THROMBOPATHIC THROMBOCYTOPENIA: SUCCESSFUL TRANSFUSION OF BLOOD PLATELETS

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The opportunity presented itself recently for the study of a case of chronic idiopathic thrombocytopenic purpura characterized by the presence of unusually large agranular platelets. Several features indicated that the platelets were physiologically defective and led to the diagnosis of a thrombopathic or thrombasthenic type of thrombocytopenic purpura. When a blood transfusion from a polycythemic donor with an excessive number of normal blood platelets was administered by means of silicone coated syringes, it was possible to make certain studies of platelet physiology and platelet survival time. The resulting observations and the deductions drawn from them form the basis of this report.

CASE REPORT

L. W., a 94 year old white male of Jewish parentage, was admitted to the Boston Floating Hospital* on May 19, 1949, with the chief complaint of epistaxis of six weeks’ duration. Hemorrhagic phenomena had been present since the age of 12 years, when numerous ecchymoses became apparent. None had been observed earlier. A blood examination at the age of 1 years revealed a low platelet count. Subsequently, frequent severe nose bleeds occurred, requiring transfusions on several occasions. Splenectomy was performed when the patient was 14 years old and resulted in an immediate rise in the platelet count and in the disappearance of symptoms for a period of six months. However, the platelet count then dropped, the nose bleeds recurred and occasional transfusions were again required.

There was no family history of hemorrhagic diathesis. Both parents were healthy and blood examinations showed no abnormality of the platelets. Six weeks before admission the patient had a severe hemorrhage from the right nostril following slight trauma to the nose. Several transfusions were required. Since then, five severe nasal hemorrhages had occurred, requiring transfusion on each occasion. Cauterization of a bleeding point in the right nostril was of only transitory benefit. During this six week period, biweekly platelet counts by Dr. Weisfuse showed variations between 9,000 and 300,000. Weekly examination of the blood smears showed the platelets to be large and bizarre. The leukocyte count varied from 2,000 to 5,875 with 34 to 86 per cent granulocytes. On two occasions, the bleeding time was 4 and 7 minutes respectively, clot retraction was poor and the tourniquet test was doubtful.

The laboratory findings on admission were: hemoglobin 8.3 grams; R.B.C. 2,300 M; W.B.C. 3900; polymorphonuclears 52 per cent, lymphocytes 19 per cent, monocytes 14 per cent, eosinophiles per cent. One normoblast was seen in counting 100 white cells. The platelets numbered 180,000 per cu. mm. The stained blood film showed an extreme abnormality of the platelet morphology which was present in 95 per cent to 99 per cent of all the platelets. They were unusually large and varied in size between one-third to the full size of a red cell (7.5 micra). They were uniformly round to oval with smooth outlines, took only a very pale blue stain and were remarkable for their almost complete lack of granularity (fig. 1, A). They occurred singly, with only an occasional group of two being seen. The remaining 1-4 per cent of the platelets were normal in appearance but they, too, occurred singly. Sternal puncture showed...

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* We are grateful to Dr. Louis H. Weisfuse, Brooklyn, New York for referring this patient.
Fig. 1.—The blood smear in thrombopathic thrombocytopenia

(A) Before transfusions. Note the uniformly abnormal appearance of the platelets (P) and their failure to agglutinate.

(B) Immediately after transfusion of polycythemic blood by means of silicone coated syringes. Note the presence of normal platelets and their tendency to clump around the patient's abnormal platelets (P).

These two photomicrographs were taken with suitable filters to bring out the very pale-staining abnormal platelets in as dark a contrast as possible. The difference in the staining characteristics between the pathologic and the normal platelets is not altered, however.
normal or slightly increased numbers of megakaryocytes. The cytoplasms of these giant cells were agranular and took only a very faint blue stain (fig. 2). Large platelets, as described above in the blood, were at times seen to be clinging to these cells and to lie in the lacunae of their cytoplasm. The identical appearance of the megakaryocytic cytoplasm with that of the platelets was striking. Otherwise, except

Fig. 1.—The megakaryocytes in thrombopathic thrombocytopenia
(A) Low power microscopic field. Megakaryocytes are numerous; they show a "fragile" amorphous appearance and a lack of granularity.
(B) Megakaryocyte at high power. Note lack of granularity in the cytoplasmic borders and "buds."
(C) Two agranular megakaryocytes.
(D) Promegakaryocyte. There is normal granulation in a crescentic area near the nucleus but the remainder of the cytoplasm lacks granules.

for a moderate degree of normoblastic hyperplasia in keeping with the hemorrhagic anemia, the appearance of the marrow was normal.

