Liability for Blood Transfusion Injuries

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LIABILITY FOR BLOOD TRANSFUSION INJURIES

INTRODUCTION

The development of the modern blood transfusion in the past half century is recognized by the medical profession as one of its finest achievements. Without today's blood transfusion many of the modern surgical practices would not be possible, and hemorrhage would be a far greater cause of death.

Although the transfusion of blood or pooled blood plasma is now a routine therapeutic procedure, injuries may still result. This Note is designed to (1) describe the two major types of transfusion injuries, that is, reaction due to transfusion of incompatible blood, and transmission of disease through a blood or plasma transfusion; and (2) discuss the legal liability for these injuries of those persons manufacturing or supplying the blood or plasma as well as those persons administering the transfusion.

I. TRANSFUSION OF INCOMPATIBLE BLOOD

A. Blood and Blood Groups

While almost everyone knows the general attributes of blood, few people know of blood's detailed constituency or bodily function. Blakiston's Medical Dictionary describes blood as:

The fluid tissue which circulates through the heart, arteries, capillaries, and veins, supplies oxygen and food to the other tissues of the body, and removes from them carbon dioxide and waste products of metabolism. It is made up of plasma and cellular elements. The latter consists of erythrocytes [red blood cells], leukocytes [white blood cells], and blood platelets. Plasma of the blood from which fibrinogen has been removed is called serum.

The red cells of the blood contains antigens; the serum of the

1. Prior to the turn of this century, little was known about blood groups or proper transfusion procedures. See Gradwohl, Legal Medicine 524 (1954) (hereinafter cited as Gradwohl); Kabat, Blood Group Substances 2 (1956) (hereinafter cited as Kabat).


4. Fibrinogen is a soluble protein in the blood plasma, which by the action of thrombin is converted into fibrin, thus producing clotting of the blood. Dorland, Medical Dictionary 551 (21st ed. 1948) (hereinafter cited as Dorland).

5. See Dorland 1132.

6. An antigen is any substance which stimulates the production of antibodies or reacts with them. Blakiston 88.
blood contains antibodies. Several antigens may commonly group together in red blood cells; one such specific group of antigens contained in blood is called an agglutinogen. Similarly, specific antibodies may occur together in serums; one such group of antibodies found in the serum of the blood is called an agglutinin. In 1900, Karl Landsteiner discovered two distinct agglutinogens and that all human blood contain one, both or neither of these agglutinogens in the red blood cells. He labeled the agglutinogens as A and B. Landsteiner also discovered that a person having one agglutinogen always has the reciprocal agglutinin. Thus, an individual having the agglutinogen A in his red blood cells has the agglutinin anti-B in his serum; and an individual with the agglutinogen B in his red blood cells has the agglutinin anti-A in his serum. An individual classified as group O has neither the agglutinogen A nor B, but has both the agglutinins anti-A and anti-B. The group AB was recognized later by Decastello and Sturli. The blood of an individual of group AB possesses the opposite of group O, that is, both the agglutinogens A and B in the red blood cells, but neither the agglutinin anti-A nor anti-B in his serum.

The problem of blood incompatibility arises, in essence, when a particular agglutinogen is combined with the improper agglutinin. For example, if blood containing the agglutinogen A is mixed with blood containing the agglutinin anti-A, the process of agglutination, or clumping, of the red blood cells would occur. This process often causes serious reaction.

The blood factor, or agglutinogen, Rh is also of extreme importance. Landsteiner and Wiener in 1937 discovered that the red blood cells of approximately 85 per cent of the population contained

7. An antibody is a substance, either natural or induced by exposure to an antigen, which has the capacity to react as agglutinins, lysins, precepts, etc., with the specific or related antigens. Blakiston 87.

8. An agglutinogen is an antigen which stimulates the formation of a specific agglutinin. This, in turn, has the capacity to agglutinate the antigen. Blakiston 36.

9. An agglutinin is an antibody in the serum which, when added to a suspension of its homologous, particulate antigen, causes the antigen elements to adhere to one another, forming clumps. Blakiston 36.


