A phenomenon worthy of mention is the disappearance (de-differentiation) of some secondary proctodea. The pigmented pit and its elevated border may regress to a flat pigmented spot which may in turn disappear. On the other hand, some induced proctodea persist and eventually communicate with the gut lumen.

Summary. Transplantation experiments show that the capacity of ventral blastoporal lips to induce a proctodeum is inhibited by the proximity of the rudiments of the axial organs. This suggests an explanation as to why, in previous experiments, proctodea were induced by only 50% of the transplants, and for the observed ventral position of accessory proctodea.

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Recently Isolated Strains of Pneumococci as Incitants of Experimental Pneumonia in Rats.*

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Although the infectivity of recently isolated strains of pneumococci has been tested both in routine injection of mice for type-identification, and in special studies,[1] the difference in ability of recovered strains to produce experimental pneumonia in animals has not been determined.

We have compared experimental pneumonic infections in rats with the clinical course in the patient from whom the pneumococci were isolated, and have correlated certain characteristics of the disease in man and rat with the type of pneumococcus involved. Cultures were obtained from 23 patients with pneumonia, and from one with otitis media during the winters of 1937-38 by Mr. Gerhardt Burde, of the Michigan State Department of Health. The pneumococci were retyped, and used in these experiments within 2 weeks of their initial isolation.

Rats were inoculated intrabronchially in the left lobe of the lung with 10^{-5} cc of cultures of these organisms suspended in sterile mucin, according to a method previously described.[2] Surviving

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1 Rosenau, M. J., Felton, L. D., and Atwater, R. M., *Am. J. Hyg.*, 1926, **6**, 463.

2 Nungester, W. J., and Jourdonais, L. F., *J. Infect. Dis.*, 1936, **59**, 258.

TABLE I.
Correlation of Clinical Pneumonia with Experimental Pneumonia Produced in Rats, Using Strains of Pneumococci Obtained from Patients.

Case No.	Type	Clinical course of patient	Number of rats: Inoc.	Indicated percent of lobe involved: 90-100%	25-90%	0-25%	Marked pleurisy	Died		"Toxin" tested in mice; mld in g†
33	1	Mild	2	2			1	1		.030
22	1	Moderate	3	2			3	0		.004
30	1	"	2	1		1	0	0		.030
23	1	Severe	3	3			3	1		.060
39	1	"	2	1		1	0	0		.030
34	1	" D*	3	2	1	1	1	0		.030
27	1	(Otitis media) D	2	2			1	1		.030
		Total	17	13 (76%)	1	3	9 (53%)	3 (18%)	Avg	.031
50	2	Mild	3	2	1		2	3		.020
31	2	Moderate	2	2			0	2		.030
28	2	Severe	2	1	1		1	2		.010
26	2	" D	2	1	1		1	2		.015
49	2	" D	2	2			1	2		.030
14	2	" D	3	2	1		0	3		.030
		Total	14	10 (72%)	4		5 (36%)	14 (100%)	Avg	.023
9	3	Severe	3	2	1		1	2		.020
37	5	"	3	1	1		0	0		.005
12	6	"	3	1		1	0	1		.030
18	6	"	3	3			0	0		.010
7	7	"	3	2		1	1	0		
10	10	Mild	3	3			2	3		.020
32	13	"	3	3			0	0		.030
13	15	Severe D	3	3		1	0	0		.015
16	19	"	3		2	1	0	0		.030
36	19	Moderate	2	2			0	0		.005
8	27	Mild	3	2		1	0	1		.030
		Total	32	22 (69%)	4	5	4 (13%)	7 (22%)	Avg	.019

*D—Patient died.

†The toxins were prepared and tested by Burde, Barret, and Glassen. The authors are grateful for the privilege of including this material.

animals were etherized on the seventh day after inoculation. All animals were necropsied, and gross changes were observed.

