Hemolytic Disease and Polycythemia in Parabiosis Intoxication

By Rosanna N. Chute, M.A. and Sheldon C. Sommers, M.D.

A COMPLICATION of experimental parabiosis is the development of various indications of incompatibility between the animal partners. Parabiosis poisoning or intoxication (Parabiosevergiftung) may involve one or several body systems. In the preparation of a large series of parabiotic rats for studies of radiation reactions, attention was attracted to the blood changes. One rat partner became plethoric, the other anemic. To reach a better understanding of the underlying mechanisms and the tissue changes, an analysis of the hematologic abnormalities was undertaken. About 76 such disharmonious rat pairs were studied.

Paul Bert first popularized experimental junctures of 2 animals. Möller-Christensen has reviewed rat parabiosis poisoning historically and emphasized the importance of serologic and toxic factors. Mayeda in 1921 believed rat blood isoagglutinins, which he demonstrated twice, to be responsible for this phenomenon. He also observed bone marrow damage and myeloid metaplasia. Parabiosis intoxication was ascribed by Matsuyama to different degrees of body vitality. Becher believed the circulatory system of the stronger rat partner pushed excess blood across the vascular connection, producing a polycythemia in the weaker rat. Hermannsdorfer thought the blood displacement to be due to toxic effects on vessel tone. Recently, Finerty and Panos observed a 64 per cent mortality in non-littermate parabionts due to parabiosis poisoning, and less frequent occurrence of this syndrome in littermates.

EXPERIMENTS

An operative technic of celiotomy, modified from Bunster and Meyer, has been employed. Rats from the Hisaw and Slonaker strains were paired according to strain, sex, weight, and when possible, littermates.

In approximately 30 per cent of 253 parabiont pairs parabiosis poisoning supervened. Healing and growth of hair along the anastomotic skin line were delayed. About ten to thirty days postoperatively, when good vascular connections had become established, the incompatibility became apparent. Either the left or right partner developed hyperemic ears, paws and tail; while the other was correspondingly pale and anemic (plate I, fig. 1). For convenience they may be referred to respectively as the “red” and the “white” rat. A normal rat would have pink ears, paws and tail.

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The "red" rat showed increasing hyperemia and edema of ears, extremities and tail. It was more active and warmer to touch than normally. Exudate at times appeared and crusted around the eyes and nose, in either partner. Meanwhile the "white" rat became paler, cold and inactive, with ruffled fur. It was anorexic, and its back humped. Either the "red" or "white" parabiont might be larger, and failure to appreciate this has caused some confusion in the German literature.5-4

Deaths generally occurred in one partner within one week. Although the "white" partner initially appeared sicker, it generally outlived the "red" rat. In either case the moribund state was characterized by labored breathing and complete inactivity. When one partner died, the other soon expired.

Hematology

Blood drawn during life by cardiac punctures of anesthetized rats showed characteristic gross differences. Despite careful technic the plasma and serum of the "red" rat were usually moderately hemolyzed. The blood from the "red" rat had an increased proportion of erythrocytes, with a hematocrit in three instances of 64 to 80 per cent. The corresponding "white" rat had a reduced hematocrit value of 21 to 22 per cent and in some a marked gross lipemia (plate I, fig. 2). The hematocrit range found in 5 normal parabiotic rat pairs was 41 to 50 per cent. Determinations of blood volumes10 in 6 pairs with parabiosis poisoning and 6 control pairs showed an abnormally increased blood volume in the plethoric rats and some decrease in the anemic partners. Plethoric rats had high blood pressures and anemic rats low blood pressures11 (table 1).

Erythrocyte counts demonstrated polycythemia, 11 to 13 million cells per cu. mm., in the "red" rats and moderate to severe anemia of the "white" partners (table 2). In blood smears of both there was a marked polychromatophilia. In both partners anisocytosis with spherocytes and target cells was present (Plate I, fig. 3). Reticulocytes were abnormally increased in the "white" partners. Nucleated erythrocytes circulated predominantly in the "white" rat, and were at times present in numbers from 75 to 635 per hundred leukocytes (plate I, fig. 4). Normoblasts and late erythroblasts were also seen in small numbers in the "red" parabionts.

Leukocytes were also occasionally increased in the "red" partners, and normal or decreased in "white" rats. Differential counts showed an eosinophilia in sev-
PLATE I

See legend, facing page.
eral "red" rats, and sometimes a relative neutropenia. Leukocytoid lymphocytes appeared increased in one or both partners (table 2).

