into healthy adults. Survival time of red cells in newborns is shorter than that observed in adult subjects. There was no appreciable difference in results obtained in newborns at sea level and at high altitudes.

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Effect of Pyridoxine in Acute Alcoholic Intoxication. (24591)

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Wordsworth(1) stated that "Vit. B_6 is a specific antidote to alcohol." He based this conclusion on his experience with 6 human cases of acute alcoholic intoxication observed clinically. Small and co-workers(2) made a more extensive and objective investigation of the possible antagonism between alcohol and pyridoxine in patients suffering from acute alcoholic intoxication and in volunteers given varying doses of alcohol. A battery of tests of nervous function was used, and an attempt made to correlate degree of intoxication as appraised by these tests with blood alcohol concentration. The plan of the experiments was to give repeated doses of whiskey until a fairly marked degree of intoxication was demonstrated, then to give pyridoxine in large doses to see whether a sobering effect was produced. In no case was a significant amelioration of the intoxication demonstrated. The authors comment on the difficulty in assessment of a drug for treatment of such a complex of psychic and organic alterations as is comprised in human alcoholic intoxication; they found variations in performance which did not correlate with blood alcohol levels or clinical state of the subjects. It seemed worth while to investigate the problem in an experimental situation more readily controlled than when human drunkenness was involved.

Methods. Over many years we found that degree of alcoholic intoxication in the dog may be quite reliably estimated by the trained observer, utilizing an arbitrary scale of 9 degrees, stage 9 being abolition of corneal reflex, stage 1 slight ataxia on climbing a flight of stairs(3). Seven female dogs varying in weight from 10 to 15 kg received intravenous infusion of 3 g of ethyl alcohol/kg, diluted with physiological salt solution to 15% w/v, in approximately 30 minutes. One hour from start of infusion, and subsequently at hourly intervals for 7 hours, samples of venous blood from a foreleg were analyzed for alcohol(4) and degree of intoxication observed and recorded. These constituted the control runs. The same animals were subsequently subjected to the same procedure, except that they received 100 mg of pyridoxine intravenously just prior to alcohol infusion; while on another occasion 3 dogs received pyridoxine 2 hours after start of infusion. The rate of fall of blood alcohol concentration in mg/100 ml of blood/hour, a reliable criterion of rate of alcohol metabolism, was calculated by subtracting final blood alcohol concentration from that found on first sample and dividing by number of hours elapsed between the 2 samples.

Results. In control runs this averaged 20.6

mg/100 g blood/hour, with pyridoxine 20.5 mg, so that it can be said definitely that pyridoxine under these experimental conditions has no effect on rate of alcohol metabolism.

By plotting degree of intoxication against blood alcohol concentration for each experiment, it was possible by interpolation to estimate degree of intoxication at successive decrements of blood alcohol concentration of 20 mg/100 g. By averaging these values for 7 control runs and 10 runs with pyridoxine it was possible to plot curves of degree of intoxication at different blood alcohol concentrations with and without pyridoxine. From this graph it was apparent that at no blood alcohol concentration was there a difference of more than half a degree of intoxication that could be ascribed to pyridoxine, and where a difference occurred, this was most often a greater degree of intoxication with pyridoxine than without.

Conclusion. In the dog pyridoxine is ineffective in either increasing rate of metabolism of alcohol or reducing degree of intoxication at a given blood alcohol concentration. This is confirmatory of previous work in man, and is conclusive evidence of the uselessness of pyridoxine as an antagonist of alcoholic intoxication.

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Localization of I^{131} Labeled Antibody to Rat Fibrin in Transplantable Rat Lymphosarcoma.* (24592)

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It was possible to prepare an antibody obtained from antisera to the transplantable Murphy-Sturm rat lymphosarcoma that, after labeling with I^{131} and intravenous injection into tumor bearing rats, will localize with a high degree of preferentiality in this tumor (1,2,3). Our recent work indicated that such labeled antibody prepared against this tumor grown in rats, showed a very low degree of tumor localization when injected intravenously into hamsters bearing this tumor as a cheek pouch transplant(3). Also *in vitro* tests indicated that the hamster-grown tumor showed a weak immunological reaction with antibody prepared against the rat-grown tumor. These results combine to suggest that an important antigen in the rat-grown tumor responsible for *in vivo* antibody localization might be some substance produced or deposited by the rat host rather than some antigen intrinsic to the tumor cell itself. Day, Pressman, and associates have recently reported (personal communication) and we have confirmed that *in vivo* localizing antibody isolated from Murphy tumor antisera shows a strong tendency to react immunologically with the rat fibrinogen-fibrin system(4). This suggests that an important antigen responsible for localization of antibody in the rat Murphy tumor might be fibrinogen or fibrin deposited in the tumor by the host, possibly in response to an inflammatory reaction by host tissues to tumor growth. We have therefore immunized rabbits with rat fibrin, clotted the rabbit antisera with normal rat plasma, and from the washed clot isolated and labeled with I^{131} a substance, presumably an antibody, that shows a strong preferential tendency to localize in the Murphy-Sturm lymphosarcoma.

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