found to contain spheres up to 500 μ in diameter. Spheres larger than 500 μ were lodged in the arteries and did not pass to the veins.

The anatomical location of the A-V shunts was ascertained from radiographs of the lungs injected with radiopaque media, in which the course of the vessels and the spheres themselves could be visualized; from dissections of the latex-injected lungs; and from the casts of the specimens injected with vinyl acetate. No A-V shunts were present in the hilar region nor along the course of the larger branches of the pulmonary artery and vein. At the apex of the lobular subdivision of the bronchopulmonary segments, however, shunts up to 500 μ in diameter were found. Smaller shunts (50-100 μ) were located at the level of the smaller bronchi and respiratory bronchioles, and the smallest, (20-25 μ) near the alveolar sacs and the alveoli. Shunts up to 200 μ in diameter also occurred in the pleura.

Comment. Perhaps a larger number of spheres and a higher percentage of successful passages of spheres through the pulmonary veins might have been found if gelatin, or some other solution of greater viscosity than the saline perfusate, had been used, since many of the spheres settled in the more dependent parts of the arteries and veins. Such viscous solutions, however, would have interfered with the subsequent injection of latex or vinyl acetate. Unless the A-V shunts were relatively large, they could not be demonstrated by the vinyl acetate technic, apparently due to the shrinkage and fixation of the vessels by the pre-injection acetone perfusion or the acetone content (approximately 80%) of the vinyl acetate and the small percentage (less than 20) of vinyl resin remaining after the specimen was macerated.

Summary. Arteriovenous shunts, many times the accepted diameter of capillaries, were demonstrated in isolated human lungs with little or no pulmonary pathology, by the passage of glass spheres from the pulmonary artery to or through the pulmonary veins. Injection of these vessels with liquid latex or vinyl acetate indicates that the shunts are located at the apex of and within the lobular divisions of the lungs.

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Antigenic Pattern of Strains of Influenza A and B. (18361)

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Studies on the antigenic pattern of influenza A and B viruses have revealed heterogeneity in each of these groups(1-5). The significance of this multiformity as it relates to the occurrence and recurrence of strains of a particular antigenic composition in the human population and to the selection of viruses for use in the vaccine has not been fully evaluated. The present communication presents data on the antigenic patterns of a number of strains of influenza A and B which have been recovered since 1933.

Materials and methods. Virus strains. Certain pertinent information about the viruses used in the present work is summarized in Table I. Additional data are given in Table II on the strains designated CAW-1, CAW-2, CAW-3, CAW-4A, CAW-5, Canadian Arctic 1A and Canadian Arctic 12A which were recovered from specimens collected from Es-

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1. Magill, T. P., and Francis, T., Jr., *Brit. J. Exp. Path.*, 1938, v19, 273.

2. Francis, T., Jr., and Magill, T. P., *Brit. J. Exp. Path.*, 1938, v19, 284.

3. Smith, W., and Andrewes, C. H., *Brit. J. Exp. Path.*, 1938, v19, 293.

4. Gordon, I., *J. Immunol.*, 1942, v44, 231.

5. Burnet, F. M., Beveridge, W. I. B., Bull, D. R., *Aust. J. Exp. Biol. and Med. Sci.*, 1944, v22, 9.

TABLE I. Data on Strains of Influenza A and B Included in Studies.

Type	Strain	Year isolated	Passage history	Origin
A	WS	1933	F?/M?/>E5	England
"	PR8 (ISSC)*	34	F1/M691/E7-8	Puerto Rico
"	Hyde	35	F?/M?/>E9	Probably Alaska
"	Henry	36-37	M32/E4	N. Y.
"	Hickcox	40-41	F1/M12/E4	N. Y.
"	Weiss	43	M79/E6-7	Mich.
"	FM1 (ISSC)*	47	M7/E15-17	N. J.
"	Hume	47	E7	Md.
"	Wilfong	48	E21-22	Calif.
"	4ML-1-48/49	48-49	>E2	Heidelberg, Germany
"	Canadian Arctic 1A	49	E3	Victoria Island, Can
"	Canadian Arctic 12A	49	E3	" " "
"	CAW-1	49	E3	" " "
"	CAW-2	49	E3	" " "
"	CAW-3	49	E3	" " "
"	CAW-4A	49	E3	" " "
"	CAW-5	49	E3	" " "
"	SF-1-49	49	E6-7	Calif.
"	AH-1-50	50	E2-3	Ga.
"	Berkeley-1-50	50	F1/E5	Calif.
"	FW-1-50	50	E3	Wyoming
B	Lee (ISSC)*	40	F8/M310/E7-8	N. Y.
"	Hawaii	44-45	>E3	Hawaii
"	Czech	45	E6-7	Mass.
"	Warner	48	>E4	Australia
"	Seattle-2-49	49	E8	Washington
"	Gt. Lakes 34	50	E6-8	Ill.
"	Gt. Lakes 38	50	E5	"
"	Gt. Lakes 40	50	E4-5	"
"	IB1	50	E7	N. Y.
"	L1-B-50	50	E8	N. J.
"	MB	50	E6-9	N. Y.
"	Meguro	50	E7-8	Japan
"	POOH	50	E14-16	"

