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I. ANTIBODIES IN TUBERCULOSIS

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Advances in the accuracy of the roentgenologic and bacteriologic diagnosis of tuberculosis have overshadowed serologic methods during the past twenty years. Perusal of current textbooks concerned with tuberculosis indicates the low opinion most authorities now have for serologic tests in this disease. In fact, the non-specific sedimentation rate is the only serologic test of activity which is in general use at the present time. This has not always been the case. In 1898, Aloing and Courmant[1] first noted that the serum of tuberculous patients and animals would agglutinate a suspension of tubercle bacilli in vitro. The test was enthusiastically received but soon fell into disrepute because it was often positive in the absence of active tuberculosis. In the ensuing years numerous methods were devised for precipitin, opsonin and complement-fixation tests[2]. The literature relating to the subject soon became both extensive and confusing. A thorough review by Pfannenstiel[3] in 1924 concluded with a bibliography covering 33 printed pages. None of the methods proved reliable in predicting activity of the disease, and they all were eventually discarded.

Difficulties with the foreging methods centered about the lack of sensitive and yet specific antigens. Cannon and Marshall[4] and Weir[5] attempted to surmount these difficulties by using Collodion particles sensitized with extracts of tubercle bacilli in an agglutination system. The method, however, did not prove satisfactory. After a few exploratory trials in our laboratory it was abandoned because of non-specific reactions. In 1948 Middlebrook and Dubos[6] gave new impetus to the search for a satisfactory diagnostic serologic test for tuberculosis. They found that sheep erythrocytes sensitized with an aqueous extract of tubercle bacilli were agglutinated by sera from tuberculous humans and animals. The present report is concerned with experience in our laboratory with this new serologic method. Data are also included relating to a hemolytic modification of the method recently devised by Middlebrook[7].

METHODS AND MATERIAL

Hemagglutinins. -- The method was that described by Dubos and Middlebrook[6]. The serum was diluted 1:2, inactivated at 56°C. for 30 minutes and its heterophile antibody absorbed with sheep cells. Serial twofold dilutions of the serum (1:4 through 1:128) were added to sheep erythrocytes sensitized with a concentrated old tuberculin* solution as recommended by Scott and Smith[8]. After incubation for 2 hours at 37°C. and overnight at 28°C., agglutination of the sensitized sheep cells was recorded by resuspension. Appropriate controls were included with each experiment.

Hemolysins. -- The method employed was identical with that reported by Middlebrook[7]. It differed little from that described above. Fresh guinea pig serum (complement) was diluted 1:3 and 0.05 ml. added to each tube after addition of the patient's serum and sensitized sheep erythrocytes. After incubation for one hour at 37°C., the degree of hemolysis was recorded. Appropriate controls were always employed.

Coombs Hemolysins.-- A hemolytic modification of the indirect Coombs test was devised[9]. For the most part, human (group O) erythrocytes were employed in place of sheep cells so as to avoid non-

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*Old tuberculin, 4x concentrated, lot no. 2725-12, obtained through the courtesy of Dr. H. D. Piersma, Lederle Laboratories, Pearl River, N. Y.

specific reactions due to heterophile antibodies. After sensitization with tuberculin, a 2 per cent suspension of these cells was placed in a series of Kahn tubes. Serially diluted, inactivated immune serum was added to each tube, and the mixtures were incubated one hour at 37°C. The cells were washed three times and resuspended in buffered saline solution (pH 7.0). Coombs anti-human globulin rabbit serum was added, and incubation was repeated for one hour at 37°C. Fresh guinea pig serum (complement) was then added, and a final incubation for one hour at 37°C. completed the test. Hemolysis in these tubes as well as in control tubes was immediately recorded.

"Blocking" Antibodies. -- Two methods were used: (1) A small amount of immune serum of high antibody titer was added to each of a series of tubes containing serial twofold dilutions of the suspected blocking serum. Sensitized sheep cells were then added, and hemagglutination after incubation was compared with that in a control tube containing no "blocking" serum. The hemolytic test with fresh guinea pig complement was also employed. (2) High titer immune sera were diluted twofold serially in three diluents -- physiologic saline solution, normal human serum and the "blocking" serum. The hemagglutinin and hemolysin titers of the immune serum were then determined in these diluents in the usual fashion.

