alkaloidal reagents and finally compared by Nagai's alkaline copper sulphate test. Then these extracts were used for physiological testing. Three intravenous injections of each of the two Californian Ephedras before and after an identical injection of Peking Ephedra were made with a large dog, suitably anesthetized and canulated for blood pressure tracing.

The chemical assay and testing, as compared with known extracts of specimens of Peking Ephedra containing ephedrine, showed absolutely no ephedrine in our sample of *E. californica, Wats,* nor was there anything more than possibly an exceedingly small trace in *E. nevadensis, Wats.* Both species gave no rise in blood pressure as did the preparations from Peking Ephedra. These results lead to the inference that the results formerly obtained with the infusions of Ephedra were probably due to the presence of simple colloids, and certainly not to basic substances related to ephedrine, unless there be exceedingly great seasonal variation in the alkaloidal content of this plant.

[1] Clark, G. W., and Groff, G. W., PROC. SOC. EXP. BIOL. AND MED., 1927, xxiv, 325.

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Blood of Animals in Hematoporphyrin Shock.

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Experiments designed to throw light on the state of the blood of animals sensitized by hematoporphyrin and exposed to light were carried out on cats, guinea pigs, rats and mice.

One to 2 cc. per 100 gm. of body-weight of a 0.5 per cent solution of crystalline hematoporphyrin hydrochloride (Nencki) in n/10 NaoH or 1 to 2 per cent $NaHCO_3$ were injected subcutaneously, intraperitoneally, or intravenously. Sunlight was usually employed. Normal animals and animals similarly injected but kept in the dark were studied throughout as controls.

In preliminary observations no difficulty was encountered in securing the usual reaction of such sensitized animals on exposure of the skin to light.

Exposure of the peritoneum alone was followed by shock, but exposure of blood flowing through a glass canula inserted in the

femoral artery or between the femoral artery and femoral vein produced shock in only 1 of 5 cases.

Mice fed on hematoporphyrin by mouth for 8 weeks did not develop any sensitization to light.

Blood Pressure. The carotid pressure of cats sensitized by hematoporphyrin and exposed to light falls from normal values of 120 to 160 mm. Hg. to 30 to 50 mm. within 35 to 120 minutes. The rapidity of fall depends upon the intensity of the light. A prompt reaction to adrenalin is maintained. The heart beat and the respiration cease simultaneously.

Pulse Rate. Both pulse and respiration rate increase markedly during the development of shock and are slowed towards the end of the experiment.

Blood Cells. The number of red blood cells remains practically unchanged; during the early stages a leucocytosis of 13,000 to 18,000 appears, which is followed by a leucopenia of 4,000 to 5,000.

Blood Chemistry. The amount of sugar, total non-protein nitrogen and creatinine was found to be essentially the same as in control animals.

The oxygen content of mixed venous blood from the right ventricle of guinea pigs in hematroporphyrin shock falls from 11 to 14 volumes per cent to 6 to 10 volumes per cent, while the oxygen capacity remains unchanged.

The carbon dioxide combining power of the serum falls from 37 to 54 volumes per cent to 16 to 20 volumes per cent in spite of the introduction of 10 to 15 cc. of 1 to 2 per cent $NaHCO_3$ in which the hematoporphyrin was dissolved.

Toxicity of the Blood. From 0.5 to 1.5 cc. of the heart's blood of guinea pigs in hematoporphyrin shock was injected into mice and produced no effect different from that of the injection of the same amount of heart's blood from control animals.

In a parabiosis of 2 rats hematoporphyrin shock induced in one was not communicated to the other.

Effect of Light on Sensitized Blood in vitro. Sensitized blood exposed *in vitro* to light changes its color to dark reddish brown. The resistance of the red blood cells to hypotonic saline solutions falls and hemolysis occurs.

Gas analysis shows changes similar to those found *in vivo,* decrease in the oxygen content without change in the capacity, and increase of the carbon dioxide content.