Myxobacteriales.[5] Following the key to the genera of the family Myxococcaceæ proposed by Stanier[5] the species should be placed in the genus *Chondrococcus,* because the fruiting bodies on agar are not deliquescent and are surrounded by a firm membrane. The species, therefore, may be properly named *Chondrococcus columnaris.* It should be pointed out, however, that the production of fruiting bodies in aqueous media is a new observation and is not considered in determining the systematic position of a myxobacterial species. It is possible that star formation, which was observed by Stapp and Bortels[6] and by Stanier[5] in liquid cultures of non-fruiting myxobacteria, is an analogous phenomenon.

We have begun an investigation of bacterial gill disease, a disease causing heavy loss of trout and salmon fingerlings. Cultures of myxobacteria have been consistently isolated from the gills of infected fish. These cultures, however, fail to produce organized fruiting bodies, and their systematic position has not yet been determined.

Summary. A myxobacterial species, *Chondrococcus columnaris,* has been isolated in pure culture and shown to produce a fatal disease in various fishes.

[5] Stanier, R. Y., *Bact. Rev.*, 1942, **6**, 143.

[6] Stapp, C., and Bortels, H., *Zent. Bakt. Parasitenk.*, 1934, II, **90**, 28.

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Relation of Sedimentation Rate to Amount of Precipitate Formed in Plasma by Type III Pneumococcus.*

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It has been known for many years that normal human sera contain considerable quantities of protein-polysaccharides.[1,2] According to Friedemann and Sutliff,[3] when sera from various pathologic states are inoculated with pneumococci, following an incubation period a voluminous white precipitate is obtained, while in normal sera only a slight cloudiness develops. This precipitate is not bacterial debris, but is caused by the production of unusually large quantities of acid which precipitate the serum proteins. The acids which are formed in normal sera by the organisms can be accounted for entirely by the free sugar which is fermented.[3] However, in abnormal sera growth continues at an undiminished rate after the free sugar has been completely utilized. Friedemann and Sutliff summarize their observations by stating that ". . . the phenomenon is due to the presence of abnormal quantities of a polysaccharide which supports rapid growth and which is readily fermented by the pneumococcus. It thus differs from the normally present polysaccharide, which is not apparently metabolized by the microörganism, since growth stops in normal sera when the free sugar is consumed."

These observations suggested the possibility that this abnormal polysaccharide which appeared in the blood might have some connection with the increased sedimentation rate of pathologic blood specimens. To investigate this, the following studies were carried out.

Methods and Results. Specimens of blood were obtained from a number of patients in the University Hospital, and normal blood samples were provided by members of the laboratory staff. Ten ml of blood were taken in the usual

* Submitted in partial fulfillment of the requirements for the degree of Doctor of Science in the University of Michigan.

[1] Rimington, C., *Biochem. J.*, 1929, **23**, 430.

[2] Hewitt, L. F., *Biochem. J.*, 1938, **32**, 1554.

[3] Friedemann, T. E., and Sutliff, W. D., *Science*, 1939, **90**, 335.

TABLE I.
Relation of Sedimentation Rate to Amount of Precipitate Formed in Plasma by the Growth of Type III Pneumococcus.

Clinical state	Precipitate formed in plasma inoculated with Type III pneumococcus*	Sedimentation rate in mm at the end of 1 hr
Normal	—	1
"	—	2
"	—	3
"	—	5
"	—	13
Ulcerative colitis	—	4
Essential hypertension	—	24
Neoplasm of stomach	+	8
Tuberculosis	+	30
"	++	3
Carcinoma of colon	++	75
Coronary infarction	++	75
Acute pharyngitis	+++	42
" rheumatic fever	+++	95
Pneumonitis (unknown etiology)	+++	112
Tuberculosis	++++	78
Lymphoblastoma	++++	80
Pneumococcus pneumonia	++++	110
Pneumonia (unknown etiology)	++++	113
Hypertension and upper resp. infection	++++	138

* Maximum precipitate following 48 to 96 hours of incubation.

manner from the median basilic vein and transferred promptly to a sterile tube containing 0.5 ml of 4.0% potassium oxalate; all specimens were kept at 4°C until used. Portions of each blood specimen were taken for the determination of the sedimentation rate within a few hours after they were obtained; a modified Westergren technic[4] was employed. The remainder of the blood specimens were centrifugated and the plasma removed.