Material. -- The tuberculous patients included in this study were, for the most part, hospitalized on the Tuberculosis Service of the Minneapolis Veterans Hospital; permission to carry out the investigation was generously given by Dr. William B. Tucker. They had predominantly pulmonary tuberculosis; the diagnosis in each instance was proved bacteriologically by culture of sputum and/or gastric washings as well as roentgenologically. Sera from several patients having active pulmonary tuberculosis were provided by Dr. Burton Waisbren at the Minneapolis General Hospital. A total of 151 patients having active pulmonary tuberculosis are included in this report. Most of the patients had extensive pulmonary tuberculosis, and a few had evidence of extra-pulmonary dissemination.

Sixty-one subjects having a positive Mantoux test and negative chest x-ray were also studied. A dose of 0.1 ml. 1:1000 dilution old tuberculin was used for the intradermal test for hypersensitivity. The subjects were physicians, medical technicians and patients having non-pulmonary disease. No individual was included who had received intradermal tuberculin or tuberculoprotein within three months before the blood sample was obtained[14]. All the subjects had a recent chest x-ray film which disclosed no evidence of tuberculosis.

A total of 67 healthy subjects having a negative Mantoux test and chest roentgenogram are included. They were selected in the same manner as the foregoing group. Thirty-four of the group were entering freshmen at the University of Minnesota; their serum was collected for this study through the cooperation of the staff of the Health Service.

RESULTS

Technical Observations. -- Proper cleansing of glassware proved important. Dichromate solution was unsatisfactory; concentrated sulfuric acid plus 5 per cent nitric acid was used routinely. Few difficulties arose regarding sera; the most important were related to absorption of heterophile antibodies, particularly when present in high titer, as in infectious mononucleosis. Dilution and heat-inactivation of the serum before absorption with sheep cells were necessary. Sheep cells stored for more than three weeks were unsatisfactory. The simpler absorption method of Dubos and Middlebrook[6] proved just as effective as the method of Scott and Smith[8]. Complete removal of heterophile antibody was not always possible with two absorptions; this was particularly evident when the hemolytic test was employed. These difficulties were avoided through the use of human (group O, Rh negative) cells in place of sheep cells. Human erythrocytes, however, were less easily sensitized with old tuberculin. Adequate sensitization of sheep cells was accomplished readily with a 1:12 dilution of

4x concentrated old tuberculin (Lederle), but a 1:8 dilution was required to sensitize human cells.

Both human and sheep erythrocytes were adequately preserved in any of three anticoagulant solutions: modified Alsever's solution[6], 3.8 per cent sodium citrate or acid-citrate-dextrose solution (Baxter). The first of these was used routinely. The age of the cells was most important. Sensitization was more readily accomplished after aging for three days in a refrigerator. Hemolysis became a problem only after more than three weeks storage. Sheep blood was procured under unsterile conditions in a sterile flask at a packing plant; streptomycin or dihydrostreptomycin (1000 units per ml.) was added to prevent bacterial growth.

Sensitization of sheep cells with old tuberculin required careful attention to certain details. Old tuberculin, 4x concentrated (Lederle), gave best results if diluted 1:12 for sensitization of sheep cells and 1:6 or 1:8 with human erythrocytes. Less dilute solutions of this tuberculin were hemolytic; weaker solutions yielded inadequate sensitization. Another less concentrated tuberculin prepared from an avirulent strain of human variety tubercle bacilli* also proved satisfactory, when used in a dilution of 1:8. For best results it was necessary to use 48 to 50 volumes of diluted tuberculin to sensitize one volume of packed, washed sheep cells. Incubation of sheep cells with the tuberculin for 2 hours at 37°C. gave maximum sensitization of the cells. An attempt was made to increase the sensitivity of tuberculin-sensitized sheep cells by exposure to trypsin[21]. Concentrations greater than 2.5 per cent proved hemolytic, and lesser amounts of trypsin did not increase the sensitivity. We were not able to sensitize sheep erythrocytes either with purified protein derivative (PPD)** or with a polysaccharide extract (S_4)*** of tubercle bacilli[10,11,23].