F = Ferret, M = Mouse, E = Egg.
* Standard diagnostic strains of the Influenza Strain Study Center, Commission on Influenza, Armed Forces Epidemiological Board.

TABLE II. Data on Strains from Victoria Island Outbreak, April 1949.

Strain	Isolated by	Isolated from patient		Material
		Ref. #6	Ref. #7	
CAW-1	Nagler	B.T.	Too	Nasopharyngeal wash.
CAW-2	"	M.C.	Car	" "
CAW-3	"	A.W.	Wil	" "
CAW-4A	"	B.P.	—	Heart blood-autopsy
CAW-5	"	M.M.†	Meg‡	Lung autopsy
C.A.* 1A	van Rooyen	—	Ban	Nasopharyngeal wash.
C.A.* 12A	" "	—	Meg‡	Lung autopsy

* C.A. = Canadian Arctic.
† M.M. of Table II (not Table I) in reference 6.
‡ Strains CAW-5 and C.A. 12A were isolated in independent laboratories from lung material of patient Meg (M.M.).
This table compiled from information furnished by Drs. F. P. Nagler and C. E. van Rooyen.

kimos on Victoria Island, N.W.T., Canada during April 1949(6,7). The epidemic in the Arctic was characterized(6) as "an unusually virulent outbreak of influenza, reminiscent of the 1918 pandemic" in which there was an attack rate of 100% and case fatality rate of

6. Nagler, F. P., van Rooyen, C. E., and Sturdy, J. H., *Canad. J. Pub. Health,* 1949, v40, 457.

7. van Rooyen, C. E., McClelland, L., and Campbell, E. K., *Canad. J. Pub. Health,* 1949, v40, 447.

20% in the 90 Eskimo inhabitants of the island. In several instances, the same patients and materials were given different designations by Dr. F. P. Nagler and Dr. C. E. van Rooyen. Only two of the strains in the present study, *i.e.*, CAW-5 and Canadian Arctic 12A, were recovered from the same patient (M.M. or Meg.).

Hyde and Hume viruses(8) were furnished by Dr. B. Eddy. The Henry and Hickcox strains were obtained from Dr. T. P. Magill and the Berkeley-1-50, from Dr. G. Meikeljohn. The sources of the remaining A type strains used in the present study were given in an earlier report(9). It may be mentioned that our FW-1-50 strain(9) is sometimes designated Cuppett by other workers. The Lee and Czech strains of influenza B virus were furnished by Dr. T. P. Magill and the L1-B-50 and Seattle-2-49 by Drs. M. M. Sigel and A. S. Lazarus, respectively. The Meguro and POOH viruses, isolated at the 406th Medical General Laboratory, Tokyo, Japan, were sent by Major K. F. Burns, V. C., U.S.A. and Dr. F. L. Horsfall supplied the MB and IBI viruses. The Warner agent was obtained from Dr. S. G. Anderson and the Hawaii strain, isolated at the 18th Medical General Laboratory, Honolulu, Hawaii, was from the strain files of this department. The Great Lakes 34, 38 and 40 viruses, from a recent outbreak of influenza B at the Great Lakes Naval Training Station, Illinois, were furnished by Commander John R. Seal, M.C., U.S.N.

