STUDIES IN EXPERIMENTAL TRANSFUSION.*

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I. NORMAL ISO-AGGLUTININS IN CATS.
II. THE DEVELOPMENT OF ISO-HEMOLYSINS AND ISO-AGGLUTININS AS THE RESULT OF TRANSFUSIONS IN CATS.
III. THE EFFECTS OF AGGLUTINATIVE AND HEMOLYTIC TRANSFUSIONS.
IV. GLYCOSURIA ACCOMPANYING INTRAVASCULAR HEMOLYSIS.

I.

NORMAL ISO-AGGLUTININS IN CATS. — Preliminary to experimental transfusion in cats we made a short study of the normal occurrence of iso-antibodies in their blood.

Ingebrigtsen[1] first described the occurrence of iso-agglutinins in normal cats. He examined forty cats in groups of ten. Only six cats showed agglutinins in the serum which affected the red cells of other cats. These were scattered in such a way that no definite groupings could be determined, such as occur in man and such as have been shown by Ottenberg and Friedman to occur in some of the lower animals. Of these six agglutinative sera, two agglutinated the red blood cells of one other cat, two those of two others, one those of four others, and one those of seven others.

In general we are able to confirm these findings, but on making repeated examinations of the same cats at intervals of a few weeks we find that the agglutinins are not constant. They vary considerably from time to time although those sera which are strongly agglutinative to the cells of a number of other cats usually remain so over considerable periods (see Charts I. and II.).

Auto-agglutination, that is, agglutination of the animals' suspended red cells by its own serum, was found a number of times. Those strongly agglutinative sera which affect the red cells of a large number of other animals are usually those which are auto-agglutinative.

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This fact together with the occurrence of spontaneous variations in agglutinative power from time to time would seem to rob iso-agglutination as it occurs in cats of some of its biological significance in differentiating individuals, or groups of individuals, of the same species.

Our observations were made on forty cats taken in various groups of ten or twelve at irregular intervals extending over about six months. Independent readings of the results by each of us were made in all cases. The technic followed was that of Epstein and Ottenberg.[2]

II.

THE DEVELOPMENT OF ISO-HEMOLYSINS AND ISO-AGGLUTININS AS THE RESULT OF TRANSFUSIONS IN CATS. — Iso-hemolysins do not occur among normal cats. The only exception to this, in a great number of tests, was the hemolysis of the cells of one cat in a series of twelve by the serum of a cat (22), which had been used only as a donor for two transfusions. This hemolytic power was absent in tests repeated ten days later (see Charts V. and VI.).

It was thought likely that by repeated injections of blood or by direct transfusions it would be possible to develop iso-hemolysins in cats, as has been done in other animals. The present experiments were undertaken for the purpose of studying the method of their development. It seemed probable from experimental dog transfusions[3] and also from the fact that iso-hemolysins in man only develop on the basis of already formed iso-agglutinins, that hemolysins would only develop in animals which already possessed agglutinins for the blood of the donors used. This did not prove to be the case. Hemolysins developed in the serum of some animals that had no agglutinins as well as in animals in which agglutinins were present. They failed to develop in the serum of some animals transfused from donors whose cells were agglutinable.

Twenty-nine transfusions were done on seven cats. The intention was to do repeated transfusions on the same cat, so far as possible from the same donor or from donors showing

similar agglutination reactions. Two of the recipients, however, received only one transfusion each, one received two transfusions, two three transfusions, one eight transfusions, and one ten transfusions. The transfusions were direct from artery to vein or vein to vein by means of the Elsberg or Bernheim canula (usually the latter). They were done at intervals of three to five weeks. Ether anesthesia was used. The donors and recipients were weighed accurately before and after transfusion. The amounts transfused on each occasion varied between thirty and eighty grams, the average being about fifty grams. The symptomatology will be discussed later.

The development of new agglutinins. — A large number of cats which were used as blood donors, or were not used in transfusions at all, and five cats which were repeatedly used as recipients in blood transfusions were retested at intervals during a year with regard to agglutination towards one another's blood. It was found that those cats which were not transfused and whose serum at the beginning agglutinated the red cells of very few or no other cats showed practically no change in agglutinative power. Occasionally, however, they developed agglutinins for the blood of several cats which they had not previously agglutinated. The cats which received repeated transfusions, and which had no or very few agglutinins at the start, developed marked agglutinins for a large number of other cats, usually after two transfusions. When no further transfusions were done these agglutinins generally disappeared after a few months. They could be made to re-appear by again performing transfusions. One cat, which was strongly agglutinative to the majority of other cats before any transfusions were done, remained so after two transfusions had been done, but two months later was found to have lost all agglutinins. The development of iso-agglutinins was apparently independent of whether the cells of the donor cat in each case were agglutinable by the recipient's serum or not.

