STUDIES IN GLOMERULONEPHRITIS.

III. An Attempt to Produce Glomerulonephritis by Repeated Injections of Bacteria.

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Plate 54.

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The present paper records an effort to reproduce glomerular nephritis in animals by inoculations of bacteria, especially by repeated inoculations such as might be expected to evoke phenomena of immunity, allergy, or sensitization. The indication for the attempt is found in the hypotheses put forth by several writers in explanation of diffuse glomerulonephritis, especially that occurring as a late sequel or complication of scarlet fever.

Volhard and Fahr state that it is characteristic of diffuse nephritis that it appears at a time when immune processes are active and speak of a "diffuse toxic action." Ophüls has suggested that the glomerular endothelium acquires strong bacteriolytic properties which cause an "explosive" reaction when the bacteria are again introduced into the tissue, lesions being caused by disruption products of the bacteria. The most elaborate development of the theory is that of von Pirquet and Escherich and Schick, who apply it also to the other late complications of scarlet fever. It may be summarized as follows: At the beginning of the disease, during the acute symptoms, the virus circulates in the body, sensitizing the cells in the same way as do all the foreign proteins. The immune substances (ergins) begin to appear at the end of the 2nd week. At this time a reinvasion of the body from various foci occurs, a specific reaction or combination between antigen and antibody takes place, and this, after the latent period common to allergic reactions in general, leads to active signs and symptoms of the scarlatinal sequelæ. The latter actually occur in the 3rd and 4th weeks, between the 19th and 22nd days. It is also part of the theory that the smaller the amount of protein acting in the preliminary sensitization, the sharper will be the later allergic reaction.

Von Pirquet and Escherich and Schick state that in the case of scarlet fever experimental proof of the allergic hypothesis must await the discovery of the

specific virus of the disease, but Löhlein asserts that streptococci are either demonstrable or probably associated in all cases of diffuse nephritis, and it is commonly accepted (Holt and Howland) that many of the complications of scarlet fever, including nephritis, are probably due to secondary infection with streptococci.

It therefore seemed logical to attack this problem, in a way somewhat like that in which arthritis was previously studied, by investigating the effect upon the kidney of repeated inoculations of streptococci. In view of the fact that streptococci cause the production of only small amounts of recognizable antibodies in the serum, it was conceived that a more decisive test of the part played by humoral bacteriolysis[1] at least, would be furnished if bacteria (*Bacillus coli communior*) capable of evoking large amounts of serum antibodies were used. As a still further test, the effect of repeated large doses of these bacteria in a highly immunized and therefore resistant animal was investigated. Finally, several strains of *Staphylococcus aureus* were used for repeated inoculation.

EXPERIMENTAL.

With the exception of *Bacillus coli communior*, all the bacteria used in the following experiments were isolated from the throats of patients in the 1st week of scarlet fever. The first inoculation was usually from the second or third generation. In general, the organism found in greatest abundance was used. As a rule this was the streptococcus which was found in a majority of instances to be of the non-hemolytic type (*Streptococcus salivarius*). In a few instances a predominant growth of *Staphylococcus aureus* was obtained and once the Klebs-Loeffler bacillus outgrew the other varieties.

Rabbits were used exclusively, usually young females weighing from 1,200 to 1,800 gm.

All tissues were fixed for routine examination in Orth's solution, and in formalin for frozen sections and for sections to be stained for bacteria

[1] Here and elsewhere in this paper the term bacteriolysis is used to imply the proteolysis that is assumed to occur when bacterial antigens are subjected to the action of complement and antibody. Jobling and Petersen have recently shown that the phenomenon may be rather a process of solution than of proteolytic cleavage.

by the Giemsa method. All sections, except the last named, were stained by Van Gieson's method.

Giemsa stains were used on nearly all the kidneys, which were carefully examined, without success, for bacteria.

The results of the experiments are given in Table I.