The degree of hemagglutination by immune sera was recorded after incubation for 2 hours at 37°C. and overnight at room temperature. Lesser periods of incubation proved inadequate. The tubes were observed under a strong light, and the cells were resuspended by brief agitation in order to record clumping. The clumps were usually compact and granular but occasionally were loose, flocculent and easily broken up. Determination of hemagglutination by observation of the pattern of the erythrocyte "button" after standing or after slow centrifugation did not yield reliable endpoints. High speed centrifugation often led to false positive hemagglutination.

In many ways the hemolytic modification proved technically more satisfactory. The hemolytic test was more rapid, the endpoints were easier to read and antibody was detected with greater sensitivity. Some technical difficulties were encountered. Scrupulous care was required to remove heterophile antibodies. Fresh guinea pig serum diluted 1:3 proved to be the best source of complement. Lyophilized guinea pig serum was usually satisfactory, but certain lots were low in complement titer. Human serum was not a satisfactory source of complement. In order to obtain maximum hemolytic antibody titers, it was necessary to add complement immediately after addition of the immune serum to the sensitized sheep cells. Hemolysis was recorded immediately after incubation at 37°C. for one hour. The velocity of hemolysis varied considerably with various human sera and was not directly correlated with their hemolytic antibody titer. Two sera having a hemolytic antibody titer of 1:128 were added to tuberculin-sensitized sheep cells plus guinea pig complement, and hemolysis was recorded in a Coleman Junior Spectrophotometer. Serum A produced complete hemolysis in 6.5 minutes, serum B in 70 minutes. The cause for this difference is not known. We have been unable to produce convinc-

*Lot no. 491536, provided by Dr. C. E. Roach, Lilly Research Laboratories, Indianapolis, Ind.

**Obtained from Parke, Davis & Co., Detroit, Mich.

***Generously supplied by Dr. Dennis Watson, Department of Bacteriology.

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ing evidence that delay or absence of hemolytic activity in the serum of tuberculous patients is due to any anti-complementary substance.

Clinical Observations. -- Table I shows the hemagglutinin titer of 151 patients having active pulmonary tuberculosis and 128 subjects whose chest x-ray revealed no evidence of active pulmonary tuberculosis. The median hemagglutinin titer was 1:8 among the patients and less than 1:4 among the controls. A considerable overlap was noted. Thus

Table I.

Anti-tuberculin Hemagglutins and Hemolysins in Patients Having Active Pulmonary Tuberculosis

ANTIBODY TITER	ACTIVE TBC		CONTROLS			
			+ Mantoux		- Mantoux	
	No.	%	No.	%	No.	%
Hemagglutinins						
>128	4	2.6	0	0	0	0
128	1	0.6	0	0	0	0
64	9	5.9	0	0	1	1.5
32	21	13.9	3	4.9	0	0
16	28	18.5	4	6.5	2	3.0
8	22	14.6*	6	9.8	4	6.0
4	31	20.5	9	14.7	20	29.8
< 4	35	23.2	39	63.9*	40	59.7*
Total =	151		61		67	
Hemolysins						
>128	18	15.6	1	2.0	1	1.6
128	8	7.0	1	2.0	0	0
64	16	13.9	1	2.0	2	3.2
32	20	17.4*	7	14.0	3	4.8
16	17	14.7	5	10.0	7	11.3
8	15	13.0	7	14.0	7	11.3
4	12	10.4	3	6.0	8	12.9
< 4	9	7.8	25	50.0*	34	54.8*
Total =	115		50		62	

*Median

43.7 per cent of the patients had a titer less than 1:8 while 15.6 per cent of the controls had a titer of 1:8 or greater. Very high hemagglutinin titers (1:128 or more) were seldom observed (3.3 per cent of patients) and only in the presence of active tuberculosis. There was no significant difference observed in the hemagglutinin titers of the Mantoux positive as compared to the Mantoux negative control group.

