The peripheral white blood cell count under stable conditions is usually a fairly constant phenomenon. Data from studies on animals indicate that up to five times the number of lymphocytes present in the bloodstream at any one time are delivered to the circulation via the thoracic duct during each twenty-four hours. Since the lymphocyte count of the blood remains relatively unchanged, an equal number must be removed from the circulation during the same period. A dynamic equilibrium exists, therefore, between lymphocytes entering and leaving the circulation. The mechanism for controlling this equilibrium is poorly understood. Several mechanisms have been proposed. Loss of lymphocytes in the saliva and in the intestines has been demonstrated but in other experiments intravenously injected lymphocytes were rapidly removed from the circulation in the absence of the entire gastrointestinal tract. Adrenocortical hormones are considered to play a role in the removal of lymphocytes, perhaps by lysis in the germinal centers of the lymph nodes. The leukopenia of anaphylaxis in dogs and rabbits is associated with adherence of white cells to the pulmonary capillary walls. Similar adherence of lymphocytes to the capillary walls of ear vessels has been shown to occur in the temporary leukopenia following intravenous injections of glycogen, acacia, and certain proteins in rabbits. Intravenous introduction of leukocytes and their disintegration products into rabbits also results in leukopenia with increased numbers of cells being found in the liver and spleen.

The increased peripheral white cell counts in leukemia of man may result from interference with some poorly understood removal mechanism as well as from an accelerated production of such cells. An attempt was made to determine in man the rate of delivery of lymphocytes from the thoracic duct into the vascular circulation. A catheter was placed in the left innominate vein just proximal to the thoracic duct opening. It was found impossible to demonstrate an increase in the lymphocyte count. Approximately 400,000 lymphocytes flow into the circulation each second from the thoracic duct. At a blood flow of 10 cc. per second through the innominate vein, the increase in the blood lymphocyte count in the innominate vein would be in the order of only 2 per cent, and errors in counting lymphocytes exceed 2 per cent.

To detect and study a possible white cell removal mechanism in the lungs of man, experiments were designed in which large numbers of white blood cells were...
TRANSFUSION OF LEUKEMIC LEUKOCYTES IN MAN

Transfused into selected volunteer patients. Patients with leukemia represent one source of large numbers of fresh white cells. By the introduction of such cells into vessels leading to the lungs and by examination of blood samples from arteries close to the heart it was possible to detect increases or disappearance of the cells in the arterial blood. The presence or absence of white cells after their circulation through a tissue or organ would indicate whether or not that site plays a part in the removal mechanism under the conditions of the experiment.

Table 1.—Hematologic Data in Experiment 2

<table>
<thead>
<tr>
<th>Time: Minutes and Seconds</th>
<th>WBC/cu.mm.</th>
<th>Mononuclear Cells/cu.mm.</th>
<th>PMN/cu.mm.</th>
<th>EOS/cu.mm.</th>
<th>T-1824 µ/cc.</th>
<th>Per cent Donor Blood</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Donor</strong></td>
<td></td>
<td></td>
<td></td>
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Recipient: Averaged Pretransfusion Counts

<table>
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<th>Time: Minutes and Seconds</th>
<th>WBC/cu.mm.</th>
<th>Mononuclear Cells/cu.mm.</th>
<th>PMN/cu.mm.</th>
<th>EOS/cu.mm.</th>
<th>T-1824 µ/cc.</th>
<th>Per cent Donor Blood</th>
</tr>
</thead>
<tbody>
<tr>
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<td>10,000</td>
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Recipient: Left Femoral Artery

<table>
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<th>Mononuclear Cells/cu.mm.</th>
<th>PMN/cu.mm.</th>
<th>EOS/cu.mm.</th>
<th>T-1824 µ/cc.</th>
<th>Per cent Donor Blood</th>
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</thead>
<tbody>
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<td>10,900</td>
<td>600</td>
<td>0</td>
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<tr>
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<td>3,400</td>
<td>10,900</td>
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<tr>
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<td>36,100</td>
<td>12,000</td>
<td>8,700</td>
<td>430</td>
<td>12.7</td>
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</table>