In general the glomerular changes were strikingly slight, though a majority showed one or more of the minor changes commonly found in glomerular nephritis, such as swelling of the endothelium, increase of the endothelial nuclei, and small amounts of granular exudate in the capsular space. If the data are examined closely it will be seen that the extent of such changes bears no appreciable relation to the number of injections or the character of the bacteria injected. Lesions are perhaps somewhat more frequent in those animals which died spontaneously than in those which were killed. In the one or two instances where a true albuminous exudate into the capsular space occurred a coincident spontaneous nephritis was observed. This complicates matters and renders generalizations hazardous. Leaving out these cases, we are at the most justified only in saying that bacterial inoculation produced a very mild grade of intraglomerular damage, but nothing comparable to the diffuse extraglomerular nephritis seen in man, with its exudate of fibrin, serum, and cells into the capsular space.

For bacteriolytic experiments, organisms of the typhoid-colon group are, as is well known, much better suited than streptococci, and it was for this reason that the experiments of Series D with *Bacillus coli communior*, which is highly virulent for rabbits, were carried out. In at least three, and probably all, of the animals of this series, a high degree of immunity, measured in terms of agglutinins was induced (Table II). After immunization it was possible to inject such massive doses of bacteria as in untreated animals would cause sudden death. It was assumed on the basis of the modern belief in the unity of the so called sensitizer antibodies (Zinsser) and our agglutination tests that the animals had acquired greatly heightened powers of bacteriolysis and, hence, that the massive inoculations employed were followed by an inundation of the body with split bacterial protein. Indeed, marked prostration followed the injections and was taken as direct evidence of proteotoxic action. The glomerular changes in this series were perhaps slightly

TABLE I.

Renal Changes after Repeated Intravenous Injections of Bacteria.

Rabbit No.	Original weight of animal.	Culture used.	No. of injections.	Amounts of culture injected.	Day of experiment on which injections were given.	Interval between last injection and death.	Died or killed.	Combined weight of kidneys.	Renal changes.
Series A, *Streptococcus hemolyticus*.									
	gm.			*cc.*		*days*		*gm.*	
1	1,400	No. 1	4	1.0, 1.0, 1.0, 1.0	1, 5, 6, 7	2 hrs.	D.	9.9	Moderate increase of endothelial cells, some with pyknotic nuclei. Granular material in capsular space.
2	1,500	" 1	4	2.0, 2.0, 2.0, 2.0	1, 5, 6, 7	10	"	9.6	Slight desquamation of epithelium of Bowman's capsule.
3	1,500	" 1	4	0.05, 0.05, 0.05, 0.05	1, 5, 6, 7	9	"	8.6	Occasional red blood cells in capsular space. Occasional dilated loop in tufts. Changes slight.
4	1,500	" 1	4	0.02, 0.02, 0.02, 0.02	1, 5, 6, 7	5	"	9.0	Slight increase of endothelial cells and in places stratification of parietal layer of Bowman's capsule.
5	1,750	" 1	3	1.0, 1.0, 1.0	1, 2, 3	14	"	12.1	Considerable increase of endothelial cells of tufts. Some granular exudate in capsular space. Some hyaline casts.
6	1,750	" 1	3	2.0, 2.0, 2.0	1, 2, 3	18	"	10.0	A few glomeruli show increased endothelial cells.
7	1,200	" 1	5	10M.,*† 10M., 10M., 100M., $\frac{1}{3}$ Bl.	1, 2, 3, 33, 44	12	K.	10.8	Endothelium of glomeruli slightly swollen in places. No other notable changes.
8	1,800	" 1	3	10M., 10M., 10M.	1, 2, 3	4	D.	15.2	No notable changes.