Clotting time in glass (Lee-White method) was 12 minutes in the first tube and 15 minutes in the second and third tubes. Clotting time in lusteroid, performed in the same way, was 19 minutes in the first tube, 24 minutes in the second, and 30 minutes in the third. Prothrombin time (Quick) was 13
seconds. A prothrombin consumption test* showed a prothrombin time of serum of 14 seconds one hour after clotting. Clot retraction was rapid and complete. No significance was attached to the occasional appearance of a heavy sediment of red cells at the bottom of the test tube. Bleeding time (Duke) was greater than 10 minutes, at which time bleeding was stopped by applying pressure. A tourniquet test was negative.

Further Laboratory Studies: Patient's blood was type O, Rh positive. Urine showed no red cells; stools were guaiac negative. A tuberculin skin test was negative. X-ray films of the chest revealed no abnormalities.

Course

The patient was asymptomatic until the night of the fifth hospital day when he suddenly developed a severe hemorrhage from the right nostril and vomited swallowed blood. The pulse became rapid and the blood pressure dropped sharply. Examination of the nose at this time revealed brisk bleeding from

* This and the other laboratory methods are described in the Appendix, "Methods."
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the entire free border of the right inferior turbinate. Packing of the nose controlled the bleeding and the patient was given 500 cc. of bank blood. No further bleeding was noted during the remainder of the patient’s hospital stay.

On the morning of the sixth hospital day the patient’s red cell count was 2.04 M; hemoglobin 7.4 grams; platelet count 158,000 per cu. mm. Despite the transfusion given only a few hours previously, the blood smear showed no evidence of transfused platelets and 95.5 per cent of the platelets were of the large, abnormal type. During the afternoon of the sixth hospital day, a transfusion of compatible citrated blood from a normal donor, drawn one hour before transfusion, was given. After 250 cc. of this blood had been given, the bleeding time was still more than ten minutes. Blood drawn at this time showed a prothrombin consumption curve entirely similar to those obtained prior to the transfusion. The remaining 250 cc. of this transfusion were then allowed to run in. On the next morning, 11 hours after the transfusion had been completed, the platelet count was 149,000. At this time, examination of the stained blood smear revealed that 29.3 per cent of the platelets were small and that one-quarter of these occurred in groups of two or more. The red cell count at this time was 3.72 M; hemoglobin was 7.4 grams. On the morning of the eighth day, a direct transfusion of 300 cc. of polycythemic blood was performed, using silicone coated syringes and needles as described in the Appendix ("Methods"). Immediately after transfusion, there was no undue bleeding from the site of the venipunctures for the first time since the patient had come under observation. The platelet count was now 230,600. A blood smear showed the majority of the platelets to be small, of normal appearance, and occurring in clumps (fig. 1,B). Prothrombin consumption was now entirely normal and the bleeding time was 7 minutes. The various hematologic data collected during the patient’s hospital stay are presented in table 1.

During the first five days after the last transfusion, stained smears showed decreasing numbers of agglutinated small platelets (table 2) and eventually almost complete disappearance of all small platelets. During the same time the platelet count and the bleeding time gradually returned to their pretransfusion levels. The prothrombin consumption again became almost zero (fig. 3).

DISCUSSION

The Nature of the Bleeding Tendency

The bleeding diathesis in this case was apparently not due to an abnormality of the known humoral clotting factors, as is evidenced from the normal clotting and prothrombin time. Since the hemorrhagic tendency was abolished temporarily following a direct blood transfusion, it is evident that it was not due to an abnormality of the vascular factors.