Recipient: Right Median Basilic Vein

<table>
<thead>
<tr>
<th>Time: Minutes and Seconds</th>
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<th>Mononuclear Cells/cu.mm.</th>
<th>PMN/cu.mm.</th>
<th>EOS/cu.mm.</th>
<th>T-1824 µ/cc.</th>
<th>Per cent Donor Blood</th>
</tr>
</thead>
<tbody>
<tr>
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<td>3,400</td>
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<td>530</td>
<td>0</td>
<td>0</td>
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<tr>
<td>0 27</td>
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<td>3,400</td>
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<td>530</td>
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<tr>
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<td>9,100</td>
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</table>

**Donor Blood:** Volume: 86 cc.; plasma hematocrit 62.5 per cent

**Recipient:** Plasma hematocrit, average 55 per cent; range 53.5–55.5 per cent

**METHOD**

Rapid transfusions of leukemic blood with high mononuclear cell counts were given to volunteer nonleukemic, cancer-bearing patients in whom the prognosis was of short duration. The recipients' white blood and differential cell counts were determined at close intervals from various parts of the circulation throughout the procedure. T-1824 (Evans' blue) dye was added to the donor blood and determined in the donor and each recipient specimen. The dilution of the dye in the recipient's specimens enabled prediction of expected cell counts. These values were compared with those obtained by count-

**FIG. 1.—Experiment 1:** The donor was AD, a 66 year old white male with a diagnosis of chronic lymphogenous leukemia. The recipient was KS, a 56 year old white female with a diagnosis of giant follicular lymphosarcoma. Arterial, venous and right ventricular leukocyte counts during and after transfusion of 500 cc. leukemic blood. Time is measured from the beginning of the transfusion. (10-5-48.)
Fig. 1.—See legend, opposite page
Fig. 1.—See legend, opposite page
ing. The count of less than the predicted value indicated removal of cells. Comparison of approximately simultaneous counts from various sampling sites demonstrated the part of the circulation in which removal took place.

Transfusions were indirect, with citrate or heparin anticoagulant. Blood was administered usually within four hours of its withdrawal. Leukocyte counts were done with National Bureau of Standards certified pipets and counting chambers, and the greater of either 600 cells or two complete chambers were counted. Wright's stained cover slip smears were used for differential counts and at least 600 cells were counted. Lymphocytes and monocytes were recorded together as mononuclear cells. Plasma dye concentrations of T-184 were measured spectrophotometrically. Hematocrits were determined on a donor and several recipient specimens.

An example of the calculations follows: in Experiment 6 (fig. 6), T-184 concentration in the donor's plasma was 1447/cc. The plasma hematocrit was 68 per cent; therefore, the plasma T-184 content per cubic centimeter of whole blood was 0.68 X 144 or 97.9 y. An arterial specimen taken 19 minutes 15 seconds after beginning the transfusion contained 7.84 y of T-184/cc. plasma and had a plasma hematocrit of 56 per cent. The plasma T-184 content per cubic centimeter of whole blood was, therefore, 0.56 X 7.85, or 4.40 y of T-184. The percentage of donor blood in this specimen was calculated to be 4.40/97.9 X 100, or 4.5 per cent. The percentage of original recipient blood in the mixture was 100 - 4.5, or 95.5 per cent. The donor blood contained 85,800 mononuclear cells/cu.mm. and the recipient's blood before transfusion had 12,000 mononuclear cells/cu.mm. A mixture of the two bloods in the above proportions should contain (85,800 X 4.5 per cent) plus (12,000 X 95.5 per cent) or 500 mononuclear cells/cu.mm. This number was compared with the value found by white cell and differential counts, which was 560 mononuclear cells/cu.mm. The difference between these two figures indicates a removal of 200 mononuclear cells/cu.mm. Similar calculations were done for polymorphonuclear cells and eosinophils.

The question of the significance of the differences between the cell counts predicted from the T-184 and plasma hematocrit concentrations and the cell counts actually obtained was complicated by the inherent inaccuracies of leukocyte and differential counting technics. Berkson has shown that the error in a leukocyte count is a function of, at least, the number of cells counted, the number of pipets used, the number of hemocytometer fillings and, usually, an error due to the personal equation of the technician doing the counting. His findings, plus estimated errors in the differential counts, appear to indicate that, within the range of leukocyte and differential counts generally found in this experiment, the difference between an observed and a predicted count was significant at about the 5 per cent level, using Fisher's "t" test of significance, when the difference exceeded 17 per cent of the observed count. This value is based on the assumption that white cell and differential errors lay within the limits found from the formulae employed but ignores the added significance resulting from consistency of serial results.