9	1,600	No. 1	5	10M., 10M., 10M., 100M., ⅓ Bl.	1, 2, 3, 33, 44	12	K.	12.0	Moderate endothelial swelling. Occasional dilated empty loops.
10	1,750	" 1	5	20M., 40M., 80M., 100M., ⅓ Bl.	1, 2, 3, 26, 37	12	"	16.8	Moderate endothelial swelling. Occasional empty loops, not dilated.
11	1,700	" 1	5	20M., 40M., 80M., 100M., ⅓ Bl.	1, 2, 3, 26, 37	12	"	14.5	About same as No. 10.
12	600	" 1	6	⅙ Sl., ⅛ Sl., ¼ Sl., ¼ Sl., ½ Sl., ½ Sl.	1, 2, 3, 4, 5, 6	8	D.	4.7	Considerable granular exudate in capsular space.
13	750	" 1	5	⅙ Sl., ⅛ Sl., ¼ Sl., ¼ Sl., ⅓ Bl.	1, 2, 3, 4, 5	12	K.	7.8	Considerable endothelial proliferation and empty loops.
14	750	" 1	7	⅙ Sl., ⅛ Sl., ¼ Sl., ¼ Sl., ½ Sl., ½ Sl., ⅓ Bl.	1, 2, 3, 4, 5, 6, 7	12	"	10.3	Slight endothelial proliferation and some empty loops.
15	—	" S 1	1	6.0	1	½	D.	9.5	Leukocytes in glomeruli. Endothelial nuclei stain deeply.
16	1,300	" S 5	2	10.0, 6.0	1, 10	2	"	13.2	Glomeruli enlarged. Abundant granular and reticular exudate in capsular spaces. Endothelial nuclei stain deeply.
17	2,775	" Ch 1	6	5.0, 5.0, 5.0, 5.0, 5.0, 6.0	1, 6, 12, 19, 27, 33	11	K.	15.3	Moderate swelling and nuclear increase of glomerular endothelium. Yellowish pigment granules in epithelium and lumen of distal convoluted tubules.
18	3,425	" S 16	6	5.0, 5.0, 5.0, 5.0, 5.0, 6.0	1, 6, 13, 19, 27, 33	11	"	24.0	Marked swelling and nuclear increase of glomerular endothelium. Leukocytes in tufts increased. Many hyaline casts.
19	2,100	" S 17	6	5.0, 5.0, 5.0, 5.0, 5.0, 6.0	1, 6, 12, 19, 27, 33	11	"	15.4	Moderate swelling and nuclear increase of glomerular endothelium.
20	1,425	" N 1	7	6.0, 6.0, 6.0, 5.0, 5.0, 5.0, 6.0	1, 38, 44, 50, 57, 65, 71	11	"	—	Practically normal.

*Killed by heat (60°C., 1 hour).

†M. indicates million bacteria; Bl., Blake bottle, agar culture; Sl., agar slant culture. All doses are given in cc. of a 24 hour broth culture, unless otherwise specified.

TABLE I—*Continued.*

Rabbit No.	Original weight of animal.	Culture used.	No. of injections.	Amounts of culture injected.	Day of experiment on which injections were given.	Interval between last injection and death.	Died or killed.	Combined weight of Kidneys.	Renal changes.
				Series B, *Streptococcus viridans.*					
	gm.			*cc.*		*days*		*gm.*	
21	1,450	No. S 6	3	14.0, 5.0, 5.0	1, 11, 38	5	D.	12.2	Glomeruli swollen and bloodless. Capsular space obliterated. Endothelial nuclei increased in places.
22	1,450	" S 7	1	6.0	1	12	"	—	Practically normal.
23	1,540	" S 9	2	6.0, 6.0	1, 28	4	"	10.5	No notable changes.
24	2,900	" S 6	6	6.0, 5.0, 6.0, 5.0, 5.0, 6.0	1, 6, 12, 19, 27, 33	11	K.	16.3	" " "
				Series C, *Staphylococcus aureus.*					
25	2,050	No. S 8	1	6.0	1	1	D.	19.5	Glomeruli large. Endothelium markedly swollen. Occasional leukocytes in capsular space. Many hyaline casts.
26	2,100	" S 10	1	6.0	1	1	"	17.2	No notable changes except spontaneous nephritis.
27	2,700	" S 10	5	2.0, 5.0, 5.0, 3.0, 3.0	1, 7, 12, 19, 34	10	K.	15.5	Glomeruli greatly congested, but endothelium not swollen. Nuclei somewhat increased. Occasional red blood cells in capsular space.
28	2,700	" S 12	6	2.0, 5.0, 5.0, 3.0, 3.0, 3.0	1, 7, 12, 19, 27, 34	10	"	14.2	No notable changes.
29	2,725	" S 10	6	2.0, 5.0, 3.0, 3.0, 3.0, 3.0	1, 7, 12, 19, 27, 34	10	"	16.8	Moderate increase of endothelial nuclei. No other notable changes.