The development of iso-hemolysins after transfusions. — Of the seven cats which were recipients in transfusions, three developed iso-hemolysins. The serum of the first of these three, Cat 16, was found to be hemolytic for the cells of four out of nine cats one month after the first transfusion. This transfusion was from a donor whose cells before transfusion were slightly agglutinated by the serum of the recipient cat. Cat 16 subsequently escaped and could not be observed further.

The second cat (Cat 19) was transfused from a donor whose cells its serum did not agglutinate, but two months after the second transfusion the serum was found to be strongly hemolytic for the cells of the donor as well as for those of three other cats. The four cats whose cells were susceptible to the serum of Cat 19 were the identical ones whose cells were susceptible to the serum of the preceding Cat 16. Two of the three whose cells were laked were the respective donors of the two cats. It thus appears that there is a definite group or that there are at least two definite groups among cats with regard to artificially developed iso-hemolysins (see Tables III. and IV.). Cat 19 was then transfused ten times over a period of about a year from these cats and from other cats for whose cells its serum was subsequently found to be hemolytic. The serum remained hemolytic toward the same cats throughout the entire time.

The third cat (Cat 18) was transfused five times, at intervals of about a month, from cats whose cells its serum agglutinated. Nevertheless, no hemolysins developed until two months after the sixth transfusion. This sixth transfusion was from a new donor which had never been used before and whose cells were strongly agglutinated by the serum of Cat 18.

The four other cats which were transfused a smaller number of times, one, two and three times, respectively, failed to develop hemolysins. None of the other cats kept under observation ever developed iso-hemolysins although they were repeatedly tested for them.

It thus appears that iso-hemolysins may develop after one

or more transfusions. Probably their appearance or failure to appear depends on the use as donor of a cat belonging to the particular biological group whose cells are susceptible to the hemolysin which the particular cat used as recipient is capable of developing. This would appear to be the case from the failure of Cat 18 to develop hemolysins after five transfusions from four different donors and its development of hemolysins after one transfusion from a fifth donor. Thus it appears probable that the four cats which failed to acquire hemolysins after a smaller number (not more than three) of transfusions would have done so if further trials with new donors had been made.

The titer of the hemolysins developed was not determined, but in mixtures containing equal amounts of fresh serum and of five per cent cell suspension laking occurred in most cases in ten or fifteen minutes at room temperature.

III.

THE EFFECTS OF AGGLUTINATIVE AND HEMOLYTIC TRANSFUSIONS. — In the cases where agglutination alone was found in preliminary tests in vitro, there were no symptoms whatever after transfusion and no subsequent changes different from those where no agglutination occurred.

The effect of the transfusion of blood whose cells were susceptible to laking by the serum of the recipient cat was regularly an attack of hemoglobinuria, presumably caused by the destruction of the received blood. This occurred in every one of the ten hemolytic transfusions with the exception of one in which only jaundice developed. Hemoglobinuria was never observed in any of the seventeen non-hemolytic transfusions. This complete correspondence between test-tube hemolysis and intravascular hemolysis confirms the opinion that similar tests for human transfusions can be relied on completely to prevent hemolytic accidents.

The hemoglobinuria was accompanied, as we found in numerous instances, by hemoglobinemia, which occurred within a half hour of the time of transfusion, possibly sooner. Frequently cats did not pass any urine at all for the first

twenty-four hours. The urine contained not only dissolved hemoglobin but sometimes shadows of red cells and numerous blood casts as well. There was regularly a pronounced leucocytosis accompanying the hemoglobinuria (as was also observed in experimental dog transfusions [3]). On the second and third days the hemoglobin in the urine was replaced by bile and usually the cats were jaundiced.

Blood smears made immediately after transfusion showed on a number of occasions that the hemoglobinemia was accompanied by phagocytosis of red cells in the circulation. This phagocytosis was accomplished to some extent by polymorphonuclear cells, but to a greater extent by large mononuclear cells with more deeply staining cytoplasm than is seen in the large mononuclear cells of human blood.

