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The animals given neostigmine showed a definite hypersensitivity. The hyperglycaemia seen 1 hr after $500 \,\mu\text{g/kg}$ noradrenaline was significantly greater than the control, so too were the differences between the control and neostigmine-treated groups during the 3 hr period of the experiment when adrenaline was administered in a dose of $200 \,\mu\text{g/kg}$ (Tables 1 and 2).

On the basis of our results cocaine and imipramine are relatively less effective than neostigmine in enhancing the hyperglycaemic action of catecholamines. It is possible that enzymes described as destroying tropine derivatives in rabbits (Werner, 1965) could be responsible for the weak activity of cocaine.

It has been shown that neostigmine potentiates the blood pressure response to noradrenaline (Fekete, 1966). The present experiments show that this effect of neostigmine can also be demonstrated on glucose mobilization caused by catecholamines in atropinized animals.

Research Institute for Pharmaceutical Chemistry, Budapest 4/1, Ujpest, P.O.B. 82, Hungary. January 18, 1968 M. Fekete Ilona Macsek

References

Fekete, M. (1966). Medna Pharmac. exp., 14, 246-258. Hagedorn, H. C. & Jensen, B. N. (1923). Biochem. Z., 135, 46-48. Hardman, J. B. & Mayer, S E. (1965). J. Pharmac. exp. Ther., 148, 29-39. Schmidt, H. & Späth, M. (1963). Arzneimittel-Forsch., 13, 761-764. Werner, G. (1965). Arch. exp. Path. Pharmak., 251, 320-334.

The origin of epileptiform seizures caused by oil of Artemisia caerulescens L. (Correction)

SIR,—An error has arisen in the Letter to the Editor on the above topic (Srebočan & Stern, 1968). In all instances γ -aminobenzoic acid should be replaced by γ -aminobutyric acid.

Institute of Pharmacology, School of Medicine, University of Sarajevo, Yugoslavia. March 8, 1968 P. STERN

Reference

Srebočan, S. & Stern, P. (1968). J. Pharm. Pharmac., 20, 160-161.