The reduction in the number of platelets together with their abnormal morphology suggested that all the hemorrhagic phenomena were due to an abnormality of the platelets, both quantitative and qualitative. The name thrombopathic thrombocytopenia has been suggested for this condition.1

Thrombocytopenia may be due to various mechanisms which may be classified as follows2: (1) disease of the bone marrow—aplasia, hypoplasia, infiltration by abnormal cells or tissues, etc.; (2) the hypersplenism of such conditions as cirrhosis of the liver, Felty’s syndrome, Boeck’s sarcoid, Gaucher’s disease, etc.; (3) idiopathic. The last named group differs from the other two in the presence of at least normal numbers of megakaryocytes in the bone marrow and in the absence of a disease known to cause splenomegaly. Because both these criteria were present in our patient, the diagnosis of idiopathic thrombocytopenic purpura could be made. However, a number of features were present which indicated at least a variant of the typical form of the disease. These were as follows:

1. A more marked bleeding tendency: Severe, exsanguinating hemorrhages are almost never seen in patients with idiopathic thrombocytopenic purpura whose platelet
count is over 100,000 (one-fifth normal), although easy bruising and moderate menorrhagia may be present. Similarly, the bleeding time, although usually prolonged, is almost never longer than 10 minutes at such platelet levels. In our patient, however, severe hemorrhages and a prolongation of the bleeding time over 10 minutes were present with platelet levels between 150,000 and 280,000.

2. A marked diminution in prothrombin consumption: In a series of cases with thrombocytopenia we were able to confirm Quick's findings that the degree and to some extent the rate of prothrombin consumption were proportional to the platelet count provided the other coagulation factors were normal. In our series this relationship held true irrespective of whether the thrombocytopenia was idiopathic or secondary to leukemia.*

On the other hand, our patient with thrombopathic thrombocytopenia showed a much slower and much more diminished prothrombin consumption than would be expected from the relatively slight reduction in platelet count which was present (fig. 4).

3. Platelet morphology: While marked variations in the size of the platelets together with large bizarre forms and irregular arrangement and staining characteristics of the granules are the rule in idiopathic thrombocytopenic purpura at low platelet levels, platelets of normal appearance predominate at levels of over 100,000. In our patient the great majority of the platelets showed marked morphologic abnormalities even though the platelet count was always above 150,000. Moreover, although considerable anisocytosis of the platelets was present in our case, they were almost all extraordinarily large and differed from the platelets in typical cases of idiopathic thrombocytopenic purpura by their lack of granules and weak staining (fig. 1, A).

4. The morphology of the megakaryocytes: In idiopathic thrombocytopenic purpura,

* It was found, however, only in patients whose platelet count was fairly constant from day to day. Hence, only such patients were included in the graphs.
the megakaryocytes show no significant histologic deviation from the normal except for a great diminution of platelet production from the cytoplasmic borders. Granularity of the megakaryocytic cytoplasm is within normal limits. In our case, the abnormal morphology of the platelets was reflected by similar histologic abnormalities of the megakaryocytes. These cells were almost entirely devoid of granules and their cytoplasms stained very weakly, giving them a hyaline appearance identical with that of the platelets (fig. 2). This parallelism in the histologic abnormalities provides further evidence for the now well established view that the platelets are derived from the megakaryocytes. It also suggests that in thrombopathic thrombocytopenia the megakaryocytes are not merely affected in their ability to produce platelets, as is the case in idiopathic thrombocytopenic purpura, but that they are intrinsically defective. This abnormality of the megakaryocytes may thus be considered as a fundamental feature distinguishing thrombopathic thrombocytopenia as a special type of idiopathic thrombocytopenic purpura.

Since the megakaryocytes in the thrombopathic form are agranular and give rise to similarly agranular platelets, it is probable that the diminished physiologic effectiveness of the platelets (lack of agglutination, almost complete inability to cause prothrombin consumption and failure to prevent spontaneous bleeding) is associated with this morphologic defect. Whether the factors involved in the abnormality of the megakaryocytes are identical with those causing the thrombocytopenia cannot be answered at present.

Megakaryocytes with deficient granulopoiesis have recently been found by Schwarz in a case of idiopathic thrombocytopenic purpura. Similar observations in the past are listed in the same report.

Cases in which a bleeding tendency was apparently due to a physiologic defectiveness of the platelets have been reported by Glanzmann and Fonio. In some of Glanzmann's cases and in the case described by Fonio, morphologic abnormalities of the platelets are described which are strikingly similar to those observed in our case. In Glanzmann's cases, however, the bleeding time was normal while the clot retraction was absent or incomplete. In Fonio's case, too, the bleeding time was normal but the clotting time was prolonged.