No allowance was made for loss of T-184 into areas outside the circulation. The magnitude of the loss is indicated by the discrepancy of approximately 10 per cent between blood volume estimates by dye and radioactive red cell methods. Disappearance of dye from the circulation would result in lower predicted counts, and the difference should, therefore, be even larger than the one recorded.

Results

Eight transfusions of leukemic blood from 7 donors were given to 5 volunteer nonleukemic cancer-bearing recipients. Six donors had lymphogenous and 2 myelogenous leukemia. Hematologic data have been presented pertaining to six of these transfusions (figs. 1-6; table 1). The data in two remaining experiments were

Fig. 2.—Experiment 2: The donor was CB, a 56 year old white male with myelogenous leukemia; the pathologic cells were immature and quite uniform in appearance. Inasmuch as they were morphologically mononuclear cells, they were counted as such. The recipient was FB, a 58 year old white male with an anaplastic carcinoma of the bronchus with metastases to the left axilla. Arterial and venous blood leukocyte counts during and after leukemic transfusion. Predicted counts were estimated from dilution of T-184 dye mixed with the transfused blood. Observed mononuclear cell counts fell below predicted counts first in arterial blood, later in venous blood. (The shaded graph at the bottom represents the difference between predicted and found mononuclear cell counts.) (11-4-48.)
Fig. 3.—Experiment 3: The donor was TL, a 55 year old white male with a diagnosis of chronic lymphogenous leukemia. The recipient was FB, a 58 year old white male with an anaplastic carcinoma of the bronchus with metastases to the left axilla. Observed and predicted arterial and venous leukocyte counts during administration of 645 cc. leukemic blood over twenty-five minutes. Arterial mononuclear cell deficits precede venous. (The shaded graph at the bottom represents the difference between predicted and found mononuclear cell counts.) (11-17-48.)
LANMAN, BIERMAN AND BYRON

Volumes of leukemic blood transfused varied from 220 cc. to 645 cc., with from 80,000 to 302,000 leukocytes/cu. mm. consisting of from 93.9 to 99.1 per cent mononuclear cells. Transfusion times varied from 20 seconds to 25 minutes. All transfusions were given intravenously except for the intra-arterial transfusion of Experiment 6 (fig. 6).

Variations occurred in the recipients' white cell counts and were due largely to changes in the mononuclear cell counts. These rose transiently in all cases except one (Experiment 5 [fig. 5]); maximum values varied from 168 to 411 per cent of the respective pretransfusion counts. These rises occurred within ten minutes of the beginning of the transfusions. Predicted mononuclear cell counts calculated by the dye dilution method rose more rapidly and reached higher maximum values of from 319 to 1057 per cent of pretransfusion counts.*

Mononuclear cell counts fell below predicted values in arterial blood earlier than in venous blood. The deficit in arterial blood in Experiments 2, 3, and 4 was 113, 21 and 73 per cent, respectively, of the correspondingly observed counts within one minute of the beginning of the transfusion. Simultaneous venous specimens showed a maximum deficit of 2.5 per cent and contained insignificant amounts of T-1824, as reflected by predicted mononuclear cell counts of only 0, 26, and 8 per cent, respectively, above pretransfusion levels.

Leukemic blood was given into the right femoral artery in Experiment 6 (fig. 6). Samples were taken from the right femoral vein and left brachial artery. During the first three and one-half minutes of the 300 cc. transfusion, the venous mononuclear cell counts fluctuated widely, due to the irregular rate of administration, but each lay within 15 per cent of its expected value. Arterial counts were 29 per cent below expected levels in 1 minute 45 seconds and 54 per cent below in 2 minutes 45 seconds.

In Experiment 1 (fig. 1), a right ventricular blood sample taken by cardiac catheter during the transfusion contained 9533 mononuclear cells per cu.mm. The average pretransfusion mononuclear cell count was 144/cu.mm. This rise of 6,613 per cent was the highest observed. No dye was used in this transfusion, so that no prediction of expected counts was possible.

