The essential results are summarized in Table I. The effects of phlorhizin alone on the blood constituents were similar to those reported by Kastler, except for the absence of a significant increase in serum inorganic phosphorus. While the serum potassium was regularly reduced by phlorhizin alone, it remained unchanged when phlorhizin administration was accompanied by the ingestion of potassium chloride in large amounts. On the other hand, the reduction of serum potassium as a result of phlorhizin administration was apparently accentuated by the feeding of excessive amounts of sodium chloride. In neither instance, however, did the extra salt ingested significantly influence the degree of phlorhizin hypoglycemia. The glycosuria due to phlorhizin administration was likewise essentially unaffected by either salt. The slight increase observed in some experiments was obviously due to an accentuation of the phlorhizin diuresis by the salt ingested. Slight variations which were noted in the excretion of nitrogen and of minerals could be accounted for in the same way. The calcium, magnesium, sodium, non-protein nitrogen, chloride and CO_2 content of the serum were not significantly altered by the procedures employed.

It is concluded from these experiments that the disturbance in carbohydrate metabolism due to phlorhizin poisoning bears no relationship to that of diabetes mellitus as regards its response to ingestion of excessive amounts of sodium or potassium salts. The characteristic effects of these salts on hepatic glycogenesis in the normal dog appear to be abolished by phlorhizin.

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Use of Aluminum Metal in Contact with Blood in Perfusion Systems.

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In attempting to use the Bayliss-Fee oxygenator in isolated mammalian heart studies it was found that the life of the preparation was very short, as little as 30 minutes. The coronary flow was found to diminish progressively and the hearts failed rapidly. In investigating possible causes it was found that considerable $Al(OH)_3$ accumulated in the system. The oxygenator is made of cast aluminum. The corrosion of the aluminum metal obviously led to the production of $Al(OH)_3$. Therefore all surfaces were

covered with a uniform thin coat of clear lacquer. The life of the isolated heart in the system was thereby greatly prolonged.

To elucidate the mechanism of the effect of aluminum two types of experiments were performed. First, frog hearts were set up on Straub cannulæ and Ringer containing 200-400 mg % of $Al(OH)_3$, as alumina cream, was substituted for the plain Ringer placed in the cannula for control observations. These amounts of $Al(OH)_3$ had no toxic effects upon the frog hearts, rather there was a slight tonic effect and the hearts kept beating longer than did simultaneous controls.

A second group of experiments were run upon rabbit hearts perfused by the Langendorff technic. There was a trace of blood in the perfusion fluid in the first experiments. Addition of 2 cc of 2% $Al(OH)_3$ in Ringer to the perfusion fluid caused almost immediate stoppage of the heart, and of the flow of fluid through the coronaries. In other experiments the heart was perfused with Ringer containing no blood and the perfusate discarded. It was washed with 1500 cc of Ringer, after which the emerging fluid was protein-free by the tungstic acid test. Addition of 15 cc of 2% $Al(OH)_3$ thereafter did not stop the heart, although the amplitude of contraction was somewhat diminished. In still another experiment the coronaries were thoroughly washed with plain Ringer as before. Addition of 40 mg % of $Al(OH)_3$ to the perfusate had a noticeable tonic effect. Perfusion of this solution, oxygenated, was continued for 40 minutes without deterioration. Then 10 cc of the rabbit's blood were added to the $Al(OH)_3$ containing Ringer. At the instant that this fluid reached the heart the beat became feeble and the coronary flow fell off sharply. The heart was dead within 5 minutes.

A single experiment was performed with the ordinary heart-lung preparation in which, first, 8 cc of 2% $Al(OH)_3$ was injected slowly into the blood as it flowed to the right atrium. There was no detectable effect upon the heart. Next 2 cc of the same solution was slowly injected directly into the left atrium. The heart began to fail immediately and stopped beating within 5 minutes. It is apparent that in the first case passage through the lungs removed the toxic agent, and that injection directly into blood entering the coronary arteries was fatal. The lung is an efficient filter and this property probably accounts majorly for the superiority of the heart-lung preparation over isolated heart circuits for cardiac physiology studies.

We conclude that $Al(OH)_3$ is toxic only to hearts perfused through coronary arteries, and that the damage in the latter case is

probably due to capillary embolism resulting from the precipitation of plasma protein by $Al(OH)_3$. Obviously, one should avoid the use of aluminum metal in contact with blood in any perfusion systems.

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Origin of Bone Marrow Plasma Cell Associated with Allergic and Immune States in the Rabbit.*

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The observation of a plasmacytosis of the bone marrow of a man with a fatal endocarditis, coupled with Downey's description of a great number of plasma cells in the bone marrow of a rabbit with an incidental, non-specific, subcutaneous abscess suggested an experimental method for increasing the bone marrow plasma cell content. This reaction was also indicated by the bone marrow from rabbits allergic to and immunized against *Streptococcus viridans,* obtained through the courtesy of Dr. B. J. Clawson of the Department of Pathology, University of Minnesota. In these animals it was noted that the allergic and immune animals showed a more extensive medullary plasmacytosis than those that were immune but not allergic. Through the study of the myeloid tissue during the development of a plasmacytophilia, we felt something of the origin and function of the bone marrow plasma cell might be determined.

There are many conflicting theories regarding the origin of the myeloid plasma cell (Downey,[1] Michels,[2] Rohr,[3] Jordan and Morton,[4] and Osgood and Ashworth[5]). The current theories make the lymphocyte, histiocyte, fibroblast, myeloblast, erythroblast, plasmacytic reticulum cell and specific plasmablast the parent of the bone marrow plasma cell.

The procedure we employed was as follows: Obtain a control

* This work was carried out under the direction of Dr. Hal Downey in the Hematological Laboratory of the Department of Anatomy, University of Minnesota.

1 Downey, Hal, *Folia Hæmat.*, 1911, **11**, 275.

2 Michels, N. A., *Arch. Path.*, 1931, **11**, 775.

3 Rohr, K., *Folia Hæmat.*, 1936, **55**, 305.

4 Jordan, H. E., and Morton, C. B., *Am. J. Anat.*, 1937, **61**, 407.

5 Osgood, E. E., and Ashworth, C. M., Atlas, 1937, J. W. Stacey, Inc., San Francisco.