whether the above differences were due to the differences in temperature. This was shown not to be the case: it was the illumination and not the temperature which rendered the drug more toxic. Further experiments revealed that it was the light waves from the violet end of the spectrum that were the most effective in increasing the toxicity of quinidin. In still other experiments the rays of an electric arc lamp were utilized instead of sunlight and the same potentiation in toxicity was qualitatively noted. Finally a few experiments on excised frogs' hearts were performed and the results obtained so far have maintained the above findings.

In the above experiments an aqueous solution of quinidin was used because quinidin solutions are known to lose much of their fluorescence when alkalis or even sodium chloride are added to them. Tests made however by adding various amounts of blood serum and even sodium chloride to solutions of quinidin sulphate indicated that while these decreased the fluorescence markedly they did not abolish it completely; and it was further found that even such poorly fluorescent substances showed the difference in toxicity as between light and darkness described above.

Quinin solutions were found to behave in much the same way as those of quinidin. It may also be added that the toxic effect on frogs appears much more rapidly if the animals are placed in such a position as to allow the sunlight to fall directly on the white unpigmented skin of the abdomen, instead of the pigmented skin of the rest of the body. Further and more extensive work on the subject is in progress and will be continued. The present note is published as a preliminary communication and in order to fix priority of date of discovery.

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The therapeutic effect of germanium dioxide in anemia.

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The erythropoietic action of germanium dioxide in animals and in one normal man was demonstrated by Hammett and

Müller.[1] We have treated ten patients suffering from anemia, administering the germanium (N. J. Zinc Co. product) by mouth in 0.2 per cent. water solution. The dosage was between 100 and 200 mgs. of germanium dioxide given daily, or in some cases every two or three days, until between 950 and 1,400 mgs. had been given. In three cases of anemia following hemorrhage there were maximum increases in the number of erythrocytes per cubic millimeter of blood amounting to 77.2, 71.4 and 41.5 per cent. above the control counts. The hemoglobin in these cases increased to a maximum of 53.2, 35.1 and 56.7 per cent. above the controls. In addition to the germanium, one and one half ounces of "ovoferrin" were given daily to the last patient, who had carcinoma of the uterus. The condition of the patient became worse, however, and she died on the day of the maximum red-cell count.

In five cases of secondary anemia, with diagnoses including visceroptosis, colitis, tachycardia, carcinoma of the breast and malignant endocarditis, after treatment with germanium the red cells increased to a maximum of 23.4, 26, 53.4, 25.8 and 5.4 per cent. above the control counts, with increases in hemoglobin up to 9.5, 10.1, 14.5, 20 and 3.1 per cent. above the controls. In one of these cases after the increase in red cells of 53.4 per cent. there was a drop to 40 per cent. above the control one week after the last dose of germanium, the count remaining at this level for about ten days and then dropping to 7.7 per cent. above the control. A second treatment with several doses of germanium resulted in increases up to 24.5 per cent. above the control count.

In a man suffering from chronic cardiovalvular disease there were small increases and decreases over the control red-cell counts, and decreases as great as 15.9 per cent. below the control hemoglobin content after the germanium treatment. It appears that in this case the medication was of no value.

A woman with carcinoma of the cervix showed decreases in both red cells and hemoglobin after germanium. This patient had been treated with radium one week before the first dose of germanium.

[1] Hammett, F. S., Nowrey, J. E. and Müller, J. H., *J. Exp. Med.*, 1922, xxxv, 173. Müller, J. H., and Iszard, M. S., *Am. J. Med. Sci.*, 1922, clxiii, 364. Hammett, F. S., Müller, J. H. and Nowrey, J. E., *J. Pharm. Exp. Therap.*, 1922, xix, 337.

In all of the cases studied there was no significant change in the white blood-cell counts. Judging from the percentages of blood total solids, the increases in red blood cells were not due to a concentration of the blood from loss of fluid. The color index generally dropped early in the treatment with a gradual rise later. A study of chemical blood and urine analyses in certain cases revealed no apparent effect of the germanium on the functional activity of the kidneys. The periodic effect on the red-cell count noted by Hammett and Müller was also noted in this study.

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Influence of ischemia on infection.

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The following experiments furnish a striking illustration, suitable for class-room demonstration, of the influence of temporary ischemia on the local resistance to infection:

Two or three slight cuts are made on the upper or middle third of each ear of a rabbit, by closely clipping the hair from the dorsal surface of the ear; lifting with forceps a small fold of the skin, and snipping this fold away with scissors. This makes small wounds of about 4 by 7 mm., usually without hemorrhage. The wounds may then be smeared with active agar-cultures of staphylococcus or pyocyaneus; or they may be left without artificial infection. Within a few minutes after making the wounds, 1 c.c. of epinephrin, 1 : 1000, is injected into the root of one ear, close to the entrance of the vessels. This produces an intense ischemia of the entire ear, persisting for several hours.[1]

From the following day, the two ears present a striking difference in appearance: The wounds on the normal ear show signs of healing, even if they were severely infected. The wounds on the ear that had been rendered anemic appear much more inflamed, and may be covered with pus, and a perforating ulcer

[1] Auer and Meltzer, *Proc. Soc. Exper. Biol. and Med.*, 1916, xiv, 54.