Sera. In general, pairs of roosters weighing 5 to 8 lb were injected on one occasion by the intravenous and intraperitoneal routes with 5.0 ml amounts of crude infected allantoic fluid which had been freshly harvested. However, birds receiving MB, IBI, POOH, L1-B-50 or Seattle-2-49 viruses were inoculated with these volumes on 2 successive days since it was found that the single inoculation failed to evoke a satisfactory antibody response to these agents. The roosters were bled 10 days following the initial injection and their sera stored in lyophilized form until used in the tests. Sera against strains IBI, Warner, POOH, L1-B-50 and SF-1-49 titered 1:400 with homologous antigen; all other sera titered 1:800 or greater. Sera obtained from roosters prior to immunization did not contain demonstrable antibody against influenza virus.

Hemagglutination technic. The hemagglutination and hemagglutination-inhibition titrations were performed by a technic which conformed to the standard diagnostic method recommended by the Committee on Serological Procedure of the Armed Forces Epidemiological Board(10). In the latter procedure, 0.25 ml volumes of serial dilutions of serum were mixed with 0.25 ml of virus (4 units) and 0.5 ml of 0.5% human type "O" red cell suspension. The pattern of sedimented cells was read after 50 minutes and the titer taken as the highest initial dilution of serum giving complete inhibition of hemagglutination. The antigens were preserved by lyophilization and rehydrated within 24 hours of their use in the tests. In the tables, the serum titers were adjusted to give a 1:800 titer with homologous virus so that the results with the individual sera could be compared more easily.

Results. Influenza A. Tables III and IV summarize the results of tests with strains of influenza A. Table III gives the numerical values for certain of the serum titers summarized in interpretive form in Table IV. In the latter table, a titer of 1:800 is represented by 4+, 1:400 by 3+, 1:200 by 2+, 1:100 by 1+ and 1:50 by ±. In 3 instances the adjusted serum titer obtained in tests with heterologous antigen was 1:1600, *i.e.*, 2 times as high with homologous antigen. This difference was not considered significant, and for the purpose of simplification, this titer was represented as 1:800 (4+) in the table. The values given in the tables were obtained in tests with single serum specimens. Essentially identical cross-reactions were obtained in those instances in which viral anti-

8. Ward, T. G., and Eddy, B. E., *Science*, 1950, v112, 501.

9. Hilleman, M. R., Mason, R. P., and Rogers, N. G., *Public Health Repts.*, 1950, v65, 771.

10. Committee on Standard Serological Procedures in Influenza Studies, *J. Immunol.*, 1950, v65, 347.

TABLE III. Numerical Summary of Agglutination-Inhibition Tests with Representative A Type Influenza Strains.

Rooster antisera	Serum titers obtained with antigens: WS (1933)	CAW-3 (1949)	PR8 (1934)	FM1 (1947)	CAW-5 (1949)	Wilfong (1948)	FW-1-50 (1950)
WS 1933	*800**	0	400	50	200	0	0
CAW-3 '49	0	*800*	800	0	50	0	0
PR8 '34	0	200	*800*	0	0	0	0
FM1 '47	0	0	50	*800*	800	800	50
CAW-5 '49	ND	0	100	800	*800*	800	100
Wilfong '48	0	50	100	400	800	*800*	200
FW-1-50 '50	0	0	50	400	800	800	*800*

* All titers expressed as reciprocals of fractions.
ND = Not done.

TABLE IV. Interpretive Summary of Agglutination-Inhibition Tests Showing Relationships of Influenza A Strains.

Rooster antisera	Antigens: WS 1933	CAW-3 1949	PR8-ISSC 1934	Weiss 1943	Henry 1936-37	Hickcox 1940-41	Hyde 1935	Hume 1947	FM1 1947	CAW-5 1949	Wilfong 1948	SF-1-49 1949	FW-1-50 1950
WS 1933	*4+*	0	3+	2+	±	1+	0	0	±	2+	0	0	0
CAW-3 '49	0	*4+*	4+	2+	1+	±	1+	1+	0	±	0	0	0
PR8-ISSC '34	0	2+	*4+*	2+	4+	±	3+	2+	0	0	0	0	0
Weiss '43	0	1+	2+	*4+*	2+	1+	2+	1+	0	0	0	0	0
Henry '36-7	±	2+	3+	3+	*4+*	2+	4+	3+	1+	2+	+	0	±
Hickcox '40-1	2+	1+	4+	4+	3+	*4+*	4+	3+	2+	3+	2+	1+	±
Hyde '35	0	±	1+	2+	4+	2+	*4+*	3+	1+	1+	1+	0	0
Hume '47	0	±	1+	3+	3+	3+	4+	*4+*	1+	2+	3+	0	0
FM1 '47	0	0	±	2+	3+	3+	4+	3+	*4+*	4+	4+	±	±
CAW-5 '49	ND	0	1+	ND	ND	ND	ND	ND	4+	*4+*	4+	2+	1+
Wilfong '48	0	±	1+	2+	2+	2+	2+	2+	3+	4+	*4+*	2+	2+
SF-1-49 '49	0	0	±	0	1+	1+	1+	1+	4+	4+	3+	*4+*	3+
FW-1-50 '50	0	0	±	0	2+	2+	2+	0	3+	4+	4+	4+	*4+*