Transfusion Data: Platelet Survival Studies

In 1910, Duke reported that by direct transfusion of whole unmodified blood he had been able to cause temporary cessation of spontaneous bleeding in 3 patients with idiopathic thrombocytopenic purpura. This was accompanied by a rise in the platelet count, lasting about two days, in the 2 patients in whom such counts were performed. In 1947, Lawrence and Valentine showed that by means of a carotid-to-carotid anastomosis it was possible to transfuse platelets from a healthy cat to one made thrombocytopenic by x-ray radiation over the bone marrow. By following the platelet counts of their animals after they had been returned to their own circulation they found that the transfused platelets survived for about three days. Subsequently, however, a report by the same author revealed that massive transfusion of unmodified whole blood by the multiple syringe or the Pennell technic did not result in a rise of the platelet count in two throm-
bocytopenic patients. Similarly, Krasso in 1927 did not find an immediate rise in platelet count following massive direct transfusions.

In our patient, the transfusion of 500 cc. of compatible citrated bank blood (one week old) from a normal individual had no effect on the platelet count and the bleeding time. Examination of the stained blood smear failed to reveal the presence of transfused platelets (table 1). This was due presumably to destruction of the platelets during storage of the blood. Transfusion of 250 cc. of fresh citrated blood from a normal donor (fig. 3 and table 1) also had no effect on either the bleeding time or prothrombin consumption. Further transfusion of another 250 cc. of fresh citrated blood, while it had no definite effect on the platelet count, produced a slight though definite rise in the percentage of normal small platelets as noted in the stained smear. There was no change in the bleeding time and only a very slight rise in the prothrombin consumption. It was thus evident that the transfer of a small number of platelets could be accomplished by a relatively large transfusion of fresh citrated blood. It is of interest that in this instance the transfused platelets would have escaped detection had we relied solely on the platelet count. The relatively small number of normal platelets was undoubtedly responsible for the lack of any concomitant changes in the bleeding time and the very slight effect on the prothrombin consumption.

Transfusions of fresh, citrated blood from polycythemic donors with high plate-
let counts to patients with aplastic anemia and idiopathic thrombocytopenic purpura have been employed since 1929 by one of us (W. D.) as a means of raising the platelet level. In several patients on whom studies were performed a definite rise in the platelet count was noted after these transfusions. In the present case, blood from a polycythemic donor with a very high platelet count was transfused into the patient without the use of anticoagulants and by means of the multiple syringe technic, syringes and needles having been coated previously with silicone. It was hoped that the use of silicone and the absence of an anticoagulant might result in the greatest possible number of intact platelets reaching the recipient.

This transfusion had as its most striking clinical effect the immediate cessation of all excessive bleeding following venipunctures and skin punctures incidental to the taking of blood samples. Since the patient was not bleeding spontaneously at the time, the effect of the silicone transfusion on spontaneous hemorrhage could not be determined. Whereas before the transfusion, the bleeding time had always been longer than 10 minutes and pressure was always required to stop bleeding, the bleeding time immediately after transfusion was 7 minutes. Three hours later, it was 5 minutes and did not return to the pretransfusion level until the fifth day after the silicone transfusion. The platelet count, which had ranged between 140,000 and 150,000 before the transfusion, rose to 230,000 immediately afterwards and returned to its basal level between forty-eight and ninety-six hours later. This rise, although of low magnitude, is significant even if one assumes a possible error in the platelet count of ±15 per cent. (The error with this method as worked out by Dameshek was ±7 per cent). More striking and more convincing as evidence of successful platelet transfusion was the sudden increase in the percentage of normal appearing platelets on the stained smear from 29.5 per cent before the transfusion, to 73 per cent immediately afterwards (fig. 1, B). While before the transfusion even the small platelets appeared almost always singly, they were now found in clumps. The percentage of small platelets with normal granularity gradually decreased, and fewer and fewer were found to be agglutinated, until on the sixth post-transfusion day only 5.5 per cent of all platelets were of the small type and were almost all unagglutinated. These data are summarized in table 1 and figure 3. As long as transfused platelets were present they tended to agglutinate (table 2).

Prothrombin consumption, which had been almost nil before the silicone syringe blood transfusion, became normal immediately afterwards and returned to its original level after the third or fourth day (fig. 5).