Series D, *B. coli communior.*

30	1,700	*B. c. c.*	5	10M., 10M., 10M., 100M., 1.0	1, 2, 3, 33, 44	12	K.	15.4	Dilated empty loops and swollen epithelium in many tufts. Occasional pyknotic nuclei. Considerable granular exudate in capsular space.
31	1,500	"	5	10M., 10M., 10M., 100M., 1.0	1, 2, 3, 33, 44	12	"	10.7	Moderate swelling and multiplication of endothelium. Occasional dilated empty loops.
32	1,600	"	4	20M., 40M., 80M., 100M.	1, 2, 3, 26	10	D.	13.1	Marked increase of endothelial nuclei. No other marked change.
33	1,600	"	5	20M., 40M., 80M., 100M., 1.0	1, 2, 3, 26, 37	12	K.	12.2	Many empty loops but no marked endothelial swelling.
34	1,250	"	1	$\frac{1}{100}$ Bl.	1	2	D.	—	General capillary and venous engorgement. Occasional red blood cells in capsular space. A few hyaline casts.
35	1,600	"	10	$\frac{1}{100}$ Bl., $\frac{1}{100}$ Bl., $\frac{1}{100}$ Bl., $\frac{1}{3}$ Sl., $\frac{2}{3}$ Sl., $\frac{2}{3}$ Sl., 1 Sl., 2 Sl., $\frac{1}{3}$ Bl., $\frac{1}{3}$ Bl.	1, 6, 12, 20, 21, 22, 23, 24, 25, 27	3	"	13.0	Very marked spontaneous nephritis. In fibrotic areas, many of the capsular spaces of the glomeruli contain yellow-staining albuminous exudate, and several others are greatly dilated and contain a non-staining homogeneous exudate. Most of the glomeruli are enlarged, bloodless, with swollen epithelium. Many eosinophils in glomerular and intertubular capillaries. No bacteria found.
36	1,060	"	7	$\frac{1}{100}$ Bl., $\frac{1}{100}$ Bl., $\frac{1}{100}$ Bl., $\frac{1}{3}$ Sl., $\frac{2}{3}$ Sl., $\frac{2}{3}$ Sl., 1 Sl.	1, 6, 12, 20, 21, 22, 23	1	"	12.4	Glomeruli enlarged with many dilated empty loops. Endothelium swollen. Some pyknotic nuclei. No exudate in capsular spaces.
37	1,460	"	9	$\frac{1}{100}$ Bl., $\frac{1}{100}$ Bl., $\frac{1}{100}$ Bl., $\frac{1}{3}$ Sl., $\frac{2}{3}$ Sl., $\frac{2}{3}$ Sl., 1 Sl., 2 Sl., $\frac{1}{3}$ Sl.	1, 6, 12, 20, 21, 22, 23, 24, 25	1	"	14.1	Glomeruli moderately enlarged. Slight granular exudate occasionally found in capsular spaces but no marked or extensive glomerular changes.

more marked than in the series in which streptococci and staphylococci were used, but again they failed to correspond in intensity with the number or the amounts of the injections. Thus, Rabbit 37, which received six large injections, suffered but a small amount of glomerular injury. On the whole, it is difficult in our experiments to find support for the bacteriolytic theory of glomerulonephritis.

The fate of bacteria in the kidney is a matter of some interest, but could not be fully investigated in the present work. However, a few observations bearing on this point were made in connection with experiments, previously reported, when successive injections of diphtheria toxin and *Bacillus coli communior* were given. Large amounts of bacteria injected intravenously led to rapid death of the animal. The bacilli were found in dense clumps (Fig. 1), mostly in the glomeruli and

TABLE II.

Agglutination Tested for Rabbits 35, 36, and 37 on the 17th Day after the First Injection.