In Table I the severity of the disease in the patient, as well as in the experimentally inoculated rats, is indicated. The number of animals showing varying degrees of consolidation of the infected lobe, the number having marked pleurisy, and the number that died are recorded. Also, an estimation is given of the minimal lethal dose of "toxins" produced from the same strains of pneumococci

by Burde, Barret and Glassen,[3] according to the procedure described by Dick and Boor.[4]

It will be noted that in the group of 17 rats infected with Type 1 pneumococci, the mortality was 18%, whereas in a group of 14 animals infected with Type 2 strains, the mortality was 100%. This difference parallels the severity of the disease in the patients from whom the organisms were isolated. Further, we have been informed by Dr. Alvin Price that among the patients with lobar pneumonia at Receiving Hospital, Detroit, during the winters of 1937-38, a mortality of 19% was noted in the 42 patients with Type 1 pneumonia, while among the 68 patients with Type 2 infections, the mortality was 35%. Thus the differences observed in the rats are seen to be in agreement with the clinical findings in this geographical region during that period. The mortality of rats inoculated with available strains of other types of pneumococci was about 22%.

The incidence of pleurisy was somewhat higher in Type 1 than in Type 2 infections. This was in contrast to the results already discussed on the difference in mortality produced by the two types, and may be accounted for by the fact that the average survival of rats that died was 4.6 days in the Type 2, and 2.8 days in the Type 1 group. Further, 82% of the animals inoculated with Type 1 pneumococci were etherized after surviving for 7 days. None in the Type 2 group lived for this length of time. It has been suggested that at least 2 or 3 days are required for the development of marked pleurisy in experimental lesions in rats.[2] Nevertheless, the development of pleurisy is dependent in part on the organism. This is indicated by the fact that a very low incidence (13%) of pleurisy was noted in the group of rats inoculated with strains of pneumococci Types 3 to 27. In this group, 78% of the animals survived until sacrificed at 7 days. The average survival of these animals that died was 3.7 days.

These factors, time of survival of the rat and strain of pneumococcus, which affected the spread of infection to the pleura, did not control the spread of the pneumonic process within the pulmonary parenchyma, however. It will be noted that the incidence of involvement from 90 to 100% of the inoculated left lobe in the 3 groups of animals, Type 1, Type 2, and Types 3 to 27, was approximately the same.

Histological sections from the lungs of 6 animals infected with Type 1, 5 with Type 2, and 8 with other types, were made and

[3] Personal communication.

[4] Dick, G. F., and Boor, A. K., *J. Infect. Dis.*, 1937, **61**, 228.

examined for the amount of edema in the tissue, the amount of red-cell infiltration into alveoli, the number of alveoli completely filled with cellular exudate, necrosis of pulmonary parenchyma, and the severity of bronchitis. No significant differences could be attributed to the type of pneumococcus producing the experimental lesion. It is not advisable to place too much emphasis on the histopathology because of the limited number of infected lungs studied.

There was complete lack of agreement in the titer of "toxins" as prepared by Burde, Barret and Glassen[3] and the severity of the experimental lesions. For example, in the third group of rats, seen in Table I, the mortality of those inoculated with pneumococci which yielded a stronger than average "toxin" was 0%; for the strains producing a weaker than average "toxin", 16%; and the mortality produced by organisms with toxins having mld's equal to the average mld was 83%.

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Influence of Lactogenic Preparations on Mammary Glands and Time of Vaginal Opening in Young Rats.*

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In studying the cycle-inhibiting action of lactogenic hormone in the rat, it has been of interest also to observe changes brought about indirectly by this hormone on the mammary apparatus. Previously, it had been shown[1] that crude anterior lobe extracts (now known to have a high lactogenic hormone content) caused lobular mammary development in normal but not oöphorectomized rats. This suggested an indirect stimulation of the mammary gland by a pituitary factor acting possibly through the luteal tissue often induced in excess by such extracts. More recent work[2] on the hypophysectomized rat has indicated that whereas the follicle-stimulating and luteinizing

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1 Evans, H. M., and Simpson, M. E., *Am. J. Physiol.*, 1931, **98**, 511.

2 Evans, H. M., Simpson, M. E., Lyons, W. R., and Turpeinen, K., *Endocrinol.*, 1941, **28**, 933.