In our case, 300 cc. of polycythemic blood having a platelet count of 4 million per cu. mm. (8 times normal) were transfused into the patient whose blood volume estimated on the basis of his body weight (26 Kg.) was 2.5 liters. The number of

* Unfortunately, the silicone transfusion was not given at the basal level; as a consequence of the previous transfusion with fresh citrated blood, the percentage of small platelets had already risen to 29.5 per cent before the silicone transfusion. Before the citrate transfusion, the percentage of small platelets was never over 5 per cent and for this reason, 5 per cent normal platelets is considered to be the patient's basal level.
Transfusion of 500 cc. of fresh citrated blood was given on the sixth hospital day.
Transfusion of 300 cc. of fresh, unmodified blood from polycythemic donor was given on morning of eighth hospital day.
* 81, Immediately after transfusion; 82, three hours after transfusion; 83, six hours after transfusion.

platelets available in the transfused blood was thus theoretically sufficient to raise the patient’s platelet count from a pretransfusion level of 150,000 to 650,000. The actual level observed was only 230,000, a rise of 80,000 instead of the calculated

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Fig. 5.—Thrombopathic thrombocytopenia; prothrombin consumption. (1) Before transfusion; (2) after 250 cc. fresh blood (normal donor); (3) after transfusion of polycythemic blood.
500,000. If one calculates the number of normal platelets present before and after the silicone transfusion (platelet count X percentage of normal platelets as seen on the stained smear) one finds a rise from about 50,000 to only 150,000, i.e., approximately one-fifth the expected rise of 500,000.

Destruction of the platelets or their "loss" during the transfusion itself must be considered as the most obvious cause of the low recovery of transfused platelets in the patient. Increase in the patient's blood volume as the result of rapid and relatively large transfusion may be another, although minor factor. The possibility of the transfused platelets having been trapped in a reservoir such as the lungs cannot be excluded. The prothrombin consumption immediately after the silicone transfusion appeared to be greater in degree than that which could be expected from the number of transfused normal platelets. It is possible, therefore, that the majority of the transfused platelets was used to satisfy the great backlog of a need for normal platelets for vascular hemostasis. The return of the prothrombin consumption to levels in keeping with the number of normal platelets within three hours after the silicone transfusion may offer some evidence in support of this view.

Another factor which may be responsible for the small rise in the platelet count immediately after the transfusion of polycythemic blood is a possible destruction of the patient's own platelets. Their number dropped from approximately 100,000 per cu. mm. before the transfusion to 50,000 per cu. mm. three hours after the transfusion. As shown in figure 1, B, these large platelets, which before transfusion had occurred singly, often appeared to be agglutinated in groups of two or more after transfusion. One may then speculate that the introduced normal platelets brought about agglutination and possible disintegration of the large previously inert platelets.

Although only a fraction of the transfused normal platelets could be recovered intact in the recipient, our data indicated that these platelets had a life span of from five to six days. As long as they were present in significant numbers, they also appeared to retain their physiologic function as shown by their ability to agglutinate and their effect on the bleeding time and the prothrombin consumption.

The life span of the mammalian blood platelets has previously been estimated to be between three and eight days by several workers using a variety of technics. In our experiment, the life span of normal platelets when transfused into our patient was found to be well in the upper range of these values. Thus, no evidence was found of a phagocytic factor as the cause of the continued thrombocytopenia in this splenectomized case of chronic idiopathic "thrombopathic" thrombocytopenia.

**Summary**

1. A case of thrombopathic thrombocytopenia, a variant of chronic idiopathic thrombocytopenia is described.
2. In addition to the customary studies, the prothrombin consumption test of Quick was used in a study of the physiology of the platelets.
3. The patient's platelets were not only reduced in number but were morphologically abnormal and physiologically defective. Their abnormality was presumably related to the lack of granularity in the cytoplasm of the platelet-producing cells, namely, the megakaryocytes.

4. Because of the uniformly abnormal appearance of the patient's platelets, the success of a transfusion of normal platelets could be readily evaluated from inspection of stained blood smears.

5. Transfusion of blood from a polycythemic donor whose platelet count was about eight times the normal level resulted in the recovery of a detectable proportion of these platelets in the patient. Possible mechanisms leading to their relatively low recovery are discussed.

6. The donor platelets survived for five to six days and retained their normal physiologic activity throughout that time.

APPENDIX: METHODS

Platelet Counts: The indirect (wet smear) technic of Dameshek13 was used. The normal range by this method is 400,000 to 800,000 platelets per cu. mm.

Differential Platelet Counts: One of us (J. F. -G.) counted 400 platelets in an evenly spread portion of the stained blood film. The number of small and abnormally large platelets encountered was noted, together with the number of agglutinated and unagglutinated platelets in the first 100 platelets encountered.

