of 200 mU or more, or an infusion of 500 mU/min. A single dose of 600 mU caused maximal vasodilatation. The hand and forearm effects outlasted the fall of blood pressure by as much as 2 to 3 minutes. Doses of 500 mU oxytocin increased cardiac output by 9% to 42 % in different subjects. On injection into the brachial artery 20 mU produced a considerable vasodilatation in hand and forearm. All the effects of oxytocin decreased with repeated injections. The actions of oxytocin on hand, forearm and blood pressure were entirely abolished by simultaneous injection or infusion with the oxytocin of 1/20 the dose of vasopressin.

KLISIECKI, A.; GARBULINSKI, T. and GOSK, A.: **A new aspect of the depressor nerve reflex.** *(Ac. Med. Wroclaw. Poland.)*

According to Cyon, Ludwig and Bayliss the blood pressure fall in the depressor reflex is the consequence of the general vascular dilatation. The heart does not lose its strength neither vagi nor sympathetic nerves participate in this reflex. Unfortunately they could not present convincing evidence of the vasodilatation. Pletysmographs of Bayliss have discovered mere traces of vasodilatation in the limbs, while the kidney diminished or augmented imperceptibly its volume during the blood pressure fall.

Hoping to solve the problem, we measured the blood flow in the intestines (coeliac or mesenteric artery) simoultaneously with that in the femoral artery. Photohaemotachometer of Cybulski was used. Excitation of the central end of the depressor gives no evidence of any vasodilatation; the blood flow passively follows the changes of pressure.

Anywise some mysterious process must occur in the depressor reflex. The inseparable junction of the blood pressure fall with the volumes of examined organs points to displacement of certain amounts of blood from arteries into veins. Generally speaking the higher the pressure the more blood contain the arteries than the veins, and vice versa. Blood pressure mm Hg 40, 60, 80, 100, 120, 140.

Volume of blood in arteries (% of the whole blood) 5, 8, 10, 12, 13.6, 14,7.

When e.g. the pressure falls from 120 to 60 mm Hg. the volume of blood in arteries diminishes from 13,6 to 8 %. In this case 5,6 % of arterial blood are displaced into veins. Appropriate displacements take place in the various organs. It now becames clear, why the described changes of colour and of volume of the organs are so unsteady and microscopic. Essential for the Cyon and Ludwigs depressor reflex is the displacement of small amounts of blood from arteries into veins.

Depressor nerve does not dilate the blood vessels, but only decreases the heart work, what is aided by the fall of the pulse rate of vagal origin.

KNIGGE, K. M.; SOLOMON, D. H. and BIERMAN, S. M.: **Time relationships in the release of pituitary thyrotropin and thyroidal I^{131} after acute exposure of the hamster to cold *.** *(Dep. Anat. Med. School Med., Univ. Calif., Los Angeles and Neurophys. Lab., Veterans Administrat. Hos., Sepulveda, California, U.S.A.)*

In the hamster, a neurohumoral mechanism exists which is responsible for an acute, cold-induced acceleration of the rate of thyroidal I^{131} release. This accelerated release is dependent upon increased secretion of thyrotropin and requires the pituitary gland with its normal anatomical connections to the central nervous system. Experiments were performed to determine some of the time relationships involved in this phenomenon. The rate of thyroidal I^{131} release was determined by daily external counting during a control period (25°C) of 3-5 days. At 1 to 2 hour intervals after either thyrotropin (1 U.S.P. Unit, Thytropar) administration or exposure to cold (5°C), groups of animals were externally counted and then sacrificed to measure thyroidal I^{127} and pituitary thyrotropin content. In other experiments, thyroxine was administered at various times before and after cold exposure to block the accelerated thyroidal release.

The results indicate that pituitary thyrotropin, responsible for accelerated thyroidal release, is discharged during the first 3 hours of cold exposure; approximately 1 hour is required for the action of thyrotropin upon the hamster thyroid, as reflected by a significant increase in the rate of thyroidal I^{131} release. Exogenous thyroxine (10 µg) prevents the accelerated thyroidal release if administered before 3 hours of cold exposure have elapsed. If thyroxine is administered after this time, degree of I^{131} release or depletion of thyroidal I^{127} at the twelfth