Platelets were not observed to be altered significantly in number or appearance.

**Gross Pathology**

At necropsy, the mucous membranes and serous surfaces showed red and white color differences corresponding to external colorations. Vascularity was accentuated in the "red" parabionts. Heart, spleen, liver and kidney were much darker red in the "red" rat. Also its liver and spleen were enlarged. Bone marrow was

| Table 1.—Weights, Blood Volumes, Hematocrits and Systolic Blood Pressures of Parabiotic Rats
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<td>Blood Volume (% of body wt.)</td>
<td>Hematocrit</td>
<td>Systolic blood pressure</td>
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<td>10.0</td>
<td>—</td>
<td>—</td>
<td>79</td>
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<tr>
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<td>5.9</td>
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<tr>
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<td>193</td>
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<td>7.3</td>
<td>45</td>
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<td>85</td>
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<td>7.7</td>
<td>7.3</td>
<td>45</td>
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<th>Hematocrit</th>
<th>Systolic blood pressure</th>
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<tr>
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<tr>
<td>Average</td>
<td>117</td>
<td>98</td>
<td>9.5</td>
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darker red, denser and more compact in the "red" parabiont. Likewise its blood was dark red and abnormally viscous. Lymph nodes were enlarged in several "red" rats, while the thymus was usually small. Uterus and ovaries in "red" parabionts appeared to show increased growth, while they were shrunken in "white" partners. The pancreas was paler and denser in the "white" rat. Differences were not observed in the pituitary, thyroid, adrenals or testes.

**Microscopic Pathology**

The most striking abnormalities were present in the lymphoid and hematopoietic organs. Typically the "red" rat showed hyperplastic lymphoid tissues of
splenic white pulp and lymph nodes. Less regularly there was an increase in the size of lymphoid masses in peritracheal, peribronchial and intestine submucosal locations, and a thickened thymic cortex. Germinal centers in the spleen were particularly prominent, with the prominent malpighian marginal zones described in human hypersplenism. Reticulo-endothelial and plasma cells were augmented in splenic and lymph node sinusoids. Both here and in liver sinusoids there were frequently a striking erythrophagocytosis (plate II, fig. 5) and hemosiderin

Table 2.—Parabiosis Poisoning; Rats with Hematologic Studies

| Sex | Erythrocytes (millions/cu.mm.) | Leukocytes (per cent of 200 cells) | Nucleated Erythrocytes (per 100 WBC) | P | Small L | Leukocyte L | M | E | B
|-----|-------------------------------|-----------------------------------|-------------------------------------|---|---------|-------------|---|---|---
| 30 red F | 9.88 | — | 58 | 28 | 11 | 2.5 | 0 | 0.5 | 0
| 30 white | 4.38 | — | 22.5 | 57.5 | 16 | 3.5 | 0 | 0 | 0
| 31 red F | 8.63 | 14.7 | 0 | 14 | 41.5 | 14 | 0 | 0 | 0
| 31 white | — | 8.6 | 0.5 | 28 | 29.5 | 38.5 | 2.5 | 1.5 | 0
| 32 red M | 8.89 | 15.9 | 0.5 | 20.5 | 57 | 16.5 | 3.5 | 2.5 | 0
| 32 white | 12.6 | 2.0 | 19.1 | 40 | 5 | 0.5 | 0 | 0 | 0
| 33 red F | 10.20 | 16.2 | 23.0 | 20.5 | 57 | 26 | 10.5 | 5.5 | 0.5 | 0
| 33 white | 1.92 | 11.1 | 90.0 | 19.5 | 26 | 48.5 | 3.5 | 2.5 | 0
| 34 red M | 12.70 | 19.1 | 0.5 | 19.5 | 26 | 48.5 | 3.5 | 2.5 | 0
| 34 white | 3.33 | ? | 635.0 | 23 | 49 | 18 | 9.5 | 0.5 | 0
| 35 red M | 13.95 | 0.01 | 29.8 | 2.0 | 39.5 | 34 | 21 | 2.5 | 3 | 0
| 35 white | 3.60 | 1.63 | 18.7 | 40.0 | 55.5 | 28 | 11.5 | 4 | 1 | 0
| 36 red M | 12.65 | 12.5 | 0 | 23 | 36 | 38 | 2 | 1 | 0
| 36 white | 3.17 | 7.2 | 6.0 | 17.5 | 64 | 15 | 2 | 1.5 | 0
| 37 red M | 12.53 | 17.5 | 0 | 14.5 | 40.5 | 42.5 | 1.5 | 1 | 0
| 37 white | 6.37 | 6.3 | 75.0 | 24.5 | 50 | 20 | 5 | 0.5 | 0
| 38 red M | 11.35 | 0.50 | 23.8 | 0 | 10.5 | 40.5 | 46.5 | 10.5 | 2.5 | 0
| 38 white | 11.35 | 0.50 | 23.8 | 1.0 | 40.5 | 46.5 | 10.5 | 2.5 | 0 | 0

