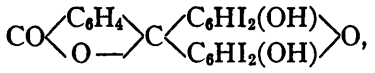
water. This free dye-acid has the following formula,



with a molecular weight of 836.

The dye-acid of iodo-eosin may be made by dissolving ten grammes of iodo-eosin (commercial dye) in one per cent. potassium hydroxide and then adding hydrochloric acid in excess. The dye-acid is precipitated at once, it can then be filtered off and the precipitate washed with hot water till the washings are acid-free. The precipitate after drying is easily soluble in ether, forming a beautiful yellow-colored solution.

When this free dye-acid in ether solution is placed inside of an intact rubber membrane immersed in ether, it can readily be noted that in a few minutes diffusion currents are visible and the ether outside of the bag becomes colored, showing that the free dye-acid has diffused.

The bearing of our results on the question of permeability and impermeability of membranes will be considered later.

ABSTRACTS OF THE COMMUNICATIONS, PACIFIC COAST BRANCH.

First meeting.

San Francisco, California, December 4, 1912.

36 (732)

Chronic lead poisoning in guinea pigs.

By W. OPHÜLS.

[*From the Pathological Laboratory of Stanford University.*]

Of the twenty-eight guinea pigs treated with sublethal doses of carbonate of lead seven (25 per cent.) showed a peculiar condition to which so far attention does not seem to have been directed. There has developed a hemorrhagic, sero-fibrinous inflammation of the pericardium, of the peritoneum in the upper part of the peritoneal cavity and occasionally also of the pleuræ. In the pericardium the lesion commences with a hemorrhagic exudate followed by the formation of fibrinous deposits especially on the parietal layer and ending with organization with marked thicken-

ing and occasionally adhesions. In the pleuræ one finds as a rule a simple serous exudate without deposits but occasionally heavy deposits of fibrin also. In the peritoneum also the condition commences with fibrinous deposits and eventually leads to marked peritoneal thickening more especially in the upper part of the peritoneal cavity. The capsules of liver and spleen are much thickened and the contraction of the thickened capsule often causes marked deformity of the liver. The lesions produced in this way are closely analogous to Curschmann's "Zuckergussleber." The spleen is always more or less enlarged, but this is due to the excessive destruction of erythrocytes by the lead and is found in all animals.[1]

It was only natural to suspect the presence of a bacterial infection of the serous membranes in these cases. A very careful bacteriological examination was made in several of the cases, but it was entirely negative.

Whether the hemorrhagic exudation which ushers in the process is due to the lead anemia appears to me a question well worth debating. Smears of the exudate show few leucocytes and often many nucleated red blood corpuscles, even megaloblasts.

In one case (46) adhesions had formed in the upper part of the peritoneal cavity with partial strangulation of duodenum and intestine and extreme dilatation of the stomach.

In the liver the periportal connective tissue is moderately but distinctly thickened in some cases. One always finds small areas of fatty degeneration and necrosis of the liver tissue. In one case (46) they were quite large and show beginning liquefaction in the center (due to apparently secondary invasion of bacteria). Whether these degenerative lesions are due to circulatory disturbances produced by the shrinkage of the thickened capsule or whether they are independent of this and toxic in origin, it is difficult to decide, although in one case the lesions in the liver were well marked but those in the capsule only slight. The bearing this may have on the modern conception of cirrhosis as closely connected with certain types of anemia is evident.

[1] It is interesting to know that Charcot and Gombault found acute pericarditis in their guinea pig 4, hemorrhagic pericarditis in their guinea pig 9, subacute pericarditis in their guinea pig 15, and pericardial thickening in their guinea pigs 6 and 14 (5 out of 15).

It is interesting to observe in these animals a condition so entirely analogous to what has been described in man as polyserositis, or a polyorrhomenitis by Italian investigators, of which Curschmann's Zuckergussleber and Pick's pericarditic pseudocirrhosis are only special manifestations. The relations of these conditions in man to chronic lead poisoning and to chronic anemia remains to be studied.

37 (733)

Chronic lead poisoning in guinea pigs. Its relation to chronic nephritis.

By W. OPHÜLS.

[*From the Pathological Laboratory of Stanford University.*]

During the last years I have been repeating the experiments of Charcot and Gombault[1] on chronic lead poisoning in guinea pigs. Twenty-eight guinea pigs were given sublethal doses of carbonate of lead for periods ranging from one month to three years and ten months. Fourteen of these experiments lasted over one year.

The lesions in the kidneys were much less striking than one would expect from the report of Charcot and Gombault. In all cases there was a limited necrosis and desquamation of the epithelium with marked evidences of regeneration especially in the ascending limbs of the loops of Henle. In some cases the epithelium in places was heavily pigmented. Occasionally there were seen a few glomeruli with slightly thickened capsules. There were only two of the twenty-eight experiments in which more advanced lesions were discovered in the kidneys. In one case (69) in which the guinea pig had received over thirty grams of carbonate of lead in three years the kidneys were actually granular and the cortex distinctly narrow. The lesions in this case consisted in collapse of tubules over large areas with marked development of fibrous tissue between them. There were many casts. The glomeruli showed marked fibrous thickening of the capsules and cystic dilatation. The other guinea pig (51) which received about four grams of carbonate of lead in twenty months showed similar con-

[1] Charcot et Gombault, "Note relative à l'étude anatomique de la nephrite saturnine expérimentale," *Arch. de Phys.*, 1881, 2 s., VIII, 126.