11. See Gradwohl 525; Kabat 3.

12. An agglutinogen is not technically the same as a blood factor. See Wiener, The Rh-Hr Blood Types, 2 J. For. Med. 224, 226 (1955); Wiener, Owen, Stormont, and Wexler, Medicolegal Applications of Blood Grouping Tests, 3 J. For. Med. 98, 99 (1956). For purposes of this Note, however, the terms will be used interchangeably.

an element that reacted to an antibody created in the serum of rabbits injected with blood from a rhesus monkey. An individual whose blood would react to this serum (called anti-Rh serum) would possess the agglutinogen Rh in his red blood cells, and was said to be Rh positive. An individual whose red blood cells were not agglutinated by anti-Rh serum was said to be Rh negative, that is, lacking the Rh factor. This discovery led to the abolition of the greatest source of post-transfusion injury since Landsteiner illustrated the A-B-O relationships. The original Rh agglutinogen was later labeled Rh (or D).

It is well established that there are subgroups in both the A-B-O system and the Rh system. It was recognized that not all red blood cells of group A individuals, when tested with an anti-A serum, were equally strongly agglutinated. Those A red blood cells that agglutinated most strongly were called A1, and those agglutinating less strongly were called A2. A still weaker and rarer variety of A was labeled A3, and an extremely weak agglutinating variety of A was called A4. The subgroups of A may be of importance in determining the AB group (where the individual has both the A and B agglutinogens). The AB group can be tested and typed as either A1B or as A2B. Because the subgroup A2 agglutinates weakly to the anti-A serum, the A2 agglutinogen in an A2B individual might be overlooked and the blood incorrectly typed as B. This A2B blood may cause a transfusion reaction if given to a recipient of pure B group.

Several subgroups of the Rh agglutinogen are also now well established. Most human red blood cells are strongly agglutinated by the anti-Rh (or anti-D) serum, and are thus considered Rh positive. It has been shown, however, that the remainder of human red blood cells are not necessarily Rh negative. Two other Rh factors which may be present are rh' (or C) and rh'' (or E). These are identified by tests with anti-rh' (C) and anti-rh'' (E) serum respectively. Both of these sera are now available commercially. In addition, there is an Rh variant, termed Rh, (or Dn), that must

15. See 3 Gray, Attorneys’ Textbook of Medicine ¶ 304.03 (3d ed. 1951) (hereinafter cited as Gray); Harley, supra note 10, at 6-7; Kabat 11-12.
16. Discovered when group A red blood cells were tested with an anti-A serum obtained from group O blood. See Kabat 7.
17. No subgroups of the agglutinogen B have as yet been found. See Harley, supra note 10, at 7.
18. See 3 Gray ¶ 304.03. There is another Rh factor, labeled rhw. See Wiener, Owen, Stormont, and Wexler, supra note 12, at 99.
19. See 3 Gray ¶ 304.11, at 3242.
also be tested for before an individual's blood can be identified as Rh negative.20

It has been recognized that persons who are Rh negative possess related factors, or agglutinogens, called Hr factors. These factors are genetically related to the three Rh agglutinogens, but the Rh-Hr factors, although related, behave as distinct agglutinogens.21 Three subgroups of the Hr factor are known and labeled, similar to the Rh system: Hr₀ (or d), hr' (or c), and hr'' (or e). These agglutinogens are tested for by the use of the serums anti-Hr₀, anti-hr', and anti-hr''.22 Injuries due to the incompatibility of the Hr factor are possible, and may result in the same manner as injuries due to Rh incompatibility.23

Another blood grouping system, independent of all others, is the M-N system. By injecting human red blood cells into rabbits, Landsteiner and Levine in 1927 were able to obtain two new antisera, each containing certain antibodies that would agglutinate the red blood cells of many humans—totally independent of that individual's A-B-O type. Landsteiner and Levine called the two new agglutinogens demonstrated by these anti-serums M and N, and the new agglutinins anti-M and anti-N.24 If both the agglutinogens M and N are present in the individual's red blood cells he is said to possess the agglutinogen MN. No case has been found of an individual lacking both of these agglutinogens.25 Related agglutinogens labeled S and s have also been recognized.26 These agglutinogens are seldom tested for prior to transfusions for they rarely cause transfusion reaction.27