Rabbit No.	Dilution.										
	1:25	1:50	1:100	1:250	1:500	1:1,000	1:2,500	1:5,000	1:10,000	1:25,000	1:50,000
35	±	+	+	++	+++	+++	++	+	+	±	0
36	+	+	+++	++	+++	+++	+	++	±	++	+++
37	+	+	+	++	++	+++	++	++	+	++	+

occasionally in the intertubular capillaries of the cortex and medulla. In these animals few leukocytes and little evidence of inflammatory reaction were found in the tufts. In animals dying 12 to 48 hours after inoculation the bacteria had usually disappeared and leukocytes, generally in large numbers, were found in the loops. In most of these animals there was a rather marked inflammatory reaction in the glomeruli. It was at first thought that a local bacteriolysis, by leukocytes, with the formation locally of poisonous bacterial disintegration products might be the cause of the inflammatory reaction, but against this was the fact that even with enormous doses (sediment of 30 cc. of broth culture) only a few glomeruli contained demonstrable organisms, whereas the inflammatory reaction involved a majority of the tufts. It was therefore assumed that the toxic substances with their diffuse action,

were circulating in the blood. It does, however, seem fairly probable that the bacteria were occluded and removed by leukocytes, and in one instance this was directly observed (Fig. 2).

The theories which we are examining are closely dependent upon an assumed bacteriolysis of streptococci and it is necessary to examine closely the question whether lysis of the streptococcus can experimentally be shown to occur. To such physical and chemical procedures as freezing and thawing, and extraction with distilled water and weak alkali the streptococcus has, in my hands, proved to be extraordinarily resistant and experiments to be described below indicate that it is correspondingly resistant also to lysis by the Pfeiffer procedure, and by immune serum and complement. The contrast in these respects with the typhoid-colon group is, in fact, so great as to suggest a division of bacteria into what might be called lysostable and lysolabile groups.

In a previous paper (Faber) on experimental arthritis in rabbits it may be recalled that injection of streptococci into the knee-joints of rabbits was followed by a condition designated as sensitization, which was revealed by the occurrence of a local reaction when homologous organisms were later injected intravenously. The term sensitization was purposely chosen to indicate altered properties of the cells of the capsular synovium, without offering a more explicit interpretation of the exact process, whether it was bacteriolysis with the local formation of toxic bacterial disruption products, or an increase in the normal defensive leukocyte-attracting properties of the cells, or a process related in some way to a disturbed cellular ferment balance consequent to local specific antibody-antigen combinations. In point of fact, certain unpublished experiments were carried out at that time in an attempt to elucidate the problem in one of these directions, and may be cited here.

Pfeiffer's Phenomenon for Joints.

Rabbit 40.—Left knee injected with mixture of 0.1 cc. of immune serum + 1 loop of *Streptococcus viridans* + 0.9 cc. of 0.85 per cent salt solution.

Right knee injected with mixture of 1 cc. of sterile bouillon + 1 loop of *Streptococcus viridans.*

Both knees aspirated after 1 hour. Cultures: left knee, heavy growth of streptococcus; right knee, no growth.

Rabbit 41.—Left knee injected with mixture of 0.1 cc. of normal rabbit serum + 0.9 cc. of salt solution + 1 loop of *Streptococcus viridans.*

Right knee injected with mixture of 1 cc. of bouillon + 1 loop of *Streptococcus viridans.*

Both knees aspirated after 1 hour. Both cultures sterile.

The immune serum was obtained from a rabbit which had received two injections of living streptococcus. Complement fixation was positive in the serum. This rabbit developed an acute arthritis immediately after receiving a third injection (shortly after the time that blood for the above serum was drawn).

In connection with the above experiments, the following cultural tests were made.

(*a*) 1 loop of *Streptococcus viridans* + 1 cc. of immune serum. Incubated 1 hour and plated. Result scanty growth.

(*b*) 1 loop of *Streptococcus viridans* + 1 cc. of normal serum. Incubated 1 hour and plated. Result heavy growth.

(*c*) 1 loop of *Streptococcus viridans* plated. Result heavy growth.

These experiments, though too few to be decisive, suggest the following conclusions. (1) Immune streptococcus serum not only fails to evoke the Pfeiffer phenomenon, but may even favor the bacteria at the expense of the tissues. (2) The normal synovial membrane has considerable power to combat infection. (3) Immune serum apparently has the power of partially inhibiting growth *in vitro.*

The third point might be used as an indication of bacteriolysis and was therefore tested more completely in another series of experiments which are given in Table III.