One entirely unexpected accompaniment of the hemoglobinuria was glycosuria. This led to some further experiments and will be discussed under a separate heading below.

The hemoglobinuria not only led to the destruction of the received blood cells, but had a further effect in producing a pronounced anemia. A gradual destruction of the animal's own blood cells occurred in the days and weeks following the hemoglobinuria. On the other hand, in transfusions where no hemoglobinuria occurred the result was a plethora. Thus in Cat 4 after the second transfusion the red cells were raised from 7,800,000 (an approximately normal count for a cat) to 12,200,000.

In Cat 18, which only developed hemolysins after the sixth transfusion, the red cells were raised as the result of the first six transfusions from 6,700,000 to 12,800,000. Two months after the sixth transfusion (cat in the meantime having developed iso-hemolysins and presumably having destroyed a good deal of the blood which it had received) looked very anemic and smears showed the presence of many nucleated red cells. The seventh transfusion now resulted in jaundice, and the following day the red cells were found to be 7,000,000. A month later they had sunk to 3,600,000 and an eighth transfusion was done which resulted in a marked hemoglobinuria. At the end of the transfusion the red cells

had risen to 8,000,000, but, on the following day, they had sunk again to 6,700,000 and eleven days later to 4,650,000, so that presumably most of the blood cells received were destroyed.

Cat 19 showed a similar course, the red cells being raised from 8,500,000 to 10,800,000 two weeks after the first (non-hemolytic) transfusion. Two months later (the cat in the meanwhile, as the result of the second transfusion, having developed iso-hemolysins) the red cells had sunk to 7,100,000. Three days after the third transfusion, at which time the cat's serum was strongly hemolytic to the donor's cells, and which was accompanied by a pronounced hemoglobinuria, the red cells had fallen to 4,600,000. After a two months' rest the cat's red cells rose again to 7,740,000, and then as a result of three transfusions accompanied by hemoglobinuria the red cells fell again in three months to 3,300,000. Normoblasts and megaloblasts were present, and there was irregularity in the size and staining of the red blood cells, some of them showing pale centers, others showing polychromatophylia.

IV.

GLYCOSURIA ACCOMPANYING INTRAVASCULAR HEMOLYSIS. — In the first hemolytic transfusion on Cat 19 routine examination of the hemoglobinuric urine revealed the fact that sugar was present. After removal of the albumin by boiling with acetic acid, the sugar was determined, using Rudisch's solution, and was found to be one per cent. In six of the seven similar transfusions with the same cat the same phenomenon was observed. In one of them sugar was absent in spite of the fact that hemoglobin was present. In one of the hemolytic transfusions on Cat 18 sugar was also present. These cats showed no glycosuria at other times.

The amount of sugar was always small, usually a fraction of one per cent. It was demonstrated by the reduction of Fehling's and Nylander's solutions and on several occasions by the fermentation test and by the polariscope. In six

transfusions in which hemoglobinuria did not occur no sugar could be demonstrated.

It was thought advisable to ascertain which particular factor in the transfusion caused the glycosuria. Accordingly, immune sera were prepared by the injection of a number of rabbits with either whole defibrinated cat's blood, washed red blood cells, or cat's serum. Of these three sera, those prepared by the injection of whole blood and of washed red cells were found to be powerfully hemolytic to cat's red cells. The one prepared by the injections of serum was feebly hemolytic.

The intravenous injections of the anti-whole blood serum into cats, in amounts varying from .4 cubic centimeter up to two cubic centimeters, caused a pronounced hemoglobinuria in four of seven cats and a glycosuria likewise in four of the seven. One cat had glycosuria without hemoglobinuria and one hemoglobinuria without glycosuria. In the first of these experiments the presence of sugar was determined not only by reduction and fermentation tests and by the polariscope, but also by the formation of crystals of phenyglucosazone. The serum which had been prepared by the injection of washed red cells (known as anti-red blood cell serum) injected intravenously into cats in amounts of from one to two cubic centimeters also produced a marked hemoglobinuria, but no glycosuria on either of the two occasions in which it was used. Both of these hemolytic sera (the anti-whole blood and the anti-red cell) were highly toxic, and amounts over 2.5 cubic centimeters usually killed the cat within a few minutes. Blood removed at once from the portal or pulmonary veins of these cats showed macroscopic red-cell agglutination, and at room temperature hemolysis occurred in fifteen to twenty minutes. On the other hand, the anti-serum serum was not toxic in amounts up to three cubic centimeters. One cat which was injected intravenously with anti-red blood cell serum had a marked hemoglobinuria the next day and showed at autopsy, one month later, thrombosis of the pulmonary arteries supplying both lower lobes and large pulmonary infarcts in these lobes. The serum prepared by injecting rabbits