The hemolysin antibody titers of a somewhat smaller number of the same patients and control subjects are also given in Table I. The median hemolysin titer of 115 patients having active pulmonary tuberculosis was 1:32 and in 112 control subjects was less than 1:4. In this instance, also, there was considerable overlap. It is significant, however, that a titer of 1:64 or greater

was observed in 45.2 per cent of the patients and in only 5.3 per cent of the controls. The difference was much less at titers below this. There was no difference in the median titer of the Mantoux positive and Mantoux negative control groups. Simultaneous determination of hemagglutinin and hemolysin antibody titers generally showed the latter to be the greater. Rarely was the hemagglutinin titer the greater and then only transiently. Simultaneous determination of hemagglutinin and hemolysin titers in the serum of 103 patients having active pulmonary tuberculosis and in 105 healthy control subjects gave the following results. In 143 the two titers did not differ by more than one dilution. In 61 the hemolysin titer exceeded the hemagglutinin titer by more than one dilution, and in only 4 did the reverse situation hold true. In no patient did the hemagglutinin titer consistently exceed the hemolysin titer by more than one dilution. The difference in titers was not ascribed to the presence of different antibodies since it is generally accepted that hemolytic methods are more sensitive indicators of antibody than are agglutination methods.

An attempt was made to correlate antibody response with the extent of the pulmonary tuberculosis. A definitive study was not possible, however, since our material was selected from hospitalized patients whose pulmonary involvement was usually extensive. Of 127 patients tested for hemagglutinins, 13 had minimal pulmonary tuberculosis, 53 moderately advanced and 61 far advanced. The median titer of hemagglutinins was 1:8 in groups 1 and 2 and 1:4 in the patients having far advanced pulmonary tuberculosis. In a similar group of 103 patients the hemolysin titer was determined; there was no difference among the three groups. In all three the median titer was 1:32.

The frequent occurrence of patients having little or no hemagglutinin or hemolysin in their serum despite active tuberculosis prompted a search for "blocking" antibodies in such sera. Little evidence was disclosed that such blocking antibodies occur. The serum of only one patient proved capable of partially inhibiting the hemolytic activity of a potent human immune serum. Hemagglutinins were not "blocked" by any of several sera tested. The addition of 15 per cent bovine albumin or normal human serum to such sera did not bring forth any increase in hemagglutinin or hemolysin activity. Coombs anti-human globulin rabbit serum did not provoke a significant rise in hemagglutinins. Hemolysins were disclosed in high titer, through the use of complement plus Coombs serum, in the serum of all patients having active tuberculosis whose hemolysin and hemagglutinin titer was low as determined by the Middlebrook techniques. (Table II)

Experimental Observations. -- In order to study more precisely the humoral anti-tuberculin antibodies during the course of immunization, rabbits were injected with large doses of old tuberculin* intravenously at weekly intervals for two to twelve weeks. The majority of the animals had a rise in antibody titer reaching a maximum of 1:32 or greater for hemagglutinins and 1:512 or greater for hemolysins within two to three weeks after the first dose of tuberculin. A few rabbits did not produce such antibodies in significant titer after two doses of tuberculin. Rabbits given tuberculin for several weeks showed an initial rise in antibody titer followed by a sharp decline; the latter occurred during the seventh to eighth week of immunization. The antibody titer dropped to less than 1:2. The titer of Coombs hemolysins followed an identical pattern. Despite repeated challenge with tuberculin, such animals never responded by producing antibodies once the decline had occurred. They were, however, fully capable of producing typhoid "O" and "H" agglutinins when given triple typhoid vaccine intravenously. "Blocking" antibodies were not demonstrable during any phase, nor were anticomplementary substances

*Five ml. 1:5 dilution of 2^x concentrated human O.T., generously provided by Dr. C. E. Roach, Lilly Research Laboratories, Indianapolis, Ind.

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Table II

Anti-tuberculin Coombs Hemolysins in Patients Having Active Pulmonary Tuberculosis but no Hemagglutinins or Hemolysins (Middlebrook).

