carbonate, sodium citrate, sulphuric acid, neutral sulphur and sodium hydroxide have been given in the diet of maize and wheat for periods of one, two and three months. One hundred and ten fowls have been used. No detectable inhibition of the parathyroid overgrowth could be detected in the fowls given magnesium carbonate, strontium carbonate, sulphuric acid, neutral sulphur, sodium citrate or sodium hydroxide,—the growth being as marked as in the controls. On the other hand, those given sulphuric acid and neutral sulphur had more marked parathyroid enlargements and softening of the bones than those given the other chemical substances or the controls. In those fowls which had received calcium there was uniformly less parathyroid overgrowth; in those given calcium hydroxide and calcium carbonate it was barely detectable; while in those given calcium lactate there was moderate enlargement. No differences ascribable to sex could be determined.

These observations suggest: (*a*) that the parathyroids of birds are more susceptible to overgrowth than those of mammals; (*b*) that calcium offers some protection against overgrowth; and (*c*) that the parathyroids (as MacCallum has suggested) are intimately associated with the function of calcium in the complex of body metabolism and nutrition.

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Liver necroses associated with Streptococcus infection.

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In a series of experiments upon rabbits to determine the tissue reactions to the infection by the *Streptococcus viridans* and having special reference to the heart, arteries, and kidneys, several sporadic examples of necrosis of the liver were encountered. Living cultures of *Streptococcus fecalis*, *Streptococcus mitis*, and *Streptococcus salivarius* were used. Repeated inoculations, from three to five, had been made at intervals of four days.

The earliest necroses appeared in eleven days and consisted

of small focal areas in the peripheral and mid-zones of the liver lobules. In them only a few cells appeared to be affected and seemed to be sporadically picked out in the midst of the liver column. Debris or the ghosts of cells, was all that remained. There appeared to be some edema in the involved area but evidence of thrombosis in the neighboring sinuses was not always demonstrable. In some instances a granular thrombus with fibrin threads was present immediately about the lesion, and at times, extended towards the central vein. Similar thrombi, however, were also observed in areas not showing necrosis.

Some liver columns appeared to show change antecedent to necrosis. In them the cells showed a diminution of nuclear staining with an eosinophile character of the protoplasm. In the vicinity of these again, thromboses were wanting.

Other areas again showed much more advanced necrosis involving not only focal areas but entire lobules or even several neighboring lobules. In all of these instances the necrosis involved the central and mid-zone, while some liver columns still persisted in the vicinity of the portal sheath. In these larger areas thromboses of the mixed fibrinous variety were common. The sinuses of the affected areas were irregularly involved, but not constantly, the central vein being most commonly plugged. These thromboses extended into the sub-lobular vein. Thrombi of agglutinated red blood cells were not observed. There was no inflammatory reaction in the large areas of necrosis nor was there any attempt at restitution either by connective tissue or liver cells.

In 1906, Pease and Pearce[1] noted the occurrence of liver necroses in horses, immunized against the streptococcus pyogenes. In their cases the liver showed diffuse necrosis but they were unable to demonstrate the nature of the process. Since then much literature has appeared in the discussion of liver necroses, and the condition has been described in a great variety of intoxications.

In the absence of thrombi and a cellular reaction in many of the early necroses observed in our cases, it would appear that they have resulted by a direct intoxication by these streptococci. The mixed fibrinous thrombi, developing in the blood channels distal to the liver involvement, probably result from ferments

[1] *Jour. Inf. Diseases*, 1906, III, p. 619.

liberated from the damaged liver cells. Such progressive thrombosis assists in producing a more widespread necrosis of the partly damaged liver tissue, even involving several neighboring lobules.

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The effect of gentian violet on protozoa and on growing adult tissue.

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This study was primarily undertaken to settle two questions raised by the observations made during the last two years on the effect of gentian violet on bacteria.[1] It was noticed early in the gentian violet studies that motile organisms not killed by the stain (violet negative organisms) retained their motility even though deeply stained; and that these stained violet negative organisms when transplanted to agar slants grew equally well with the control smears of unstained bacteria. The retention of motility by the stained organisms might in these experiments be explained as a survival phenomenon; and the growth of transplants made from the stained specimens might be regarded as arising, not from the organisms in the smear which had taken the stain, but from the few in the smear which had escaped it. It seemed altogether likely, from other observations that these explanations were not the correct ones; and that the violet negative organisms actually took the stain during life. Still, definite proof was lacking that gentian violet in these experiments was acting as a true intra-vital stain. To furnish this proof and to investigate the further problem (raised but not solved by the experiments with bacteria) as to whether the vital dye stained the nucleus or the protoplasm, two series of experiments have been done; one with a protozoon (paramecium) and another with living tissues.

EFFECT OF GENTIAN VIOLET ON PROTOZOA.

The paramecium used for this purpose came from a pedigreed race kindly furnished by Professor Woodruff. The effect of the

[1] Churchman, *Journ. Exp. Med.*, Vol. XVI, No. 2, 1912; Vol. XVI, No. 6, 1912; Vol. XVII, No. 4, 1913.