A modification of the procedure Friedemann and Sutliff used with sera was applied to the specimens of plasma to determine the presence of non-specific polysaccharide. One ml of the clear plasma was transferred to a sterile serological tube and inoculated with 0.05 ml of a 12- to 18-hour 0.1% glucose broth culture of a strain of Type III pneumococcus. The tube was then incubated at 37°C and examined at frequent intervals.

The specimens of normal plasma became faintly cloudy (—); while plasma from the patients became either opalescent (+), almost opaque (++), opaque with a small amount of precipitate (+++), or contained a voluminous precipitate (++++). No attempts were made to determine the reducing sugar in the plasma to rule out any effect due to abnormal quantities of free sugar.

The data presented in Table I indicate a definite correlation between sedimentation rate and the amount of precipitate formed in plasma by the growth of Type III pneumococcus.

Discussion. According to Friedemann and Sutliff,[3] the precipitate formed in sera inoculated with pneumococcus is due to the fermentation of an abnormal polysaccharide only found in blood from certain disease conditions. The relation of the amount of precipitate and the sedimentation rate, as indicated in the present report, suggests that the abnormal carbohydrate may be responsible, at least in part, for the increased sedimentation rate of pathologic blood specimens. However, it must be recognized that increased sedimentation rate and large quantities of non-specific polysaccharide may occur simultaneously in the same blood specimen without having any bearing on each other. It is noted that similar correlations have been made by other workers between the fibrinogen or globulin level of the plasma and the sedimentation rate.

[4] Westergren, A., *Am. Rev. Tuberc.*, 1926, **14**, 94.

Recently, Seibert and her coworkers[5] have reported that a polysaccharide distinct from tubercle bacillus carbohydrate can be detected in the sera of individuals with tuberculosis. The extent of tuberculosis can be correlated with the amount of this polysaccharide in the serum. Interestingly enough, for many years clinicians have recognized the value of the sedimentation rate as an index of the progress of tuberculosis as well as other disease conditions. From the data given in the present report it is apparent that there is a definite relationship between sedimentation rate and an abnormal, non-specific polysaccharide in the plasma, and further work is planned to attempt to determine whether or not this polysaccharide is related to the carbohydrate investigated by Seibert and her coworkers.

[5] Seibert, F. B., Nelson, J. W., and Seibert, M. V., PROC. SOC. EXP. BIOL. AND MED., 1943, **52**, 219.

Summary. A relationship existed between the sedimentation rate of blood from various disease conditions and the amount of precipitate formed in plasma by the growth of Type III pneumococcus. The possible importance of the presence of an abnormal, non-specific polysaccharide to this finding is discussed.

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Effects of Pectin and Saline Solutions on Survival Time of Dogs in Hemorrhagic Hypotension.*†

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Recent observations of Middleton and Wiggers[1] indicated that intravenous infusions of pectin solutions were generally ineffective when given more than 30 minutes after reduction of mean arterial pressure to a sustained 50 mm Hg. level of arterial pressure. Given before the lapse of such an interval, but when significant hemodilution had already occurred, they caused a further immediate decrease in hemoconcentration due to the fluid injected. However, evidence that additional dilution or increase in blood volume occurs as a result of osmotic attraction of water was not discovered. On the contrary, a slight recovery of concentration in arterial blood generally supervened. Under such conditions, 8 out of 19 dogs reinfused at the end of a 30-minute period of hypotension failed to show a satisfactory dynamic response and died, but the remaining 11 showed a sustained elevation of arterial pressure and pressure pulses of good form from 4 to 6 hours after infusion of pectin solutions. But assuming that these animals would have recovered, only 58% would have been rescued by a comparatively prompt infusion of such solution.

The present investigation, a continuation of these studies, endeavored to determine the actual survival time when animals were transfused somewhat differently and under experimental conditions which might be expected to result in more favorable actions. For example, it is conceivable that most of the readily mobilizable body water has been drawn into the vascular system at the end of a 30-minute period of hypotension, with the result that no or little additional fluid can be reabsorbed when pectin solution with high colloid osmotic pressure is infused. For these reasons, it was felt that such infusions might act more favorably if more water were made available for reabsorption. To accomplish this

* This investigation was supported by a grant from the Commonwealth Fund.

† Condensed report of results presented as a thesis in partial fulfillment of requirements for the degree M.S. in the Graduate School of Western Reserve University.

[1] Middleton, S., and Wiggers, C. J., *Am. J. Physiol.*, 1943, **140**, 326.