ND = Not done.

gens were tested with the antisera obtained from the other chicken of the pair which received a given virus. Thus, it would appear that the qualitative antigenic response of different birds to a particular virus was relatively consistent. Similar results were obtained with materials from certain of the strains examined; in most instances such reduplicative detail has been omitted from the tables. Thus, the CAW-1, CAW-2, CAW-3, CAW-4A and Canadian Arctic 1A viruses were indistinguishable from each other; hence, only data on CAW-3 are presented in detail. CAW-5 and Canadian Arctic 12A agents were essentially identical. Strain 4ML-1-48/49 could not be differentiated from SF-1-49 and the AH-1-50 and Berkeley-1-50 viruses did not differ from FW-1-50. The present hemagglutination-inhibition tests with chicken antisera revealed wide antigenic differences among the strains of influenza A examined. While some were essentially identical, such as those mentioned above as well as the Hyde and Hume strains listed in Table IV, others, *i.e.*, WS and FW-1-50, had no demonstrable relationship to each other. Although differences between the more distantly related strains were easily recognized, the precise segregation of the majority of the A viruses into sharply demarcated subgroups was rendered impossible by the existence of a graded series of intermediate forms.

Influenza B. Table V gives an interpretive summary of the findings with strains of in-

TABLE V. Interpretive Summary of Agglutination-Inhibition Tests Showing Relationships of Influenza B Strains.

Rooster antisera	Antigens: Lee 1940	Gt. L. 40 1950	Gt. L. 38 1950	Gt. L. 34 1950	Hawaii 1944-45	Czech. 1945	MB 1950	IB1 1950	Warner 1948	Meguro 1950	POOH 1950	L1-B-50 1950	Seat-2-49 1949
Lee 1940	4+	3+	3+	3+	1+	±	±	0	±	1+	1+	±	1+
Gt. Lakes 40 '50	4+	4+	3+	3+	1+	1+	1+	±	±	1+	1+	1+	2+
Gt. Lakes 38 '50	4+	4+	4+	4+	1+	2+	±	±	1+	1+	1+	1+	2+
Hawaii '44-45	2+	1+	±	±	4+	3+	2+	1+	1+	3+	2+	3+	2+
Czech. '45	3+	1+	1+	1+	4+	4+	3+	1+	2+	2+	1+	3+	3+
MB '50	1+	1+	1+	±	3+	2+	4+	1+	±	2+	1+	3+	2+
IB1 '50	2+	2+	2+	2+	4+	4+	4+	4+	3+	4+	4+	4+	4+
Warner '48	0	0	0	0	4+	3+	3+	3+	4+	4+	4+	3+	3+
Meguro '50	0	0	0	0	2+	2+	2+	3+	3+	4+	4+	3+	3+
POOH '50	0	0	0	0	4+	4+	3+	3+	4+	4+	4+	3+	3+
L1-B-50 '50	2+	0	0	0	4+	3+	3+	2+	2+	4+	3+	4+	3+

fluenza B. It is apparent that these agents fell into 3 general groups with Lee, Hawaii and Warner viruses serving as the prototypes. It is noteworthy that the B viruses studied did not display the numerous intermediate variations encountered among the A viruses examined by this method. This difference may be due more to chance selection of strains than to a real difference between the 2 groups; perhaps other intermediate forms will be found among B strains.

Discussion. Present studies on the antigenic composition of influenza viruses isolated prior to 1945 are, for the most part, limited to strains which have undergone numerous passages in animals and eggs. Since such treatment may induce significant alteration in antigenic pattern(11,12), our knowledge of the constitution of these viruses as they originally occurred in the human population and of their native relationships to each other, must remain largely theoretical.