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21. See 3 Gray § 304.25. The Rh-Hr factors are said to be allelomorph (or allele). That is, if a particular Rh factor, rh' (C) for example, is not found in the red blood cells, the corresponding Hr factor, hr' (c), would be found in that person's cells. See Gradwohl 547-48.
22. Anti-serums for testing the Hr factor and its subgroups, unfortunately, are difficult to obtain. 3 Gray § 304.25, at 3264-65. In fact, anti-Hr₀ serum is almost impossible to procure. Interview with Dr. Newell R. Ziegler, Director, University of Minnesota Hospital Blood Bank, Minneapolis, Minnesota, November, 1957.
23. Another blood factor related to the Rh-Hr system has been recognized—the f factor. See Kabat 13. Very recently another factor was recognized—the G factor, Reported at the Annual Am. Ass'n of Blood Banks, Chicago, Illinois, 1957.
25. See Gradwohl 526.
26. See Gradwohl 526-27. Since finding S, the M-N system has generally been referred to as the M-N-S system.
27. These M-N-S factors rarely cause reaction because they are of such a low titer. "Titer" is the amount of one substance which corresponds to, reacts with, or is otherwise equivalent to a stated quantity of another substance. Blakiston 1248. In this regard, it is the amount of antibodies in the serum. In addition, these factors can be detected by careful cross-matching tests.
There are several other established blood factors or blood group systems, all of which have been implicated as causes of transfusion reaction. Among these factors are K and k (Kell-Cellano factors), \( \text{Fy}^a \) (Duffy factor), \( \text{Le}^a \) (Lewis factor), \( \text{Jk}^a \) (Kidd factor), \( \text{Lu}^a \) (Lutheran factor), \( \text{P} \) and \( \text{Tj}^a \), and \( \text{U} \). The corresponding agglutinins of these agglutinogens are uncommon but when they do develop they cause a reaction when blood containing the incompatible agglutinogen of one of these types is transfused to the sensitized person. Transfusion reaction due to one of these factors generally occurs upon the second or third transfusion, or a transfusion following a pregnancy. The K (Kell) factor is believed to be one of the more dangerous of these agglutinogens but it is still not considered as important as the Rh factors.

B. Pooled Blood Plasma

Transfusions of pooled human blood plasma may be used as an alternative to whole blood transfusions. Plasma may be defined as:

The fluid portion of the blood in which the corpuscles [red and white blood cells] are suspended. Plasma is to be distinguished from serum which is plasma from which its fibrinogen has been separated in the process of clotting.

Pooled blood plasma, or normal human plasma, is defined as:

The sterile plasma obtained by pooling approximately equal amounts of the liquid portion of citrated whole blood from eight or more healthy humans. . . . Plasma is capable of serving all the purposes for which whole blood is employed, except reso-
ration of the hemoglobin, and has the advantages that human
plasmas are rarely incompatible with each other and that it can
be stored for long periods of time.34

The plasma of human blood contains only small amounts of
agglutinogens derived from broken up red cells. When the plasma
is “pooled” the divergent agglutinins are diluted and inhibited by
union with the small quantity of dissolved agglutinogens35 so that
pooled blood plasma can safely be administered to recipients of any
group.36 This makes plasma especially valuable in times of war or
domestic disaster. However, transfusions of pooled blood plasma
may be much more dangerous than whole blood, particularly in
respect to the transmission of homologous serum hepatitis.37

C. Specific Injuries

Hemolysis

The most apparent indication of a transfusion of incompatible
blood is hemolysis. Hemolysis, or a hemolytic reaction, is the de-
struction of red blood cells and the resultant escape of hemoglobin.38
The red blood cells of the incoming blood are destroyed by aggluti-
nation; that is, they clump together when they are mixed with the
foreign agglutinins of the recipient’s serum.39 When the red blood
cells of the donor clump together in the recipient’s system they are
destroyed.40 The hemolysis itself is not harmful but it is an indica-
tion of the damage to other tissues of the recipient. The most serious
of these is the damage to the kidney tissues.41 The damage to the
kidney tissues causes anuria. Anuria is the arrest of urinary out-
put.42 When the recipient is unable to release his own waste prod-
ucts death results. Even in the absence of anuria there may be some
damage done to the recipient’s kidneys.