Polymorphonuclear neutrophil and eosinophil cell counts did not deviate far from predicted levels calculated by the dye dilution method. Expected counts for polymorphonuclear neutrophil cells were within ±25 per cent of counted values in 54 of 59 determinations. Expected counts for eosinophils were within 50 per cent of counted values in 51 of 59 determinations. Since relatively few of these cells were present, small absolute differences between counted and predicted values appeared as large percentage differences.† In contrast to the consistency with which predicted mononuclear cell counts exceeded observed levels, predicted counts for polymorphonuclear neutrophil cells and eosinophils varied at random above and below observed levels.

* Differences between observed and predicted counts are thought to be significant when the difference exceeds 17 per cent of the observed count (see Method).

† The statistical analysis for appraisal of the significance of differences between observed and predicted counts does not apply to eosinophils.
Fig. 4.—See legend, opposite page
In the eight leukemic transfusions, three reactions occurred. Recipient GS (Experiment 4 [fig. 4]) received two transfusions of leukemic cells from different donors and had a chill after each. No respiratory symptoms were observed and no leukopenias followed these reactions. In Experiment 5 (fig. 5), a second transfusion of leukemic blood eighteen days later from the same donor into the same recipient resulted in an immediate, severe reaction, with dyspnea and cyanosis, followed in a few minutes by asthmatic breathing, musical and moist rales, a chill and fever. The recipient's total white cell count fell from a pretransfusion level of 18,430/cu.mm. to 1,700/cu.mm. within five minutes, during which time 35 billion white cells (95 per cent mononuclear) were infused. Polymorphonuclear neutrophil cells fell to 8 per cent of pretransfusion levels, mononuclears to 32 per cent, and eosinophils to 2 per cent. A count taken fifteen minutes after the start of the transfusion showed a slight rise toward the pretransfusion level.

Three recipients ultimately died of their pre-existing disease and one from injuries sustained in an automobile accident. The intervals between transfusion of leukemic cells and death were 19, 43, 66 and 100 days. One recipient (NH) is living eight months after transfusion. During these intervals and following the immediate post-transfusion period the total white cell counts showed no striking changes. The highest count observed in any recipient was less than double his pretransfusion level. Immature cells suggestive of leukemia were not seen in the recipients' specimens. One recipient (NH) developed an eosinophilia first noted four days after the transfusion of leukemic cells and persisting eighty-four days later. One other patient (GS) developed an eosinophilia of 6 per cent after one transfusion of leukemic cells and 2.1 per cent after a second. Two patients (FB and PG) had pre-existing eosinophilias unchanged by transfusion of leukemic cells.

**Discussion**

Administration of large numbers of leukemic white cells in these transfusions was followed rapidly by removal of approximately an equal number of leukocytes from the recipient’s circulation. The data indicate that the site of removal for these leukocytes was in the lesser circulation. Experiment 1 (fig. 1) demonstrated that intravenously injected mononuclear cells reached the right ventricle, increasing the count to 6,613 per cent of the pretransfusion level, the highest rise found in any case. In Experiments 2, 3 and 4 (figs. 2, 3, 4), arterial mononuclear cell counts were significantly below the predicted levels within one minute or less, as calculated by the dye dilution method. Simultaneous venous specimens contained little or no T-18s4, presumably because insufficient time had elapsed for the donor blood to circulate as far as the venous sampling sites. No significant recirculation could have occurred under these conditions, and the cells must have been removed between

---

**Fig. 4.**—Experiment 4: The donor was SC, an 8 year old white female with a diagnosis of acute lymphogenous leukemia. The recipient was GS, a 60 year old white male with a diagnosis of inoperable adenocarcinoma of the rectum with metastases. Observed and predicted leukocyte counts during and after transfusion of 367 cc. leukemic blood in 4 minutes 45 seconds. Arterial mononuclear cell deficits appeared before venous deficits. (The shaded graph at the bottom represents the difference between predicted and found mononuclear cell counts.) (12.6–48.)
Experiment 5: The donor was EA, a 68 year old white male with a diagnosis of chronic lymphogenous leukemia. The recipient was PG, a 54 year old white male with a diagnosis of squamous cell carcinoma of the esophagus. Arterial and venous leukocyte counts during and after transfusion of 22.0 cc. leukemic blood. Recipient had an immediate reaction resembling anaphylaxis. Observed polymorphonuclear cell and eosinophil counts fell significantly below levels predicted by T-182.4 dye dilution in this experiment only. (11-2-48.)
the intravenous infusion point and the arterial sampling site. The pulmonary circulation seems the most likely site for this removal.