SUBJECTS	SERUM DILUTIONS (Reciprocal) 2	4	8	16	32	64	128	C**
	(Coombs Anti-human Globulin Rabbit Serum)							
Normal	0*	0	0	0	0	0	0	0
Patients								
	4+	4+	4+	4+	4+	3+	2+	0
.	4+	4+	4+	4+	4+	3+	2+	0
	4+	4+	4+	4+	4+	3+	2+	0
.	4+	4+	4+	4+	3+	2+	2+	0
	4+	4+	4+	3+	1+	1+	0	0
.	1+	1+	1+	1+	1+	1+	1+	0
	(Control -- Normal Rabbit Serum)							
Normal	0	0	0	0	0	0	0	0
Patients								
	1+	1+	1+	1+	0	0	0	0
.	2+	2+	0	0	0	0	0	0
	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0

* 0 to 4+ = degree of hemolysis.
** Unsensitized sheep cell control.

present. Furthermore, there was no decline in hemolytic complement titer coincident with the fall in antibody titer.

The mechanism by which the antibody decline was brought about has not been elucidated. Further experiments in rabbits have left no doubt but that a significant reduction in plasma proteins is followed by a fall in antibody titer[12]. After repeated plasmapheresis in the immunized rabbit a gradual decline in plasma proteins set in, and coincident with this there was a moderate decline in hemagglutinin and hemolysin titer. An abrupt but temporary decline in plasma protein and hemolysin was observed after vigorous plasmapheresis. Since there was no demonstrable hypoproteinemia in the hyperimmunized rabbits, the late reduction in antibody in those animals was not ascribed to the same mechanism.

Experiments are in progress to determine the effect of destruction of the antibody production capacity of the rabbit upon the late phase of hyperimmunization with tuberculin. Rabbits were injected with tuberculin intravenously once weekly until maximum antibody response occurred. They were then given 800 roentgens of total body X-irradiation. Within 24 hours thereafter another large dose of tuberculin was given, and the course of the antibody titer was closely followed. Preliminary observations have not indicated an acceleration in antibody decline. Because antibody

formation after tuberculin immunization is variable in the rabbit, the use of live, avirulent tubercle bacilli is now under investigation.

It is true, however, that that which holds for the rabbit may not necessarily be so in the human being. The antibodies which are formed by the rabbit immunized with tuberculin differ from those found in a man having pulmonary tuberculosis (Table III). Hemolysins in such rabbit sera are inhibited by the addition of a small amount of tuberculoprotein

Table III

Inhibition of Anti-tuberculin Hemolysins by Tubercle Bacillus Protein and Carbohydrate

	Antigen Dilutions (Reciprocal)															
	1	2	4	8	16	32	64	128	256	512	1024	2048	4096	8192	C_1*	C_2**
Rabbit Immune Serum																
Protein***	0	0	0	0	2+	3+	4+	4+	4+	4+	4+	4+	4+	4+	4+	0
Carbohydrate****	1+	3+	3+	3+	3+	3+	3+	4+	3+	3+	4+	4+	4+	4+	4+	0
Human Serum																
Protein	3+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	0
Carbohydrate	0	0	0	0	0	0	0	0	0	0	0	0	0	2+	4+	0

* Immune serum + sensitized cells + saline + complement

** Immune serum + unsensitized cells + saline + complement

*** Purified protein derivative, 15.6 gamma/0.4 ml. (Parke, Davis & Co.)

**** Polysaccharide (S_4 fraction), 200 gamma/0.4 ml. (Dr. Dennis Watson)

0 to 4+ = amount of hemolysis.

Method of Middlebrook and Dubos[6].

but only by a large quantity of M. tuberculosis polysaccharide. On the other hand, hemolysins of the tuberculous human being are inactivated by minute concentrations of polysaccharide but are scarcely inhibited at all by the protein fraction[6,20].