Fortunately, the great majority of blood transfusion reactions

34. Blakiston 923.
35. See Wiener, Grant, Unger, and Workman, supra note 2, at 1438.
36. For a discussion on the preparation of blood plasma, see Hartman,
37. See discussion pp. 654-57 infra.
38. Blakiston 538.
39. A hemolytic reaction may result, although it rarely does, in the reverse
manner, by the destruction of the recipient’s red blood cells by the
agglutination caused by the incompatible agglutinins of the incoming serum
of the donor. See Davenport, supra note 20, at 335.
40. See Davenport, supra note 20, at 335; Kabat 4.
41. Interview with Dr. R. W. Koucky, Medical Executive, Minneapolis
War Memorial Blood Bank, Pathologist, Fairview Hospital, Minneapolis,
Minnesota, November, 1957.
42. See Blakiston 93; Kabat 4. Anuria, however, may be caused by an
agglutination not serious enough to amount to hemolysis. Interview with Dr.
Newell R. Ziegler, supra note 22.
are not severe; in fact, some transfusions of incompatible blood may cause no reaction whatsoever. This is due to the low titer\footnote{See note 27 \textit{supra}.} of the agglutinins in the recipient's serum. The few agglutinins that there are may be used up in the reaction with the foreign agglutinogens in the red blood cells of the incoming blood from the donor and none are left over to damage the kidney. However, such a seemingly uneventful transfusion may have caused the titer level of the recipient's blood to rise sharply, and if a second transfusion of incompatible blood were later administered, a severe hemolytic reaction could ensue.\footnote{See Wiener, Grant, Unger, and Workman, \textit{supra} note 2, at 1435.}

\textbf{Sensitization}

Transfusion injuries due to incompatibility of the Rh factor occur in a similar manner. However, hemolysis does not result from the first transfusion of Rh positive blood into an Rh negative recipient, for human blood does not naturally contain the anti-Rh antibodies necessary to cause agglutination. These anti-Rh agglutinins may be caused to form when Rh positive agglutinogens are first transfused into an Rh negative recipient. The recipient is then said to be \textit{Rh sensitized}.\footnote{An individual may become sensitized to five of the six Rh-Hr factors: Rh (D), rh\textasciitilde{} (C), rh\textasciitilde{} (E), hr\textasciitilde{} (c), hr\textasciitilde{} (e), but not to Hr\textasciitilde{} (d). Interview with Dr. R. W. Koucky, \textit{supra} note 41; 3 Gray \$ 304.14; Unger, \textit{supra} note 13, at 288-89. There are a few Rh negative individuals who, after being Rh sensitized, possess an "Rh blocking antibody" which prevents the sensitization from being exhibited. See 3 Gray \$ 304.09.} Upon a second transfusion of Rh positive blood\footnote{There is almost no danger in transfusing an Rh positive individual with Rh negative (as to all Rh factors) blood. See Wiener, Grant, Unger, and Workman, \textit{supra} note 2, at 1437. In emergencies, the hospitals use Group O-Rh negative blood and almost never have a reaction. Interview with Dr. R. W. Koucky, \textit{supra} note 41.} into the now sensitized recipient, severe hemolytic reaction and death could result.\footnote{See 3 Gray \$ 304.16; Wiener, Grant, Unger, and Workman, \textit{supra} note 2, at 1436.}

Of extreme importance is that sensitization to the Rh factor may also result from pregnancy. During the pregnancy of an Rh negative woman with an Rh positive fetus (the father would have to be Rh positive), a few Rh positive agglutinogens of the fetus may escape from the fetus to the mother and may cause the mother to develop anti-Rh agglutinins in her serum. The first transfusion of Rh positive blood to such a sensitized woman could cause a hemolytic reaction.\footnote{See 3 Gray \$ 304.14.} If the mother has been Rh sensitized, either by a
previous transfusion or by a previous pregnancy, the developed anti-Rh agglutinins in her serum will affect the red blood cells of the fetus, causing a destruction of the red blood cells of that fetus. This is termed *erythroblastosis fetalis*, and is fatal to the fetus in approximately 50 per cent of the cases in which it occurs.  

D. Legal Liability

Although there are very few reported legal actions arising out of death or injury due to blood transfusions, it is agreed by medical men that injury from such a cause is not uncommon.