The question of whether influenza viruses exist in nature in constant or changing antigenic form has been speculated upon by a number of workers(3,9,13,14). Some evidence for the latter possibility was presented in an earlier report(9) in which it was shown that certain strains of influenza A, isolated in the United States and Europe since 1947, showed progressive change in antigenic pattern in successive years. These tests were repeated in the present studies with essentially identical results giving credence to the idea of continuing change in influenza A. A possible explanation for the origin of such variants was suggested by Taylor(14) who conceived that natural passage of influenza virus in the human subject, where antibodies are encountered as a result of previous infections, may give rise to strain deviation.

It is of importance to note, however, that irrespective of whether a single line or several lines of influenza virus exist in nature in constant or changing form, strains of virus close in antigenic composition to earlier prototypes have recurred after widely separated time intervals. Thus, the CAW-3 virus, which was similar to PR8 (Table IV), occurred 15 years following the isolation of this latter agent. Likewise, the Hyde and Hume viruses which were essentially identical, were isolated 12 years apart. The similarity of Hyde and Hume strains to FM1 in hemagglutinin tests with FM1 chicken antiserum was in contrast to the findings of Ward and Eddy(8) who detected no relationship by the mouse neutralization test. Likewise, the failure of PR8 antiserum to react significantly with WS virus, as shown in this and other studies employing hemagglutination(15,16), was not demon-

11. Hirst, G. K., *J. Exp. Med.*, 1947, v86, 357.

12. Francis, T., Jr., PROC. SOC. EXP. BIOL. AND MED., 1947, v65, 143.

13. Magill, T. P., and Sugg, J. Y., PROC. SOC. EXP. BIOL. AND MED., 1943, v53, 104.

14. Taylor, R. M., *Am. J. Pub. Health*, 1949, v39, 171.

strated by the mouse test(8); with the latter technique, WS virus was strongly neutralized by PR8 antiserum. Such observations as these point up the different results obtained in certain instances by the two methods and the hazards of relying on one method alone for evaluating the relationships among strains of influenza virus.

The data (Table IV) concerning the CAW and Canadian Arctic viruses from the outbreak among Eskimos on remote Victoria Island, N.W.T., are of special interest. In the first place, 2 widely different antigenic forms were apparently involved in the outbreak: CAW-3 virus resembled PR8 and CAW-5 was similar to FM1. While strains resembling PR8 have not been found for a number of years, viruses of the same general form as FM1 were isolated from patients throughout the populated cities in the Dominion during the winter of 1949(7). In the second place, these viruses, which were antigenically similar to recognized laboratory strains, were apparently responsible for this outbreak in which the morbidity and case fatality (100% and 20% respectively) approximated that of the last great pandemic. Either virus was presumably of sufficient virulence to cause death, directly or indirectly, since strains CAW-4A (resembling PR8) and CAW-5 (resembling FM1) were recovered from autopsy material. The excessive morbidity in this outbreak may have been due to lack of recent experience with influenza virus as indicated by the low antibody titers in the Eskimos of the region prior to the epidemic(7). Many of the deaths were probably due to secondary bacterial invaders since the sulfa drugs and penicillin were highly effective in treatment(6). It is of considerable interest that these viruses which produced a limited outbreak of disease of high morbidity and mortality among Eskimos were not of unusual virulence for the 50-odd white inhabitants of the region. The latter, who were probably immunized through earlier experience with influenza virus, did not suffer serious infection at the time; indeed, only 1 required medication.

The diversity in antigenic constitution of influenza viruses necessitates the inclusion of a plurality of strains in the prophylactic vaccine to provide a complete coverage against agents from the earlier as well as more recent outbreaks of the disease. Spatial and practical considerations limit the number of strains which can be incorporated into the vaccine so that it is desirable to select a group of strains in which most of the principal antigens of influenza viruses are represented and which are satisfactory in other respects such as growing abundantly in eggs, eliciting a good immunologic response in man and being antigenically stable on inactivation and storage.