with cat's serum (anti-serum serum) produced no hemoglobinuria in any of four experiments, but after two of the injections did produce a slight glycosuria (.4 per cent of sugar in one experiment and a trace in the other). The injection of distilled water intravenously into cats (ninety and one hundred and twenty cubic centimeters in two experiments) produced a pronounced hemoglobinuria but without sugar.

With this small number of experiments we are unable to determine the significance of the glycosuria which sometimes accompanies hemoglobinuria. There is no doubt about the substance present being sugar, although in several of the experiments in which there was no sugar there was present in the urine some substance which produced distinct reduction (yellow color) of Fehling's solution without, however, the formation of a precipitate.

The failure of sugar to appear in the distilled water experiments and in the anti-red-cell serum experiments would suggest that its occurrence is more probably connected with antibodies directed against some serum constituent than with hemolysis. The subject requires further study.

CONCLUSIONS.

I. Iso-agglutinins in cats' blood do not form distinct groups. They are relatively feeble antibodies, and vary considerably from time to time in the same cats.

II. Iso-hemolysins are seldom, if ever, found among normal cats, but they often appear in the recipients of blood transfusions. Apparently their occurrence or failure to occur depends on some biological property of the blood of the donor cat. The iso-hemolysins which appear are selective, affecting not only the blood of the donor cat but of certain others also. Therefore it seems that there are biological groups within the species in this case, just as there are among goats (Erlich's fundamental experiments), among human beings (Moss), and, as Von Dungern has shown, among cats.

III. The direct transfusion of iso-agglutinative blood has no immediate harmful effects.

IV. The transfusion of blood whose cells are susceptible to laking by the recipient's serum produces a marked hemoglobinemia and hemoglobinuria, with intravascular phagocytosis of red cells, a reactive leucocytosis, and usually glycosuria.

V. This intravascular destruction of another animal's blood exerts some toxic effect, so that the recipient animal itself develops an anemia with the appearance of bone marrow cell forms in the circulating blood. There is a tendency to spontaneous recovery from this anemia.

VI. Glycosuria sometimes accompanies the intravascular destruction of blood. It does not seem to be dependent on hemolysis alone, but on some other factor, not as yet understood.

TABLE I. (Feb. 1, 1913.)

Normal iso-agglutinins.

Readings after one-half hour at room temperature.

	2	3	4	5	6	7	8	11	12	13	14	15	16	17	18	19
2	—	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
3	—	—	—	—	—	—	—	—	—	+	—	—	?	—	—	—
4	—	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
5	—	—	—	—	—	—	—	—	—	+	—	—	?	—	—	—
6	—	—	—	—	—	—	—	—	—	+	—	—	?	+	—	—
7	—	?	—	—	—	—	—	—	—	+	—	—	+	+	—	—
8	—	?	—	—	—	—	—	—	—	+	—	—	?	+	—	—
11	—	?	—	—	—	—	—	—	—	+	—	—	+	+	—	—
12	—	—	—	—	—	—	—	—	—	+	—	—	+	+	—	+
13	—	—	—	—	—	—	—	—	—	+	—	—	+	+	—	?
14	—	+	—	—	—	?	—	—	—	+	—	—	?	—	—	—
15	—	?	—	—	—	+	—	—	—	+	—	—	—	?	—	—
16	—	+	—	?	—	?	—	—	—	+	—	—	?	+	—	—
17	—	+	—	—	—	—	—	—	—	+	—	—	?	—	—	—
18	—	—	—	—	—	—	—	—	—	+	—	—	?	—	—	—
19	—	?	—	—	—	—	—	—	—	?	—	—	—	?	—	—

In the tables, + indicates agglutination; — indicates absence of agglutination; ? indicates slight or questionable agglutination; H indicates hemolysis.

The vertical columns indicate sera, the horizontal lines red cells of the cats whose numbers are given.

TABLE II. (Feb. 5, 1913.)

Normal iso-agglutinins.

Readings after two hours at room temperature.