The following points may be emphasized. (1) The lowest counts were obtained in those plates in which the dilution of the serum had been respectively 1:30 and 1:50. The maximum agglutination was obtained with a dilution of 1:40. (2) The lowering of the bacterial count was independent of the amount of complement added. (3) Lowering of the bacterial count was obtained without complement and in two instances was greater than in any of the tubes to which complement had been added.

These experiments strongly suggest that we are dealing not with bacteriolysis proper but with an agglutination phenomenon. It is

obvious that an agglutinated clump of bacteria will give, but a single colony and that agglutination in this way will cause an apparent reduction of the bacterial count when the pour-plate method is used.

TABLE III.

Tests for Bacteriolysis with Streptococcus viridans, Complement, and Immune Serum.

Complement.	Row.	Cc. of immune serum.						
		0.1	0.08	0.05	0.03	0.01	0.007	0.00
		Dilution.						
		1:15	1:20	1:30	1:50	1:150	1:200	—
		Tube.						
		1	2	3	4	5	6	7
cc.								
0.5	A	180	150	125	180	225	180	300
0.3	B	170	250	105	110	200	180	
0.1	C	160	160	75	40	140	160	
0.07	D	125	150	90	150	150	175	
0.04	E	90	150	110	19	100	110	
0.00	F	160						375

The figures represent thousand colonies per plate.

To each tube was added 0.5 cc. of a suspension of streptococci containing 750,000 bacteria per cc. All the tubes before incubation were made up to equal volume with 0.85 per cent sodium chloride solution.

DISCUSSION.

It may be assumed that when bacteria are introduced into a tissue the mechanism of defense is one of physical removal, depending on the leukotactic activity of the tissue or bacteria in question (Fig. 2). It may also be assumed that this activity is heightened by previous exposure of the tissue to the infecting agent and that the reaction will usually be accompanied by such other reactive phenomena as vasodilation and exudation of fluid. Bacteriolysis of streptococci *in situ* and the local production of toxic disintegration products is at the least an improbable explanation of the phenomenon.

The conclusions to be derived from the experiments on joints may perhaps fairly be applied to the kidney, save that in the latter we have obtained much less evidence of reactivity. It is evident that the

arrangement of the glomerular capillaries is such that small bacterial emboli can be expeditiously removed by phagocytes and that there is in the tuft no closed space in which inflammatory exudate can collect. It may, indeed, be doubted whether these emboli are permitted to remain long enough in contact with the glomerular endothelium to set up a state of sensitization. In none of the rabbits examined were bacterial emboli found more than 12 hours after injection and then in only one instance when the animal had been profoundly intoxicated with diphtheria toxin.

The capsular space, on the other hand, does supply conditions of relatively poor drainage which are increased when the excretion of water from the tuft is interfered with. Apparently, it is when the lining epithelium of Bowman's capsule has been damaged and permits toxic substances to pass through that leukocytes, fibrin, and the other materials of inflammatory exudate are attracted to the capsular space and so produce the picture of extraglomerular nephritis. This picture, it will be recalled, was produced when an injection of bacteria followed injury by diphtheria toxin. The present series of experiments with their consistently negative results constitutes strong evidence that bacteria alone are unable to produce extraglomerular nephritis, even by cumulative action or by immune bacteriolysis, since on the one hand a bacterial species known to be subject to immune bacteriolysis failed to produce the lesions under favorable conditions, and since on the other hand the organism commonly found in this disease has been shown to be extraordinarily resistant to bacteriolysis.

The evidence of the present experiments and the arguments presented against the bacteriolytic theory would also appear to apply equally well to the assumption of a local allergic reaction in the glomerular endothelium.

Embolism, even in an immune host where agglutination *in vivo* may be assumed to occur, is too sporadic in the kidney to explain lesions as widespread and evenly distributed as occur in the human disease, and it seems more than ever necessary to assume the action of a soluble poison in the blood as the immediate pathogenetic factor. For the present there is no direct proof of the nature or of the origin of this poison.

The local concentration of poisons in the tuft, whatever may be their source or nature, may, however, still be held to be the immediate cause for the occurrence of lesions at this point.

SUMMARY.