DISCUSSION

The results of our clinical investigations of the hemagglutination test and its hemolytic modification have been compared with similar studies published by others (Table IV). The early optimism of Rothbard et al[13] as to the sensitivity and specificity of the hemagglutination test for tuberculosis has not been supported by subsequent investigators. Several authors have reported the test is frequently negative in the presence of active pulmonary tuberculosis. In our study of 151 such patients, only 56.1 per cent showed hemagglutinins in a titer of 1:8 or more. The specificity of the test has also proved imperfect. Runyon et al[18] reported that 20.4 per cent of 98 subjects having a negative chest x-ray and Mantoux test showed a positive hemagglutination test for tuberculosis. Their subjects included 80 infants and children of unstated age, some of whom may have possessed antibody passively transferred from their mothers. In our study of 128 nontuberculous subjects having a normal chest roentgenogram, 21.2 per cent of those with a positive tuberculin test and 10.5 per cent of those having a negative tuberculin test had hemagglutinins in a "significant" titer.

It was our hope that the hemolytic test might be a more sensitive and yet no less specific test. The data given in Table I indicate the test is more sensitive, but as shown in Tables I and IV, it is no better in differentiating those patients having active tuberculosis from those who do not have it. Since anti-tuberculin antibodies are frequently present in patients having no other evidence of active tuberculosis, it appears likely that the greatest improvement that might be expected would be the development of a purified M. tuberculosis antigen to be used in place of old tuberculin. As demonstrated in Table III and in the publications of others[6,20], the most effective substance presumably would be a carbohydrate fraction. The use of trypsin in sensitizing the cells might be a further aid[10,21]. The sensitization of sheep erythrocytes with tubercle bacillus polysaccharide has been accomplished by others[11,23], but we have been unable to repeat it.

Even with the more sensitive hemolytic modification, the methods of Middlebrook[6,7] frequently fail to disclose antibody in the serum of patients having active tuberculosis. These negative tests are not due to "blocking" antibodies nor to anticomplementary substances. Anti-tuberculin antibody has been demonstrated in such sera by a hemolytic modification of the indirect Coombs test (Table II). The failure of these antibodies to react in the usual fashion with tuberculin-sensitized erythrocytes remains unexplained. This "incomplete" type of antibody appears to be most commonly present in patients having far advanced pulmonary tuberculosis[9].

A similar but not identical phenomenon has been produced in immunized rabbits. When injected intravenously once weekly with a large dose of old tuberculin, rabbits soon developed a high titer of hemolysins and hemagglutinins. After repeated immunization, however, the antibody titer declined. Hemolysins became slow in activity and finally all antibodies, including Coombs hemolysins, disappeared for many weeks. The animals remained capable of responding to an unrelated antigen. The explanation of the phenomenon is not yet apparent. Several possibilities have been suggested: (1) Injection of excess antigen might lead to in vivo combination of antigen and antibody removing the latter from the circulating blood. It seems unlikely that antigen-antibody complexes remain in the serum since the late phase sera have contained a normal amount of hemolytic complement and have had no anticomplementary activity. (2) Repeated antigenic stimulation might deplete the rabbit of a particular type of globulin necessary for the formation of anti-

Table IV

Anti-tuberculin Antibodies in Human Tuberculous Patients

Author: Year	Active Tuberculosis		+ Mantoux - Chest X-ray		- Mantoux - Chest X-ray	
	No.	1:8 or greater (%)	No.	1:8 or greater (%)	No.	1:8 or greater (%)
Hemagglutinins						
Rothbard et al 1950 (13)	168	92.3	33 "Cured tbc"	0	110 "Normal"	0
Smith & Scott 1950 (14)	24	70.8				
Gernez-Rieux & Taquet 1949 (15)			43	0		
1950 (16)	504	79.8			244 "Normal"	6.9
Sohier et al 1951 (17)	27	74.1	74	16.2	47	14.9
Runyon et al 1951 (18)	89	43.8	53	13.0	98	20.4
McDearman 1951 (19)	124	58.7				
TOTAL	936	82.9	170	9.7	145	17.7
Hall 1951	151	56.1	61	21.2	67	10.5
		1:32 or greater (%)		1:32 or greater (%)		1:32 or greater (%)
Hemolysins						
Hall 1951	115	53.9	50	20.0	62	9.6

tuberculin antibody. Plasmapheresis experiments, however, have indicated that a marked reduction of antibody content on this basis would be only temporary (a few hours). (3) The body fluids and tissues of the animal might become so saturated with antigen that its antibody production mechanism might be suppressed in some fashion. A similar phenomenon has been reported by Felton[22] in mice immunized with pneumococcus polysaccharides. Small doses led to marked antibody production, but large doses of polysaccharides provoked no antibodies. Felton found that the latter animals retained the polysaccharide in their tissues for a long time and called the phenomenon "Immunological Paralysis".