	3	4	5	6	7	8	11	12	13	14	15	16	17	18	19
3	—	—	—	—	—	—	—	—	—	—	—	+	—	—	—
4	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
5	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
6	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
7	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
8	—	—	—	—	—	—	—	—	—	—	—	+	+	—	—
11	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
12	—	—	—	+	+	+	—	—	+	—	—	+	+	—	+
13	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
14	—	—	—	—	—	—	—	—	+	—	—	+	+	—	—
15	—	—	—	—	—	—	—	—	+	—	—	+	+	+	+
16	—	—	—	—	—	—	—	—	+	—	—	—	—	—	—
17	—	—	—	—	—	—	—	—	—	—	—	+	+	—	—
18	—	—	—	—	—	—	—	—	+	—	—	—	—	—	—
19	—	—	—	—	—	—	—	—	+	—	—	—	—	—	—

Tables I. and II. show the variability of the iso-agglutinins. Cats which have agglutinins on one occasion for the red blood cells of many other cats, possess agglutinins on another occasion for the red blood cells of about the same number of other cats, but not always the same ones.

TABLE III. (May 16, 1913.)

Agglutinins after transfusions.

Readings after two and one-half hours at room temperature.

	4	6	7	8	11	15	16	17	19
4	—	—	—	—	—	—	+	+	+
6	—	—	—	—	—	—	—	+	+
7	—	—	—	—	—	—	+	+	+
8	—	—	—	—	—	—	+	+	—
11	—	—	—	—	—	+	+	+	+
15	—	—	+	+	—	—	—	?	+
16	—	—	—	—	—	—	+	—	—
17	—	—	—	—	—	—	+	+	+
19	—	—	—	—	—	+	+	—	+

(See footnote, Table IV.)

TABLE IV. (May 16, 1913.)

Hemolysins after transfusions. Tests for hemolysis.

Readings after two hours at 37½° C. and twelve hours in ice-box.

	4	6	7	8	11	15	16	17	19
4	—	—	—	—	—	—	—	—	—
6	—	—	—	—	—	—	H	—	H
7	—	—	—	—	—	—	H	—	H
8	—	—	—	—	—	—	—	—	—
11	—	—	—	—	—	—	H	—	H
15	—	—	—	—	—	—	H	—	H
16	—	—	—	—	—	—	—	—	—
17	—	—	—	—	—	—	—	—	—
19	—	—	—	—	—	—	—	—	—

Cat 19 has had two transfusions from Cat 7. Cat 16 has had one transfusion from Cat 11.

Tables show marked development of agglutinins and hemolysins in Cat 19 and in Cat 16. Both cats, though transfused from different donors, have developed hemolysins for the red blood cells of one another's donors and for the red blood cells of two other cats.

TABLE V. (July 5, 1913.)

Further development of hemolysins.

Readings after three hours in thermostat and overnight in ice-box.

	4	6	8	11	15	18	19	20	21	22	23	24
4.....	—	—	—	—	—	—	—	—	—	—	?	—
6.....	—	—	—	—	—	+	H	—	—	+	+	—
8.....	—	—	—	—	—	—	+	—	—	+	—	—
11.....	—	—	—	—	—	—	H	—	—	—	—	—
15.....	—	—	—	—	—	—	H	+	—	+H	—	—
18.....	—	—	—	—	—	—	—	—	—	—	—	—
19.....	—	—	—	—	—	—	—	—	—	—	—	—
20.....	—	—	—	—	—	—	—	—	—	—	—	—
21.....	—	—	—	—	—	—	H	—	—	—	—	—
22.....	—	—	—	—	—	—	—	—	—	—	—	—
23.....	—	—	—	—	—	—	—	—	—	—	—	—
24.....	—	—	—	—	—	—	—	—	—	—	—	—

TABLE VI. (Sept. 16, 1913.)

Development of agglutinins and hemolysins.

Readings after two hours at room temperature.

	4	6	8	11	18	19	22	23	25
4.....	—	—	—	—	+	+	+	—	—
6.....	+	—	—	—	+	H	—	—	—
8.....	+	—	—	—	+	+	+	—	—
11.....	—	—	—	—	+	+H	—	—	—
18.....	+	—	—	—	+	+	+	—	—
19.....	+	—	+	—	—	+	+	—	—
22.....	—	—	—	—	+	+	—	—	—
23.....	—	+	+	—	+	+	+	—	—
25.....	—	—	—	—	+	+	—	—	—

Cat 18, two weeks since last of three transfusions, shows numerous agglutinins.