Repeated injections into the blood stream of streptococci and staphylococci derived from cases of scarlet fever, and of *Bacillus coli communior* failed to produce typical glomerulonephritis even when immune antibodies could be demonstrated in the serum in high dilutions. Bacteriolysis of streptococci was not found by the usual tests *in vitro* or by the Pfeiffer procedure. It is therefore concluded that the weight of evidence is against the theory that glomerulonephritis is due to immune bacteriolysis of streptococci. The experiments also failed to give any support to the hypothesis of allergy or of sensitization as a factor in the production of the disease. Evidence is presented to show that bacterial emboli are rapidly removed from the glomerular capillaries by leukocytes, and that this embolism, even after injections of enormous quantities of bacteria, affects but a small proportion of the glomeruli. It is again suggested that a circulating poison in the soluble state is responsible directly for the disease in question.

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EXPLANATION OF PLATE 54.

FIG. 1. Rabbit 38. Intravenous injection of the sediment of 30 cc. of a broth culture of *B. coli communior*. Died 1 hour later. Large bacterial embolus in glomerulus and its afferent vessel. Giemsa stain. Leitz obj. 6.

FIG. 2. Rabbit 39. Intravenous injection of one agar slant of *B. coli communior*. Died 12 hours later. Bacterial embolus in glomerulus. Adjacent to this is a polymorphonuclear leukocyte containing a bacillus. Giemsa stain. Obj. oil immersion.

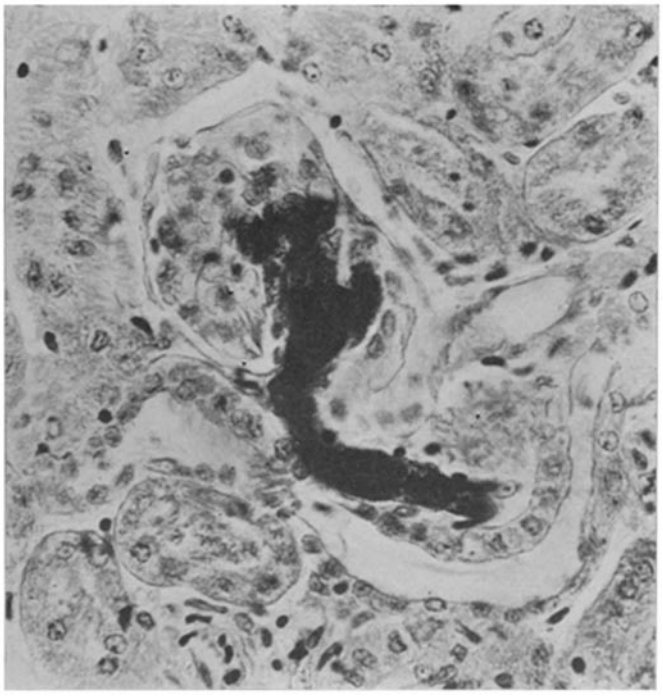

FIG. 1.

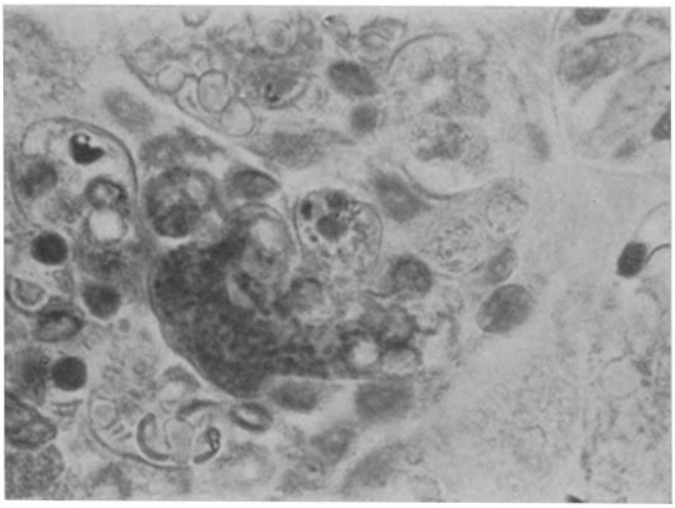

FIG. 2.

(Faber and Murray: Studies in glomerulonephritis. III.)