SUMMARY AND CONCLUSIONS

1. Hemagglutinins for tuberculin-sensitized sheep erythrocytes were found in a titer of 1:8 or greater in the serum of 56.1 per cent of 151 patients having active pulmonary tuberculosis. They were also present in 21.2 per cent of 61 Mantoux-positive and 10.5 per cent of 67 Mantoux-negative control subjects.

2. Hemolysins for sensitized sheep cells were found in a titer of 1:32 or greater in almost the same frequencies.

3. Tuberculous patients lacking hemagglutinins and hemolysins possessed Coombs hemolysins in high titer.

4. Immunization of rabbits with several large doses of tuberculin led to a late disappearance of circulating antibody similar to that observed in some patients having active tuberculosis. The "immunologic paralysis" was specific for tuberculin. Its mechanism is not yet known.

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II. MEDICAL SCHOOL NEWS

Coming Event

June 19 Duluth Clinic Lecture; "Fourteen Years' Experience with the Ballistocardiograph," Dr. Isaac Starr, University of Pennsylvania School of Medicine; Medical Science Amphitheater, 8:15 p.m.

Dr. Isaac Starr to give Duluth Clinic Lecture

Dr. Isaac Starr, Hartzell Professor of Research Therapeutics, University of Pennsylvania School of Medicine, will deliver the annual Duluth Clinic Lecture at 8:15 p.m. on Tuesday, June 19, in the Medical Science Amphitheater. Dr. Starr, who is well known throughout the world for his many contributions in the field of cardiovascular disease, will speak on the subject, "Fourteen Years' Experience with the Ballistocardiograph." In addition, Dr. Starr will participate in informal conferences with members of various departments of the Medical School. He will also give a clinic on congestive heart failure before junior medical students and staff at 8:00 a.m., Tuesday, June 19, in Todd Amphitheater. The Duluth Clinic Lecture, made possible by a gift to the University from the Duluth Clinic, will be the last of the special lectureships for this present academic year.

Thank You, Elva Lavers

The editor wishes to express a special word of thanks to Miss Elva Lavers and her staff for the wonderful spirit of cooperation which they have maintained throughout the past year. It has been a real privilege to turn to Miss Lavers with many problems which arise in publishing a bulletin of this sort. Always one can be confident that a superlative job will be done.

Summer Best Wishes

Today's issue brings to a close the series for the "Bulletin of the University of Minnesota Hospitals and Minnesota Medical Foundation" for the academic year 1950-51. As one reviews the titles of the papers presented, we find a series of subjects of great diversity and extreme interest and timeliness. We want to thank the division and department heads and the authors who have contributed to the improving quality of the publication.

We wish to thank also those members of our staff who have contributed to our discussions at the hospital staff meeting. The discussion periods have been a source of information and stimulation to all of us. We invite physician readers of the Bulletin to join with us at our Friday noon luncheon meetings when these are resumed on October 5.

Finally, we wish to extend to all students, faculty, and friends of the Medical School and to all members of the Minnesota Medical Foundation our very best wishes for the coming summer season. We hope that it may be a period during which energy may be recharged, imagination strengthened, and zest for work and recreation rekindled.

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IV.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS
Physicians Welcome

June 11 - 16, 1951

Monday, June 11, 1951

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; M-109, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Public Health Seminar; 113 Medical Sciences.

4:30 - 5:30 Dermatological Seminar; M-436, U. H.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staffs; Powell Hall Amphitheater.

Minneapolis General Hospital

8:30 - 10:00 Pediatric Rounds; Dr. Lowry; 7th Floor Annex.

11:00 - Pediatric Rounds; Franklin Top; 7th Floor Annex.