Prothrombin Consumption Test: Squibb's thromboplastin and thromboplastin-extracting fluid was used. After adding 1 cc. of the extracting fluid containing calcium to the thromboplastin, the mixture was allowed to stand in a waterbath at 37 C. for at least thirty minutes before it was used for tests. Prothrombin-free plasma was prepared by incubating the patient's oxalated plasma with barium sulfate (50 mg. for each cc.) at 37 C. for ten minutes. The mixture was then centrifuged, the supernatant poured off, and the prothrombin-free plasma thus obtained, allowed to stand at room temperature until used, but never longer than eight hours. The absence of prothrombin was always checked by adding thromboplastin to the plasma and noting the lack of clotting after three minutes' incubation. Similarly, the presence of fibrinogen was checked by adding .01 cc. of a solution containing 1000 units of thrombin per cc. and noticing if a clot appeared immediately. This was done whenever the prothrombin time of serum was markedly prolonged. To check the activity of the thromboplastin, frequent prothrombin determinations were done on the patient's plasma. The test itself was performed as follows: Blood was drawn from a vein and about 3 cc. were placed in each of two test tubes. These were immediately placed in a waterbath at 37 C. Thirteen minutes after a firm clot had formed and in some experiments at various intervals, the clot in one of the tubes was loosened by passing a wooden applicator stick once around the inside wall of the tube. The tube was then centrifuged for one minute at high speed and again placed in the waterbath where it was allowed to remain for the rest of the experiment. One-tenth cc. of serum from this tube was then added to 0.2 cc. of thromboplastin and 0.1 cc. of prothrombin-free plasma. The mixture was not removed from the waterbath until about 3 to 10 seconds before clotting was expected. The appearance of the first fibrin strands was taken as the end point. The blood in the second tube was left undisturbed and used for determining clot retraction.

Silicone Technic: Two carefully cleaned and dried 100 cc. glass syringes and two No. 18 gage needles were coated with silicone. A small amount of silicone solution (General Electric Dri-Film 9987) was poured into the barrels and onto the pistons of the syringes. By gentle rotation the surfaces were entirely covered with the material. The pistons were then run back and forth in the barrels to insure complete coating. Needles were coated by attaching them to a syringe. They were then immersed into the silicone solution while a small amount of the material was being drawn through them. After the application of the silicone each piece of equipment was rinsed twenty times with tap water, allowed to soak in weak ammonia solution for at least one hour and again rinsed twenty times with water. Syringes and needles were then dried by hot air and the needles autoclaved. Before autoclaving, the syringes were wiped with gauze to remove excess silicone and then again rinsed with water to remove lint and dust.
Direct Transfusion Technique: The patient was prepared by the administration of $\frac{1}{2}$ grain secobarbital two hours before the procedure and morphine sulfate $\frac{1}{2}$ grain and scopolamine $\frac{1}{2}$ grain one hour before the procedure. He was somewhat drowsy but able to cooperate. The patient and a carefully cross matched type O, Rh positive, Hinton negative polycyhemic donor with a platelet count of approximately 4.0 million per cu. mm. were placed in adjoining cubicles. The skin overlying the antecubital veins was cleaned with alcohol and the silicone coated needles inserted in the veins of each. One worker withdrew 100 cc. of blood from the donor into a silicone coated 100 cc. syringe. This syringe was then attached to the needle already placed in the patient’s vein and the blood slowly injected while another 100 cc. of blood was being drawn from the donor. The maneuver was repeated until the patient had received 300 cc. of whole unmodified blood. The whole procedure took about 40 minutes and at no time did the syringes become jammed and fibrin formation was not noted. On returning to the ward after the procedure the patient slept for two hours and afterwards had no recollection of the procedure. There were no untoward reactions of any kind.

Immediately upon completion of the transfusion, blood for prothrombin test was drawn from the patient’s other arm and cutaneous blood for the other studies. Further tests were done three and six hours after the transfusion and then at daily intervals.

Indirect Transfusions: Disposable “plastic” tubing and adapters were used both for drawing blood from the donor and for transfusing blood into the patient. Blood was drawn into 100 cc. Transfuso-Vac bottles and transfused into the patient directly from these.

REFERENCES

THROMBOPATHIC THROMBOCYTOPENIA: SUCCESSFUL TRANSFUSION OF BLOOD PLATELETS

ERWIN O. HIRSCH, JEAN FAVRE-GILLY and WILLIAM DAMESHEK