The blood was transfused into the right femoral artery in Experiment 6 (fig. 6) to demonstrate whether capillaries other than those in the lung could remove transfused leukocytes. Mononuclear cells in approximately predicted numbers were obtained from right femoral vein blood for over three minutes after the start of transfusion. This observation demonstrates that mononuclear cells were not removed from the blood while circulating through the capillary bed of the right leg. However, significant mononuclear cell deficits were found in the brachial artery blood in the first specimen containing transfused blood and dye which had passed through the lungs. The white cells evidently passed a peripheral capillary bed lying predominantly in muscle but were removed in the lesser circulation. No data for other capillary beds were obtained.

Weisberger and co-workers have recently reported that intravenous injection of intact or disintegrated leukocytes into rabbits was followed by leukopenia. Following transfusion of white cells labeled with radioactive phosphorus, the greatest concentration of radioactivity was found in the lungs. Histologic examination of lung tissue revealed capillaries containing large numbers of leukocytes without edema or fibrin formation. Similar but less striking increases in the number of white cells were observed in liver and spleen.

The only severe reaction to these transfusions occurred in Experiment (fig. 5). The recipient (PG) was receiving a second transfusion from the same leukemic donor employed eighteen days previously. Signs and symptoms of an anaphylactoid reaction occurred, with apprehension, hypotension, cyanosis, sweating tachycardia and rapid respirations. The hematologic data indicate a possible mechanism for the production of certain of these symptoms. The patient had a total of approximately 86 billion circulating white cells before the transfusion, a figure derived from a blood volume of 4,800 cc. by the radioactive phosphorus method and a white cell count of 18,420/cu.mm. Thirty-five billion leukocytes were transfused, so that immediately after transfusion 121 billion circulating white cells (35 plus 86 billion) should have been present. Only 12 billion were found, as estimated from the blood volume, and an averaged white cell count of 2,633/cu.mm. one and one-half minutes after the end of the transfusion. The number of cells removed from the circulation in five minutes was, therefore, 109 billion (121 billion minus 12 billion). The mean arterial white cell count for the first five minutes after start of the transfusion was below 10,000/cu.mm. so that the blood flow required to transport 109 billion cells to any given organ exceeded 10,000 cc., or 2,000 cc. per minute for five minutes. Assuming that the white cells were removed in a single organ and that the organ could remove 100 per cent of the white cells flowing through it, the only organs with sufficient blood flow to remove this number of cells are heart, lungs, and liver. Most other viscera have been excluded as sole white cell removal sites.

If the white cells removed from the circulation in Experiment adhered to the pulmonary capillary walls, their volume may have been sufficient to contribute sig-
Fig. 6.—Experiment 6: The donor was HV, a 66 year old white woman with a diagnosis of untreated chronic lymphogenous leukemia. The recipient was NH, a 72 year old white male with lymphosarcoma of the right orbit. Arterial and venous leukocyte counts during and after intra-arterial transfusion of 350 cc. leukemic blood. Venous counts were taken from the extremity into which blood was transfused and closely paralleled predicted counts for several minutes. Significant arterial mononuclear cell deficits appeared early. The 14-minute delay before the second portion of the transfusion was caused by difficulty in obtaining venous blood samples. (The shaded graph at the bottom represents the difference between predicted and found mononuclear cell counts.) (J-18-49.)
nificantly to the respiratory symptoms observed. The packed volume of white cells in the donor blood was 6 per cent, with a white cell count of 155,625/cu.mm., or 2.6 billion packed white cells per cubic centimeter. The lungs are estimated to contain about 9 per cent of the blood in the body at any one time or, in this instance, approximately 430 cc. About 25 per cent of this is estimated to lie in the large pulmonary vessels, so that the capillary bed contains about 320 cc. The 109 billion white cells removed from the circulation occupied about 40 cc. or about 12 per cent of the volume of the pulmonary capillaries. It would not be surprising that impaired aeration of blood and obstruction to blood flow might follow such deposition of cells and contribute to the cyanosis, fall in blood pressure and tachycardia observed in this reaction.

The possibility of an immunologic reaction to the second transfusion of whole blood from the same donor must also be considered as responsible for the response and leukopenia.