1:00 - 2:00 Staff Meeting; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Dr. Ulstrom; 5th Floor Annex.

Veterans Administration Hospital

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; Conference Room; Bldg. I.

1:00 - Metabolic Disease Rounds; N. E. Jacobson and G. V. Loomis; Bldg. I.

4:00 - Medical Surgical Conference; Conference Room, Bldg. I.

Tuesday, June 12

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staffs; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

1:00 - 2:00 Physiology Seminar on Cardiac Metabolism; 129 Millard Hall.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:00 - 5:00 Electrocardiographic Conference; EKG Laboratory, 6th Floor, U. H.

8:00 p.m. Journal Club; E-101, U. H.

Ancker Hospital

1:00 - 2:30 X-ray Surgery Conference; Auditorium.

Veterans Administration Hospital

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery Tumor Conference; Conference Room, Bldg. I.

1:00 - Chest Surgery Conference; T. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

1:30 - Liver Rounds; Samuel Nesbitt.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

3:30 - 4:20 Clinical-Pathological Conference; Conference Room, Bldg. I.

Wednesday, June 13

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Allen Judd and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Medicine Case; O. H. Wangensteen, C. J. Watson and Staffs; Todd Amphitheater, U. H.

Wednesday, June 13 (Cont.)

*4:30 p.m. Medical Film: "The Medical Effects of the Atom Bomb -- Part II," Todd Amphitheater, U. H.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; Robert Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

8:30 - Pediatric Rounds; Dr. Lowry; 7th Floor Annex.

9:00 - Pediatric Allergy Rounds; Dr. Nelson; 4th Floor Annex.

11:00 - 12:00 Pediatric Rounds; Franklin Top; 7th Floor Annex.

12:15 - Staff Meeting; 4th Floor Annex.

1:30 - Pediatric Rounds; Dr. Huenekens and Dr. Ulstrom; 5th Floor Annex.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic-Roentgenology Conference; Edward T. Evans and Bernard O'Loughlin; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, June 14

Medical School and University Hospitals

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.

Thursday, June 14 (Cont.)

Medical School and University Hospitals (Cont.)

*1:00 p.m. Medical Film; "The Medical Effects of the Atom Bomb -- Part II," Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Minneapolis General Hospital

8:30 - Neurology Rounds; Dr. Heilig, 4th Floor Annex.

11:30 - Pathology Conference; Main Classroom.

1:00 - 2:00 EKG and X-ray Conference; Classroom, 4th Floor Annex.

2:00 - Psychiatry Rounds; Dr. Benton; 4th Floor Annex.

2:00 - 4:00 Infectious Disease Rounds; 8th Floor.

4:00 - 5:00 Infectious Disease Conference; Classroom, 8th Floor.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff.

9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery Roentgen Conference; Conference Room, Bldg. I.

2:15 - Chest Rounds; William Stead.

Friday, June 15

Medical School and University Hospitals

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.

Friday, June 15 (Cont.)

Medical School and University Hospitals (Cont.)

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Dermatology Seminar; W-312, U. H.

4:00 - 5:00 Vascular Rounds; Davitt Felder and staff members from the departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

8:30 - Pediatric Rounds; Dr. Lowry; 7th Floor Annex.

10:00 - Pediatric Rounds; Franklin Top; 7th Floor Annex.

1:30 - Pediatric Rounds; Dr. Ulstrom; 5th Floor Annex.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Microscopic-Pathology Conference; E. T. Bell; Conference Room, Bldg.I.

1:30 - Chest Conference; Wm. Tucker and J. A. Myers; Ward 62, Day Room.

3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, June 16

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; Wallace H. Cole and Staff; M-109, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:15 - 10:00 Surgery-Roentgenology Conference; J. Friedman, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Anterior Resection for Cancer of the Rectum; Charles Mayo; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

Saturday, June 16 (Cont.)

Minneapolis General Hospital

11:00 - 12:00 Pediatric Clinic; Dr. Thomas and Dr. Good; Classroom, 7th Floor Annex.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Hematology Rounds; P. Hagen and E. F. Englund.