Present formula influenza vaccine for military and civilian use is made of PR8 (22 2/9%), FM1 (22 2/9%), FW-1-50 (also called Cuppett or Z virus—22 2/9%) and Lee (33 1/3%) viruses(17). The data in Table IV revealed that while most strains of influenza A appeared to be covered by antisera against the viruses in the vaccine, a definite antigenic deficiency was suggested in that none of the antisera reacted significantly with WS. In view of the broad antigenic pattern of Hickcox virus, in the current tests, further studies on this A strain appear warranted. The present vaccine also appears to be deficient in its coverage of B strains. The present work, as well as that of others(4,5, 18,19) showed that the Lee strain is appreciably different from a number of other B viruses. The latter shortcoming might be eliminated by incorporation of 1 of the more recently recovered strains of B virus such as IBI.

The 1233 strain of Taylor(14) together with the same or similar agent recently iso-

15. Hudson, N. P., Sigel, M. M., and Markham, F. S., *J. Exp. Med.*, 1943, v77, 467.

16. Hirst, G. K., *J. Exp. Med.*, 1943, v78, 407.

17. Laboratory of Biologics Control, National Institutes of Health, Memorandum to Manufacturers of Influenza Virus Vaccines, dated October 11, 1950.

18. Lazarus, A. S., PROC. SOC. EXP. BIOL. AND MED., 1949, v71, 317.

19. National Office of Vital Statistics, Public Health Service, Communicable Disease Summary for Week Ended May 6, 1950, Laboratory Supplement of May 11, 1950.

lated by Francis, Quilligan and Minuse(20) poses another problem as regards the current vaccine. Whether these will eventually be included as immunizing agents remains to be determined, but at the moment the question is essentially a theoretical one since the new strains are difficult to grow by the methods commonly employed for commercial production of influenza virus.

While the selection of strains of influenza virus for use in a vaccine cannot be based on *in vitro* studies alone, the agglutination-inhibition test might be expected to assist materially in the choice of new strains for detailed examinations. The marked strain specificity of chicken antisera when employed in the influenza agglutination-inhibition test appears to provide a simple and effective means for the rapid preliminary antigenic survey of a large number of strains.

20. Francis, T., Jr., Quilligan, J. J., Jr., and Minuse, E., *Science,* 1950, v112, 495.

Summary. Influenza A viruses, as revealed by agglutination-inhibition tests with rooster antisera, comprised a graded series of antigenic forms in which precise subgrouping was not possible by the present method. The B strains examined fell into 3 subgroups. The reappearance from time to time of strains closely related to an earlier prototype, necessitates the selection of a group of strains for a polyvalent vaccine which possess broad immunogenic activity. Certain possible deficiencies in the present formula vaccine were mentioned. The recent outbreak of influenza among Eskimos in remote Victoria Island, Canada, in which there was mortality and morbidity approaching the pandemic form of disease, was apparently caused by 2 different viruses which resembled the well-studied PR8 and FM1 strains of influenza A.

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Enhancing Effect of Cortisone upon Poliomyelitis Infection (Strain MEF1) in Hamsters and Mice. (18362)

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It was recently shown that ACTH and cortisone were capable of inhibiting the phenomenon of local tissue reactivity. The substances failed to suppress the state of reactivity following the preparatory injection of the bacterial toxin but conferred protection against the effect of the provocative intravenous injection of the toxin(1,2). These findings were confirmed by Thomas and Mogabgab. In extending the studies they found that when the preparatory intradermal injection of the toxin was accompanied by an intramuscular injection of ACTH or cortisone, there appeared a petechial reaction at the site of the toxin injection in the absence of the provocative intravenous injection of the toxin. In rabbits receiving no cortisone or ACTH the intradermal injection of toxin failed to produce any hemorrhagic reaction unless followed by a provocative injection of the toxin. The lesions described differed sharply in appearance from the typical reaction of the phenomenon of local tissue reactivity(3). Since the observations suggested that ACTH and cortisone may be capable of enhancing the susceptibility of tissues to the primary effect of bacterial toxins, they were led to determine the effect of the hormones upon bacterial infections. In experiments in this direction Mogabgab and Thomas

1. Soffer, L. J., Shwartzman, G., Schneierson, S. S., and Gabrilove, J. L., *Science,* 1950, v111, 303.

2. Shwartzman, G., Schneierson, S. S., and Soffer, L. J., PROC. SOC. EXP. BIOL. AND MED., 1950, v75, 175.

3. Thomas, L., and Mogabgab, W. J., PROC. SOC. EXP. BIOL. AND MED., 1950, v74, 829.