Zusammenfassung

Täglich lokal verabreichte eiweissfreie Fraktionen des hypothalamischen Extraktes zu adenohypophysären Autotransplantaten in die vordere Augenkammer hypophysektomierter Ratten hemmte die Schilddrüseninvolution und stimulierte möglicherweise den Vorgang bei Nebenniere, Hoden und Samenbläschen.

The Urinary Excretion of Noradrenaline and Adrenaline during Acute Alcohol Intoxication in Alcoholic Addicts

High urinary excretion of noradrenaline and adrenaline has been reported[1] in the course of delirium tremens and allied conditions. There was no correlation between the catecholamine output and the intensity of single clinical symptoms, with the presence or absence of ethyl alcohol or drugs in the blood, or with the laboratory findings other than blood pressure and pulse rate. In patients with syndrome B[2], however, the excretion of urinary catecholamines had a definite tendency to fall immediately after the disappearence of the blood alcohol. Therefore it was of interest to investigate, whether or not the presence of alcohol in the blood of alcohol addicts is accompanied by increased excretion of urinary noradrenaline and adrenaline independent of the above-mentioned syndromes.

Material and Method. 16 male alcoholic patients showing no acute signs of alcoholic syndromes were used as test subjects at least one week after recovery. Eight patients consumed brandy or wine, and eight other patients received intravenous infusions of ethyl alcohol 25 vol.% in Ringer solution, on an average 2.3 g/kg within ca. 5 h. The blood alcohol concentrations were determined with the method of WIDMARK[4] in all cases. The peak level varied between 1.5‰ and 3.3‰. The urinary catecholamines were determined in 24 h collections before, during and after alcohol uptake by the method of EULER and LISHAJKO[5].

Results. There was no statistical difference between the excretion of urinary noradrenaline and adrenaline in convalescent alcoholic addicts before, during, and after ethyl alcohol administration, and that of 12 healty persons[1] (Table). In no case following induced alcohol intoxication could the neurological symptoms of syndrome B be detected.

	Healthy persons	Convalescent alcoholic addicts		
		day before experiment	day of experiment	day after experiment
Noradrenaline µg/24 h	18.0±6.9	16.9±7.8	17.3±6.9	17.0±5.7
Adrenaline µg/24 h	6.4±3.1	8.2±4.4	7.6±2.7	8.1±2.8

Urinary excretion of catecholamines in convalescent alcoholic addicts before and after acute alcohol administration.

Discussion. The 24-h excretion of urinary catecholamines in alcohol convalescents showed no alteration when compared with the values of healthy subjects[1]. Ethyl alcohol administered during a short period neither increased the urinary excretion of noradrenaline and adrenaline nor did it induce the picture of syndrome B in these subjects, even if the doses were as high as 2,3 g/kg intravenously. The increase of urinary catecholamines occurring in acute alcohol intoxications together with SB in alcohol addicts[1], seems therefore to be connected with a longer lasting abuse of ethyl alcohol than was the case in our experiments. It is evidently not dependent on the immediate effect of alcohol on the organism but on some factors developing in the course of SB and alcohol hallucinoses. In this connection, it is of interest that alcohol given intravenously in two patients during delirium tremens and SB respectively, did not influence the patterns of the urinary excretion of catecholamines.

The excretion rate of urinary noradrenaline and adrenaline during the 5-h infusion period was found to be in the range of normal diurnal variations reported by EULER and LISHAJKO[5]. Since, in our experiments, the catecholamines were not measured in shorter periods, a slight transient increase such as that shown by other authors[6-8] may have escaped notice.

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Beckomberga Hospital, Stockholm-Bromma and Karolinska Institutet, Department of Physiology, Stockholm (Sweden), March 24, 1960.

Zusammenfassung

Die Katecholaminausscheidung im 24-h-Urin rekonvaleszenter Alkoholpatienten wird durch einzelne Äthylalkoholdosen (2,3 g/kg) nicht beeinflusst.

[1] E. GIACOBINI, S. IZIKOWITZ, and A. WEGMANN, A.M.A. J. gen. Psych., in press (1960).

[2] Syndrome B (SB) is the most frequent acute state following an intense or long lasting alcohol or drug abuse. It is characterized by anxiety, tremor, vasomotoric reactions, hyperhidrosis, sleep disturbances, and anorexia, but without disorientation or hallucinations (for nomenclature see IZIKOWITZ[3]).

[3] S. IZIKOWITZ, Nord. Med. *60*, 1009 (1958).

[4] E. M. P. WIDMARK, Biochem. Z. *131*, 473 (1922).

[5] U. S. v. EULER and F. LISHAJKO, Acta physiol. scand. *45*, 122 (1958).

[6] E. S. PERMAN, Acta physiol. scand. *44*, 241 (1958).

[7] I. ABELIN, CH. HERREN, and W. BERLI, Helv. med. Acta *25*, 591 (1958).

[8] G. I. KLINGMAN and M. GOODALL, J. Pharmacol. *121*, 313 (1957).

PRO EXPERIMENTIS

An Ultra-High Vacuum System Using an Oil-Diffusion Pump with a Non-Refrigerated Isolation Trap[1]

We found that an oil-diffusion pump with a modified Biondi trap[2] charged with activated alumina was very satisfactory in continuously maintaining ultra-high vacuum for periods of several months. The trap did not require refrigeration and the activated alumina beads could be reconditioned by bakeout. Because of the high conduct-

[1] This work was done for the Lawrence Radiation Laboratory at Livermore, California, under the auspices of the Atomic Energy Commission.

[2] M. A. BIONDI, Rev. Sci. instr. *30*, 831 (1959).