phagocytosis. Kupffer cells were prominent participants in these phagocytic activities (plate II, fig. 6).

Blood cells in the larger vessels were excessively packed into erythrocytic masses and rouleaux. This sludging observed microscopically corresponded to the gross viscosity of fresh blood drawn from living rats.

In the "white" rat lymphocytes were depleted in malpighian corpuscles, lymph nodes and other lymphoid depots. Large macrophages and reticulum cells with abundant pink cytoplasm remained, mixed with plasma cells. Splenomegaly was attributable to moderate or extreme exaggeration of hematopoiesis in the red pulp, including all blood cell lineages (plate II, fig. 7). Frequently extramedullary
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Hematopoiesis was present in moderate amounts also in liver sinusoids (plate II, fig. 8), and occasionally in lymph node or adrenal cortical sinusoids.

The bone marrow of the "red" rat was usually congested and at times hyperplastic (plate III, fig. 9). In the "white" rat there was a striking increase in marrow cell nuclear pyknosis and necrobiotic fragmentation, and marked hematopoietic hyperplasia of stem cells (plate III, fig. 10). The animals were sacrificed relatively early in full-blown parabiosis intoxication, so that time was not sufficient for development of the myelosclerosis reported elsewhere.6 The same sequences of marrow necrosis, compensatory hematopoietic hyperplasia and myelosclerosis with development of hemolytic anemia have been observed in human cases.13

The hyperplastic, degenerative and extramedullary hematopoietic activities described were not strictly limited to one rat partner. Lesser degrees of lymphoid hyperplasia and erythropagocytosis were at times seen in "white" rats, and rare hematopoietic foci appeared in liver sinusoids of "red" rats.

Effects upon the kidney of hemolytic anemia and hemoglobinemia were occasionally evident histologically. They consisted of hemoglobin casts in collecting tubules and hemosiderin pigmentation of tubular epithelium associated with tubular atrophy, typical of hemoglobinuric or so-called lower nephron nephrosis.34

Numerous other tissue changes in either or both parabiotic rats were less clearly associated with the blood disease. The small intestinal lamina propria was abnormally crowded with plasma cells and macrophages around lacteals. The same chronic perilymphangitis was seen in the intestinal mesentery, associated with proliferation of peritoneal mesothelium. Occasional rats showed a striking deposit of hyalinized collagen in the intestinal submucosa. Similar overgrowth and hyalinization of adrenal capsular fibroblastic tissues was noted. Endothelial cells of blood vessels, glomeruli and heart valves showed degenerative and proliferative alterations associated with hyaline and possibly fibrinoid degeneration of intercellular substances. Microscopic appearances simulated one or more of the so-called collagen diseases. For purposes of unity, these pathologic changes are discussed separately elsewhere.

Either one or both partners showed histologic evidences of the alarm reaction, including pyknosis and fragmentation of lymphocytes, depletion of pancreatic acinar cytoplasmic basophilia, adrenal cortical lipid depletion and hyperplasia of anterior pituitary acidophils.

No evidence of abnormal lipid deposition or lipodystrophy was found.

PLATE II

Fig. 5.—Spleen of a "red" parabiont. The prominent marginal zone of a malpighian corpuscle and erythropagocytosis are shown. (LH 33R). X500. Hematoxylin and eosin.

Fig. 6.—Liver of a "white" partner. Kupffer cells are prominent, with erythropagocytosis. (LH 36L). X1000. Hematoxylin and eosin.

Fig. 7.—"White" parabiont's spleen, contrasting with its partner's shown in figure 5. Red pulp has markedly increased extramedullary hematopoiesis, including numerous megakaryocytes. (LH 34L). X500. Hematoxylin and eosin.

