tr>
<tr>
<td>No. of animals</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>