In 1924, Webb demonstrated an immediate, profound leukopenia in anaphylactic shock induced by injections of horse serum into sensitized dogs. The leukopenia, which lasted one-half to three hours, was associated with adherence of clumps of leukocytes to capillary walls in the lungs but not in other organs. During development of the leukopenia, the white cell count in pulmonary vein blood was below that in pulmonary artery blood. Andrewes observed similar massing of leukocytes in the pulmonary capillaries of rabbits associated with a leukopenia induced by intravenous administration of certain bacteria.

No arterial specimens were obtained within the first three minutes of the transfusion in Part 2 of Experiment 2 and in Experiment 5 (figs. 2, 5). Since some recirculation had occurred, it could only be concluded that some removal mechanism was operating. However, in Experiment 2 (fig. 2), the 5-minute arterial specimen showed a deficit during the infusion of white cells into the venous side of the lungs. Experiments 1, 3, 4 and 6 (figs. 1, 3, 4, 6), however, indicated the lungs as the most likely site of the three possible organs in which the white cells were removed.

Transfusions of leukemic blood into nonleukemic human recipients or into a recipient with a different kind of leukemia have been performed in the past, and in no case was the disease transmitted. In fact, Thiersch deliberately attempted to transmit leukemia in man with various tissues and by many routes. Observations up to two years on some of the patients failed uniformly to reveal any evidence of transmission. None of our recipients at any time after transfusion had clinical or laboratory evidence of leukemia, but because cancer-bearing patients with short prognoses were chosen as recipients, the longest period of observation was nine months in one case (NH). On the other hand, the disease is transmissible in various mammals by transfusion and in certain inbred strains of mice by intravenous transfer of even a single leukemic cell.

Interpretation of the eosinophilia occurring after transfusion of leukemic cells in 2 cases is difficult because eosinophilia occurs in many patients with far advanced neoplastic disease, and was noted in 2 other recipients (FB and PG) before a transfusion of leukemic cells was given.

A pulmonary white cell removal mechanism is not known to function in the physiologic control of white cell levels and is certainly not the sole route by which
white cells leave the circulation. However, the sudden removal of a large volume of leukocytes in the lungs in anaphylactic shock might be in part responsible for the cardiorespiratory symptoms, which could then be relieved by a reversal of the removal process.

These studies indicate the capacity and possible importance of white cell removal mechanisms. If white cell removal is the result of an altered relationship between leukocytes and the endothelium of certain vessels, knowledge of factors that alter the "stickiness" of either leukocytes or the endothelial walls may be important in the understanding and possible therapy of conditions associated with pathologic changes in white cell levels. The classic concept of the leukemias as neoplastic diseases with unregulated and usually excessive proliferations of leukocytes has not led to any striking advance in understanding the pathologic physiology involved. The concept of an alteration of the leukocyte removal system as an important mechanism in leukemia may be of value as a different approach to the problems of etiology and therapy.

Summary and Conclusions

1. Eight transfusions of leukemic blood containing up to 143 billion mononuclear cells were rapidly infused into 5 nonleukemic cancer-bearing recipients. Each transfusion was followed by a transient rise in the recipients' leukocyte count which was less than anticipated by dye dilution methods in five experiments. The observed rise was due entirely to an increase in the mononuclear cell count. The data from two experiments were considered unreliable and were not reported with regard to the behavior of the white cells.

2. The data are interpreted as demonstrating removal of leukemic leukocytes from the recipient's blood in the pulmonary circulation.

3. A reaction resembling anaphylaxis occurred following one transfusion of leukemic cells, and was associated with an immediate profound leukopenia. It is suggested that the sudden removal in the lesser circulation of a volume of white cells estimated at 40 cc. may have caused the severe observed respiratory and circulatory symptoms.

4. Sustained eosinophilia of unknown etiology was observed following transfusions of leukemic cells in 2 recipients.

5. Hematologic and clinical observation indicated that leukemia was not transferred to any recipient during the short period of observation that followed.

References

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TRANSFUSION OF LEUKEMIC LEUKOCYTES IN MAN: HEMATOLOGIC AND PHYSIOLOGIC CHANGES

JONATHAN T. LANMAN, HOWARD R. BIERMAN and RALPH L. BYRON, JR.