TABLE VII. (Oct. 18, 1913.)

Variability of agglutinins after transfusions.

Readings after two hours at room temperature.

	4	6	8	11	18	19	22	23	25	26	27
4	—	—	—	—	—	+	—	—	—	—	—
6	—	—	—	—	—	H	—	—	—	—	—
8	—	—	—	+	—	+	—	—	—	—	—
11	+	—	—	—	—	H	—	—	—	—	—
18	—	—	—	—	—	+	—	—	—	—	—
19	—	—	—	—	+	+	—	—	—	—	—
22	+	+	—	—	—	+	—	—	—	—	—
23	—	—	—	—	—	—	—	—	—	—	—
25	—	—	—	—	—	+	—	—	—	—	—
26	—	—	—	—	—	+	—	—	—	—	—
27	—	—	—	—	—	+	—	—	—	—	—

Cat 18, one month since last transfusion, shows agglutination of red blood cells of only one cat.

TABLE VIII. (Feb. 3, 1914.)

Further development of agglutinins and hemolysins.

Readings after two hours at room temperature.

	4	8	18	19	23	25	28	29	30	31
4.....	—	—	+	+	—	—	+	+	—	—
8.....	—	—	+	+	—	—	+	+	—	—
18.....	—	—	+	+	—	—	+	+	+	—
19.....	—	—	+	+	—	—	+	—	—	—
23.....	—	—	+	H	—	—	+	+	—	—
25.....	—	—	—	+	—	—	+	—	—	—
28.....	—	—	+	+	—	—	+	+	—	—
29.....	—	—	+	+	—	—	+	—	—	—
30.....	—	—	+	+H	—	—	+	—	—	—
31.....	—	—	+	+	—	—	+	—	—	—

Cat 18 has received three transfusions since last tests recorded in Table VII., and shows again numerous agglutinins.

Cat 19 has developed hemolysins for the red blood cells of two new cats (23 and 30).

The cats for whose red blood cells Cat 19 had previously possessed hemolysins are dead.

TABLE IX. (March 10, 1914.)

Development of hemolysins in two cats.

Readings after two hours at room temperature.

	8	18	19	23	25	28	29	30	31
8	—	—	+	—	—	+	+	+	—
18	—	+	+	—	—	+	+	+	—
19	—	+	—	—	—	+	—	+	+
23	—	+H	H	—	—	+	+	+	+
25	—	+	+	—	—	—	—	+	+
28	+	+	+	—	—	—	—	+	+
29	—	+	+	—	—	+	—	+	+
30	—	+H	H	—	—	+	—	+	+
31	—	+	+	—	—	+	—	+	—

Cat 18 received its sixth transfusion Jan. 23, 1914, from Cat 27, which was used as donor for the first time, and for the red blood cells of which Cat 18 possessed agglutinins. Cat 27 died Jan. 27, 1914, which prevented further tests with its blood.

The above table shows that Cat 18 has developed hemolysins for the red blood cells of Cats 23 and 30.

TABLE X. (July 23, 1914.)

Loss of agglutinins and hemolysins after discontinuance of transfusions.

Readings after two hours at room temperature.

	8	18	23	25	28	30	32	33	35	36
8.....	—	—	—	—	+	—	—	—	—	—
18.....	—	—	+	—	+	—	—	+	—	—
23.....	—	+	—	—	+	—	—	—	—	—
25.....	—	—	—	—	+	—	—	+	—	—
28.....	—	—	—	—	+	—	—	+	—	—
30.....	—	+	—	—	+	—	—	+	—	—
32.....	—	—	—	—	+	—	—	+	—	—
33.....	—	—	—	—	+	—	—	—	—	—
35.....	—	—	—	—	—	—	—	+	—	—
36.....	—	—	—	—	+	—	—	—	—	—

Cat 18 was the recipient of its seventh and eighth transfusions March 11 and April 10, 1914, Cat 23 being the donor. Cat 18 developed hemoglobinuria after the eighth transfusion, and possessed hemolysins for the red blood cells of Cat 23 on April 10, 1914 (not in tables).

The above table shows that Cat 18, three and a half months after its last transfusion, has lost most of its agglutinins and hemolysins. The only agglutinins which it has retained are for the red blood cells of Cats 23 and 30, for which it previously also possessed hemolysins.

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