*Mistyping and failure to type*

The problem of incorrectly testing for the Rh factor with the resultant death of a fetus by erythroblastosis fetalis arose recently in *Berg v. New York Soc'y for the Ruptured and Crippled*. The hospital's technician had unexplainedly mistyped the female patient's blood group as A-Rh positive; she was in fact group A-Rh negative. She was given two transfusions of Rh positive blood. Upon a subsequent pregnancy, the fetus being Rh positive, the anti-Rh agglutinins in the sensitized woman's serum caused erythroblastosis fetalis in the child. Relying on the doctrine of respondeat superior, the woman sued the hospital for the negligence of the technician.

Before reviewing the lower court's finding of negligence, the New York Court of Appeals was faced with what has been termed the "medical-administrative" issue. The New York courts had for years applied the rule that a hospital is liable for the negligent acts of its employees only if the act was "administrative"; but if the act could be classified as "medical," the hospital would be immune from liability. This rule had produced ridiculous technical dis-

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49. See 3 Gray ¶ 304.16-.24.

50. Most transfusion reactions are very mild, and the more serious reaction injuries are generally settled out of court. See Leading Article, *Blood Transfusions—Medicolegal Responsibilities*, 163 A.M.A.J. 283 (1957); Wiener, Grant, Unger, and Workman, *supra* note 2, at 1435.


52. The plaintiff's husband was "heterozygous"; that is, his blood contained both Rh positive and Rh negative factors. A "homozygous" individual is one who is either purely Rh positive or purely Rh negative. See 3 Gray ¶ 304.04.

tinctions, and has been condemned by legal writers. The Court of Appeals, in reversing the lower court, made a new inroad on the rule. The court held that the technician’s act was “medical,” but since the technician was not a “professional” person, the hospital should be responsible for her negligent act. Recently, however, the same court completely abolished the medical-administrative rule, stating that hospitals should bear the same burden as everyone else under ordinary rules of respondeat superior; that is, if the person committing the negligent act was its employee, and was acting within the scope of his employment, the hospital should be liable.

Minnesota has adopted the medical-administrative test for the liability of hospitals. The Minnesota court, however, has not yet become embroiled in the seemingly nonsensical distinctions drawn by the New York courts’ application of the rule. While a court’s handling of this rule may exempt hospitals from liability for certain acts of its employees, by categorizing those acts as “medical,” the result may be to place liability on the physicians and surgeons under whose control “medical” acts are normally performed. By this labeling process courts never squarely face the factual question of control. Therefore, it would seem wise for the Minnesota court to abandon the medical-administrative rule and adopt, as New York has, the ordinary rule of respondeat superior which does place the emphasis on the element of control.

The second issue in the Berg case was the determination of

56. The court said that one who’s job requires only four to six weeks training, as the technician in this case, could not be considered “professional.” 1 N.Y. at 502, 136 N.E.2d at 523 (1956).
58. See Swigerd v. City of Ortonville, 246 Minn. 339, 345, 75 N.W.2d 217, 222 (1956), where the court said:
   "We adopt the rule that a hospital is liable for the negligence of its nurses in performing mere administrative or clerical acts, which acts, though constituting a part of a patient’s prescribed medical treatment, do not require the application of the specialized technique or the understanding of a skilled physician or surgeon."
59. See, e.g., St. Paul-Mercury Indemnity Co. v. St. Joseph’s Hospital, 212 Minn. 558, 4 N.W.2d 637 (1942).
whether a failure to accurately test for the blood factor constituted negligence. The Court of Appeals sustained the trial court's finding that the error of incorrectly determining the plaintiff's Rh₀ factor was negligent. In evaluating whether or not the court was correct in sustaining this finding, some familiarity with testing for blood factors is necessary. However, the discussion will not be limited to the testing for Rh factors, nor will it be limited to testing by hospitals.

