Zusammenfassung

Die Aktivität der Azetylcholinesterase von einzelnen Megakaryozyten verschiedener Reifegrade wurde mit Hilfe des Cartesischen Tauchers bestimmt. Die Megakaryozyten zeigen auf dem Megakaryoblasten- und Promegakaryozytenstadium grosse Unterschiede der Azetylcholinesteraseaktivität. In der letzten Phase der Reifung, wenn die Zellen einen Durchmesser von 45 μ erreichen, wird das Ferment am stärksten wirksam.

Post-heparin Esterase in Man

It is known that heparin has an influence on the enzymatic activity of serum and plasma. The nature of these changes has already been investigated[1]. In the investigation described, the effect of heparin on the esterolytic activity of serum in man was observed. The esterase level was determined by the titrimetric method[2], using ethyl butyrate as substrate. Human blood serum was investigated before and 10–15 min after the injection of 5000 units of heparin intravenously. An increase of from 10 to 100% was found in 30 examinations. This finding was observed in both serum and plasma; heparin, however, acted only *in vivo*. When substrates other than ethyl butyrate were used, an increase was also observed with ethyl isobutyrate; with other esters, e.g. ethyl acetate, isopropyl acetate, butyl acetate, amyl acetate, triacetin and tributyrin, the increase was small or absent. The level of esterase rose as early as 2 min after injection, it then gradually fell; however a higher value was still evident after 60 min (Table I).

Table I

Time after injection minutes	Esterase ml 0·05 *n* NaOH
0	2·72
2	4·00
20	3·68
40	3·52
60	3·32

Table II

Concentration of NaF	Pre-heparin esterase ml 0·05 *n* NaOH	Post-heparin esterase ml 0·05 *n* NaOH
–	2·68	3·56
0·001 m	1·62	2·98
0·01 m	0·72	2·36
0·1 m	0·18	1·60
1 m	0·08	0·32

Post-heparin esterase acts at pH 7–11, a higher pH is more favourable to it. It is resistant to various inhibitors, e.g. NaF (Table II), physostigmine (10^{-3} m), diethyl-*p*-nitrophenylphosphate (10^{-5} m). Similarly, it shows a somewhat greater resistance to heat than the esterase of normal serum. Sodium taurocholate decreases its effectiveness. Similarly, protamine inhibits it *in vitro*. The effect of protamine, however, can be eliminated by the addition of heparin in excess.

J. SKOŘEPA and H. TODOROVIČOVÁ

*IV*th *Medical Clinic, Prof. B. Prusík, Charles University, Praha, March 12, 1956.*

Zusammenfassung

Es werden einige Eigenschaften der menschlichen Serumesterase nach Heparinapplikation beschrieben.

Tubular Factors in the Renal Response to Arterial Hypotension

The extreme oliguria in posthaemorrhagic hypotension is well known[1]. In a previous communication[2] we were able to demonstrate that the posthaemorrhagic diminution of diuresis is less expressed in the transplanted, i.e. completely denervated kidney, than it is in its innervated partner. So the role of nervous impulses in the mechanism of posthaemorrhagic oliguria seems to be proved. There is no doubt that *one* of the factors responsible for oliguria is the reduced filtration rate (GFR), but an accurate analysis of our data[3] suggested that an increase in tubular reabsorption is also involved in the process.

In a series of experiments performed on dogs under chloralose, innervated and transplanted kidneys were compared for renal bloodflow (RBF), GFR, renal resistance (R = B.P/RBF), and excretion of sodium and water. Part of the experiments were done in osmotic or saline diuresis, part of them without any diuretics at all. RBF was determined directly by cannulating the renal vein. GFR is the product of RPF (determined directly) and the extraction ratio of inulin. Measurements were made both in the basal state, i.e. with arterial pressure normal, and in hypotension induced by constriction of the aorta just above the origin of the renal arteries.

The results are tabulated (arithmetic means with s. d.). In the basal state there was no significant difference between the behaviour of the innervated and transplanted kidneys except for RBF, which was lower; consequently, resistance was higher in the transplanted kidney. After induction of a hypotension of about 70 mm Hg, RBF decreased to about 80% in both series, leading to a decrease of renal resistance. (The hypotensive values are expressed as percentages of the corresponding basal rates.) The response of the renal vessels to hypotension of the lower half of the body was

[1] S. W. LEVY and R. L. SWANK, J. Physiol. *123*, 301 (1954); *127*, 297 (1955). – D. K. MYERS, A. SCHOTTE, and B. MENDEL, Biochem. J. *60*, 481 (1955).

[2] I. S. CHERRY and L. A. CRANDALL, Amer. J. Physiol. *100*, 266 (1932).

[1] A. C. CORCORAN and I. H. PAGE, J. exper. Med. *78*, 205 (1943). – R. A. PHILLIPS, V. P. DOLE, P. B. HAMILTON, K. EMERSON, R. M. ARCHIBALD, and D. D. VAN SLYKE, Amer. J. Physiol. *145*, 314 (1946).

[2] P. BÁLINT, A. FEKETE, K. LÁSZLO, and G. PINTÉR, Acta physiol. Hung. *6*, 69 (1954).

[3] P. BÁLINT, A. FEKETE, A. HAYDU, K. LÁSZLO, and G. PINTÉR, Acta physiol. Hung. *6*, 81 (1954).