Fig. 8.—Extramedullary hematopoietic foci in the liver of a "red" parabiont; partner's liver shown in figure 6. This pair showed hepatic hematopoiesis and erythropagocytosis in each partner, which is unusual. (LH 36R). X1000. Hematoxylin and eosin.
PLATE II

See legend, facing page.
Chemical Study

Extensive analysis of the lipemic plasma was not made, but dried films of the plasma were strongly stained by Sudan Black B, which is indicative of fat. Pretreatment with cold 80 per cent alcohol reduced the fat staining about one-half and hot 80 per cent alcohol removed all but a trace of the material. This is considered to show that the lipemia was composed mainly of neutral fat and phospholipids.

Serologic Studies

In view of the resemblance to human transfusion reactions, hemolytic anemia and erythroblastosis, the condition described in the disharmonious parabiont rats would presuppose the presence of incompatible blood groups and hypersensitization. To our knowledge there is no other evidence of blood groups in rats. Direct cross-matching using standard technic for blood grouping consistently failed to produce agglutination or hemolysis.

In an attempt to determine the presence of incomplete antibodies by applying modified Coombs technics to rats, anti-rat-serum was prepared by immunizing rabbits. Pooled normal rat serum was injected, once intraperitoneally, and thereafter 15 times intravenously into each of 6 rabbits over a period of four weeks. One week after the last immunizing injection, sera were collected from the rabbits' ear veins, and kept at ice box temperature in serum bottles with preservative added. The maximum test tube agglutination titer against saline washed normal rat erythrocytes was 1:64,000. Direct tests on slides of the erythrocytes of normal rats or rats with parabiosis poisoning and anti-rat-serum were uniformly negative.

Indirect tests were carried out with 1 per cent washed normal rat erythrocytes in saline, incubated with parabiotic poisoning sera for 30 minutes. The sera were then replaced by saline after centrifugation. One drop of these sensitized cells was added to 0.5 cc. of serial dilutions of anti-rat-serum.

As indicated in table 3, the “red” and “white” rat titers of incomplete iso-agglutinins were greater than titers of normal rat erythrocytes with the rabbit anti-rat-serum. In 4 parabiosis intoxication cases a greater increase in incomplete hemagglutinins was demonstrated in “red” rat partners, and in 2 cases “white” rats had higher titers.

Trypsinized rat erythrocytes were prepared and incubated at 37 C. for one-half hour in 1 per cent saline solution with serial dilutions of sera from disharmonious rats. Readings were corrected for the slight agglutination of trypsinized red cells in saline. Table 4 shows the enhanced hemagglutination and occasional hemolysis observed, which were more marked in the “red” partners' sera.

Attempts were made with the macromolecular compound polyvinylpyrrolidone (PVP)* to enhance incomplete antibody titers in cross-matched cells and sera of disharmonious rats, incubated together one-half hour with 10 per cent PVP, centrifuged and read for agglutination. No differences of titer were demonstrated, and the results were obscured by the tendency to agglutination of saline

* Appreciation is expressed to the General Aniline Film Corp., 22 Center Square, Easton, Pa., for providing the PVP.
controls. Mayeda twice observed hemolysis of "white" rat erythrocytes by "red" parabiont serum after 2 hours incubation together, read after 5 hours.

**Discussion**

The basic disease process studied in this group of disharmonious rat parabionts was a hypersensitization to circulating erythrocytes, simulating human erythroblastosis fetalis or acquired hemolytic anemia. From clinical observations, hematologic, tissue and serologic studies it was established that the plethoric "red" rat partner had reticulo-endothelial and lymphoid hyperplasia, with leukocytoid lymphocytosis and production of isoantibodies agglutinating and hemolyz-

![Plate III](image)

**PLATE III**

*Fig. 9.*—Bone marrow section of “red” parabiotic rat (LH 37L). The marrow is congested, and the small cells with black appearance of cytoplasms represent eosinophilic leukocytes. X500. Giemsa.

*Fig. 10.*—Section of bone marrow from the “white” partner (LH 37R) of rat shown in figure 9. Marked hyperplasia of stem cells, many immature cells with pyknotic nuclei, and increased nuclear debris are observed. X500. Giemsa.

ing erythrocytes from the anemic “white” parabiont. Destruction of the anemic rat's erythrocytes was evident from its anemia, spherocytosis, reticulocytosis, erythroblastosis and the erythrophagocytosis. Enhanced extramedullary hematopoiesis, necrosis of individual bone marrow cells and reactive marrow hyperplasia were observed in the anemic partner.

