We have had no previous experience with 2 weeks' regeneration, and it is not a part of the Weech and Goettsch procedure. In this instance the change in serum albumin after 2 weeks' regeneration confirms the distinction between methionine and phenylalanine. Other amino acids are being studied in this manner and will be reported later.

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Role of Particulate Matter in Perfusion of Blood Vessels.*

BENJAMIN W. ZWEIFACH. (Introduced by Robert Chambers.)

From the Laboratory of Cellular Physiology, Department of Biology, Washington Square College, New York University.

A series of perfusion studies were made in which the capillary vessels of the frog's mesentery were kept under observation through the microscope. Striking differences were found between the circulation obtained with perfusates containing particulate matter and that obtained with similar solutions which were particle-free. Colloidal Ringer perfusates free of particulate matter did not fill all the vessels of the capillary bed, circulating only through the a-v capillaries. In a previous publication,[1] it was pointed out that the a-v capillaries represent direct continuations of the arterioles and serve as bridging channels to the venules. The dye T-1824 (Evans blue, Eastman Kodak Co.) has been used for blood volume studies because of its poor diffusibility.[2] When solutions containing Evans blue were used, the restriction of the color to the a-v capillaries stood out in contrast to the true capillaries which remained colorless. The addition of particulate matter, either as a fine suspension of carbon or of washed, rooster red cells, to the Ringer-gelatin or Ringer-acacia perfusates altered the restricted circulation within 30 to 45 seconds by distributing the solution throughout all of the capillaries.

During the early stages of the perfusion with particle-free Ringer-gelatin solutions, it was observed that the true capillaries were quickly emptied of their contained blood cells. This was peculiar since other observations had shown that the circulation of such perfusates

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1 Zweifach, B. W., *Anat. Rec.*, 1939, **74**, 475.

2 Gregerson, M. I., and Gibson, J. G., *Am. J. Physiol.*, 1937, **120**, 494.

was sharply restricted to the a-v vessels. By observing the vessels at the commencement of the perfusion, the blood cells in the true capillaries were seen to move simultaneously towards both the arterial and venous ends of the vessels and to be swept into the a-v circulation. This phenomenon appeared to be brought about by a suction effect arising from the rapid streaming of the perfusate past the true capillary orifices in the walls of the a-v channels. The circulation of the particle-free perfusate remained limited to the a-v capillaries throughout the experiment. No change in the character of the circulation was obtained by raising the perfusion pressure from the normal level of 30 mm up to 75 mm Hg. The augmented pressure merely effected a more rapid streaming through the a-v capillaries.

The result obtained when the animal was perfused with Ringer-gelatin solutions to which a suspension of carbon or avian red cells had been added, was in marked contrast to the above. Under these conditions the circulating fluid not only coursed through the a-v capillaries, but streamed into all the capillary side branches. The repeated, alternate use of particle-free and particle-containing solutions in the same preparations emphasized the distinct difference in capillary circulation obtained with the two types of perfusates. The red cells, because of their larger size and extreme plasticity, were more effective in this respect than carbon. The particulate matter appeared to create a series of disturbances at the points of capillary branching, thereby disturbing the axial a-v current and deflecting the perfusate into the true capillary offshoots.

Ringer-gelatin mixtures, lacking formed elements in suspension, were capable of preventing edema for only 30 to 40 minutes. Ringer-gelatin solutions containing carbon, however, delayed the onset of edema for about 110 minutes. Red cell suspensions were somewhat more efficient, no edema occurring for more than 180 minutes.

An additional interesting feature of the perfusion with red cell suspensions was the part played by these cells in plugging leaks in the capillary wall. When the capillaries were perfused with artificial solutions for more than 120 minutes, the capillary wall tended to become increasingly porous. Chambers and Zweifach[3] have also shown that temporary porous spots appear in the capillary wall and can be increased or decreased by variations in the pH and calcium content of the perfusate. In this stage, a characteristic flattening of red cells against the leaky portions of the wall was observed. This was often followed by portions of the cells being squeezed into tiny openings between the endothelial cells at these points.

[3] Chambers, R., and Zweifach, B. W., *J. Comp. and Cell. Physiol.*, 1940, in press.

It is suggested that, as a result of the markedly restricted circulation with particle-free perfusates, abnormal conditions develop which alter the capillary wall and bring about excessive capillary permeability. This would account for the early appearance of edema with such solutions. The widespread distribution of particle-containing perfusates approached a more normal circulation in the capillary bed and was thereby instrumental in pronouncedly delaying the onset of edema.

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Attempt to Produce Experimental Cardiospasm in Dogs.*

JACK W. GRONDAHL AND H. F. HANEY.
From the Department of Physiology of the University of Oregon Medical School, Portland, Oregon.

In the clinical condition of cardiospasm food does not pass readily from the esophagus into the stomach although at autopsy the cardia does not exhibit hypertrophy or stenosis.[1] Postmortem studies have shown degeneration of the vagi[2, 3] and loss of ganglion cells[4–9] from the myenteric plexus of the cardia. Failure of the normal receptive relaxation of the cardia in response to the swallowing of food is cited by Hurst[10] as the cause of cardiospasm. Cannon[11] demonstrated that this mechanism is abolished in cats following section of the vagi in the neck. By cutting the vagi in the thorax Knight[12] was able to reproduce the X-ray appearance of cardiospasm in anesthetized cats. In the course of a study of the motility of the

* Aided by a grant from the John and Mary R. Markle Foundation.

1 Sturtevant, M., *Arch. Int. Med.*, 1933, **51**, 714.

2 Heyrovsky, H., *Arch. f. klin. Chir.*, 1913, **100**, 703 (Quoted by Lendrum, *loc. cit.*).

3 Loeper, M., and Forestier, J., *Aech. d. mal de l'app. digestif.*, 1921, **11**, 306. (See Lendrum, *loc. cit.*)

4 Hurst, A. F., and Rake, G., *Quart. J. Med.*, 1930, **23**, 491.

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6 Beattie, W. J. H. M., *St. Bartholomew's Hosp. Rep.*, 1931, **64**, 39.

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10 Hurst, A. F., *J. A. M. A.*, 1934, **102**, 582.

11 Cannon, W. B., *Am. J. Physiol.*, 1904, **19**, 436.

12 Knight, G. C., *Brit. J. Surg.*, 1934, **22**, 155.