	Blood pressure	V/min	Na excr. aq./min	GFR	RBF	R
	Hypotension induced by constriction of the aorta					
Innervated series (16 exp.)	109 ± 11	2·32 ± 1·41	254 ± 178	54 ± 21	472 ± 136	0·25 ± 0·08
	70 ± 6	11 ± 10%	11 ± 12%	63 ± 27%	80 ± 38%	88 ± 14%
Transplanted series (26 exp.)	121 ± 10	3·45 ± 2·92	309 ± 224	49 ± 14	332 ± 140	0·42 ± 0·11
	74 ± 9	32 ± 31%	25 ± 21%	75 ± 33%	79 ± 16%	80 ± 13%
	Hypotension induced by bleeding					
Innervated series (34 exp.)	114 ± 16	2·31 ± 1·74	–	57 ± 24	464 ± 129	0·27 ± 0·31
	71 ± 7	18 ± 17%	–	62 ± 33%	55 ± 18%	130 ± 55%
Transplanted series (27 exp.)	117 ± 17	2·22 ± 1·66	–	39 ± 31	281 ± 171	0·49 ± 0·18
	75 ± 14	43 ± 33%	–	77 ± 31%	67 ± 18%	100 ± 34%

thus a vasodilatation in the innervated and transplanted kidney alike. This "autonomy" of the renal vessels has been described by various authors[4].

Although in hypotension GFR decreased to practically the same values in both series (35 for the innervated, 36 for the transplanted kidney), there was a significant difference (as computed by Fisher's "t"-test[5]) in the excretion of sodium and water. In the hypotensive period the innervated kidney excreted less sodium and water than the transplanted one. Since the filtration was equal, this difference can be attributed only to an enhanced tubular reabsorption of sodium and water by the innervated kidney.

According to our view, the renal response to hypotension of the lower half of the body consists of two effects. The first is an autonomous adaptation of the renal vessels in the form of vasodilatation, which is independent of innervation. The second effect is an enhanced reabsorption of sodium and water by the innervated tubuli, and this effect is absent in the transplanted kidney.

For comparison, in the lower half of the Table we present the data of our posthaemorrhagic experiments. In this case hypotension was induced in the whole animal. The decrease in RBF was more pronounced, and although resistance increased in the innervated kidney, it remained unchanged in the transplanted one. GFR, i.e. filtered lead was about equal (34 for the innervated, 30 for the transplanted kidney) and oliguria was significantly more pronounced in the innervated kidney. That means that the enhanced tubular reabsorption of water must play a role in the posthaemorrhagic oliguria too.

The renal response to bleeding is thus a combination of two effects. The first is the vasoconstriction in the kidney brought about by the hypotension of the upper half of the body (according to GÖMÖRI[6] by hypoxia of the brain). The consequence of this vasoconstriction is a reduction of GFR leading to *some* decrease in water excretion. The second effect seems to be the enhanced tubular reabsorption of water in the innervated kidney induced by a lowering of arterial pressure in the lower half of the body.

A detailed account of our data is to be published in Acta Physiologica Hungarica.

P. BÁLINT, A. FEKETE, and S. SZALAY

Physiological Institute of the University of Budapest, November 22, 1955.

Zusammenfassung

Nach Verminderung des arteriellen Blutdruckes ist die Senkung der Natrium- und Wasserausscheidung grösser in der innervierten als in der denervierten (transplantierten) Niere. Es konnte festgestellt werden, dass der verminderte Blutdruck der unteren Körperhälfte bei gleicher Filtration eine vermehrte tubuläre Natrium- und Wasserreabsorption der innervierten Niere hervorruft.

Actinomycetes Antagonistic to *Polyporus annosus* Fr.

The root-rot of Norwegian spruce (*Picea abies*) and other conifers is a serious disease causing heavy financial losses in Scandinavian forests. The mode of infection of the fungus (*Polyporus annosus* Fr.) has not been established with certainty, but the general opinion is that the mycelium of the fungus is capable of growing in the soil, where it infests the dead tap root of spruce, thus entering the living roots and the stem.

However, in the laboratory it has so far not been possible to make the fungus grow in unsterilized soil, whereas the mycelium is able to grow in autoclaved soil. Experiments have therefore been made to find out whether any relationship could be traced between antagonistic micro-organisms in the uninfested soil and the occurrence of the fungus. BJÖRKMAN[1] isolated a large number of soil fungi and tested them for their antagonistic effect on the growth of the mycelium of *Polyporus annosus*, and he found an apparent correlation between the occurrence of the mycelium and soil fungi without inhibiting effects. RENNERFELT[2] in a similar experiment

[4] H. HARTMANN, S. L. ORSKOV, and H. REIN, Arch. ges. Physiol. *238*, 239 (1937). – E. ØPITZ and D. H. SMYTH, Arch. ges. Physiol. *238*, 633 (1937). – E. E. SELKURT, Amer. J. Physiol. *147*, 537 (1946). – R. E. SHIPLEY and R. S. STUDY, Amer. J. Physiol. *163*, 750 (1950).

[5] R. A. FISHER, *Statistical Methods for Research Workers*, 10th ed. (Oliver and Boyd, London 1946).

[6] P. GÖMÖRI, A. G. B. KOVÁCH, M. FÖLDI, GY. SZABÓ, and Z. NAGY, Acta physiol. Hung. *4* suppl., 42 (1953).

[1] E. BJÖRKMAN, Physiologia Plantarum *2*, 1 (1949).

[2] E. RENNERFELT, Oikos *1*, 65 (1949). – E. RENNERFELT and S. K. PARIS, Oikos *4*, 58 (1952).