Some peculiarities unknown in single animals or persons could be ascribed to the parabiotic state. The plethoric “red” partner had polycythemia, increased blood volume and high blood pressure. Since there was histologic evidence for maximal blood destruction and no striking increase in hematopoiesis in the plethoric parabiont, this polycythemia evidently had a mechanical basis. The simplest ex-
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TABLE 3.—Indirect Coombs-type Tests in Parabiosis Poisoning

<table>
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<th>Serial Dilutions of Rabbit Anti-Rat-Serum in Thousands</th>
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<tr>
<td></td>
<td>1:32</td>
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<tr>
<td>33 red</td>
<td>++</td>
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<tr>
<td>33 white</td>
<td>+</td>
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<td>34 red</td>
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<td>38 red</td>
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<tr>
<td>38 white</td>
<td>+</td>
</tr>
<tr>
<td>Normal</td>
<td>+</td>
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Normal rat erythrocytes agglutinated to 1:64,000 (+) with fresh rabbit anti-rat-serum #1, later titer below 1:32,000.

TABLE 4.—Trypsinized Erythrocyte Agglutinations

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<th>Serum from</th>
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<td>33 red</td>
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<td>33 white</td>
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<td>37 red</td>
<td>*+++</td>
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<td>37 white</td>
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* Hemolysis of trypsinized erythrocytes.

The explanation would be that the “red” rat had its circulation clogged with sludged antibody-coated erythrocytes from the “white” partner. Mechanisms for the destruction and processing of these agglutinable erythrocytes were not adequate in the plethoric rat, and the coated incompatible red cells persisted in its circulation. Hemoglobinemia and hemoglobinuric nephrosis developed as complications.
The possibility of localized vasospasm in the “white” rat along the anastomotic line was not excluded, but seemed less likely.

In the anemic rat the circulating erythrocytes included mature cells, which presumably had crossed the vascular connection from the plethoric partner, and its own reticulocytes and nucleated erythrocytes just produced. It is believed that once transferred to the plethoric rat, the anemic rat’s erythrocytes mostly became so “sticky” and agglutinable due to antibody coating that they could not readily renegotiate the small vascular connections between the parabionts and were trapped. In normal parabionts blood transfer is of the order of 0.6 per cent of blood volume per minute.17

Occasional neutropenia possibly pointed to similar effects upon leukocytes, but this would require further study, since the extreme erythropoiesis might have disturbed myelopoiesis. No abnormality of platelets was observed. Endothelial damage played a prominent part in parabiosis intoxication, but its pathologic sequences require more extensive consideration than is pertinent here.

No concomitant cross-immunizations of each partner against the other was demonstrated hematologically, although some other tissue hypersensitizations were bilateral.

Least susceptible of explanation was the marked gross lipemia of the anemic rat. It was composed of neutral fat and phospholipids. Despite the perilymphangiitis described in the intestinal lacteals and mesenteries of both parabionts, the lipemia probably was not based on abnormal fat absorption except secondarily. Whether it represented a lipemia of bleeding associated with decreased serum protein,18 was a byproduct of glomerular damage,19 or had some other origin is uncertain. Further study is required to elucidate this problem.

Summary

Disharmonious parabiotic rats were studied, which had clinical, hematologic, pathologic and serologic evidences of blood incompatibility, isohemagglutinin hypersensitization and hemolytic anemia. Spherocytosis and increased incomplete isohemagglutinin antibodies, demonstrated by indirect Coombs-type agglutinations and with trypsinized rat erythrocytes, were found in both parabionts. One partner became anemic with reticulocytosis, erythroblastosis, bone marrow necrobiosis, reactive hematopoietic hyperplasia and enhanced extramedullary hematopoiesis. The other parabiont showed lymphoid and reticulo-endothelial hyperplasia, leukocytoid lymphocytosis and erythrophagocytosis. It was plethoric with polycythemia, increased blood volume and high blood pressure. The polycythemia was explained by mechanical trapping of sludged antibody-coated incompatible erythrocytes, which the reticulo-endothelial system was inadequate to process. Complicating hemoglobinemia and hemoglobinuric nephrosis developed. Other manifestations of parabiosis intoxication are briefly discussed.

References

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