Hospitals and blood banks do not routinely test for all the blood factors. They do type both the donor's and recipient's blood for the A-B-O system and the Rh₀ factor. Because incompatibility due to the A-B-O system or the Rh₀ factor is responsible for almost all transfusion injuries, and since it is standard practice to test for these factors, proof of failure to do so should sustain a finding of negligence. In the Berg case the error was not in failure to test for the Rh₀ factor; instead, the technician inaccurately tested for Rh₀. Here, liability is not as clear as in failure to test. Weak anti-serum, used through no fault of the person testing, may result in inaccurate testing. However, in Berg, the error could not have been due to weak anti-serum, and therefore negligence seems clear.

If the donor's blood is Rh positive as to Rh₀, no further tests are made; but when the blood is negative as to Rh₀, there is some difference of professional opinion as to whether further tests with anti-rh' (C) and anti-rh'' (E) serums need be made. Most authorities suggest that if the blood is negative as to Rh₀, tests for

60. Interview with Dr. G. Albin Matson, Director, Minneapolis War Memorial Blood Bank, November, 1957. See Wiener, Owen, Stormont, and Wexler, supra note 12, at 103, 106.
61. See Wiener, Grant, Unger, and Workman, supra note 2, at 1435-36; Wolf, Preservation and Use of Blood Testing Serums, 24 Am. J. Clin. Path. 376 (1954). The anti-Rh₀ serum should contain an antibody to identify the Rh₀ variant D. Interview with Dr. R. W. Koucky, supra note 41.
62. There have been two other cases reported involving inaccuracy in testing. In National Homeopathic Hospital v. Phillips, 181 F.2d 293 (D.C. Cir. 1950), the hospital technician negligently reported the blood of the donor to be compatible with that of the patient. The transfusion of incompatible blood caused a fatal reaction to the patient. The court affirmed a judgment holding the hospital liable for the technician's negligence. In Gile v. Kennewick Pub. Hospital Dist., 48 Wash.2d 774, 296 P.2d 662 (1956), the technician incorrectly typed the patient's blood, thereby causing her death. The court dismissed the action since the negligence action was barred by an exemption statute for public hospital districts, and there was no breach of warranty for there was no "sale" of blood.
63. The hospital, however, may detect weak anti-serum by use of proper controls on serum activity. Interview with Dr. Newell R. Ziegler, supra note 22.
64. Such defective anti-serum could result in Rh₀ positive blood being typed as Rh₀ negative, but would never result in Rh negative blood being typed as Rh₀ positive.
65. See Wolf, supra note 61, at 376.
both rh' and rh" are imperative. An individual can become sensitized to rh' or rh" just as he may be sensitized to Rh, and equally serious injury can result from all three factors. Due care would appear to require that any donor's blood found negative as to Rh, (D) should be tested with anti-rh' (C) and anti-rh" (E) serums. Only upon a negative finding for all these tests, including the test for the Rh, variant D, should a donor's blood be classified as Rh negative. As to the recipient's blood, since it is not standard practice to type for rh' or rh", failure to do so should not indicate lack of due care. These factors in recipient's blood are normally exhibited in the hospital's cross-matching tests.

Transfusion injury may result from sensitization to two of the three Hr factors; hr' (c) and hr" (e). At least one medical writer has stated that blood transfusions cannot safely be given without first testing for Hr incompatibility. However, because it is known that a donor's blood that is negative for all the Rh factors is necessarily positive for all the Hr factors, hospitals and blood banks do not routinely test for the Hr factors. However, blood found to be Rh positive as to Rh, (D) may be negative as to rh' (C) or rh" (E), thereby being hr' (c) or hr" (e) positive. Thus, failure to test for the Hr factors, even when the blood is typed Rh positive because found to be positive as to Rh, (D), may cause a reaction to an Hr sensitized recipient when the incompatible Hr positive blood is transfused. Failure to test donor's blood for the Hr factors, nevertheless, would not appear to indicate a failure to exercise due care, for the anti-Hr serums are very difficult to obtain, the danger of sensitization is small, and lastly, the Hr factors are readily exhibited by proper cross-matching tests. In view of this reliance on

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66. See Unger, supra note 13, at 288-89; Wiener, Grant, Unger, and Workman, supra note 2, at 1436.

67. In the major cross-matching test the recipient's serum is mixed with the donor's cells; in the minor cross-matching test the donor's serum is mixed with the recipient's cell. If agglutination does not occur in either test the bloods are said to be compatible. Blakiston 295. There are a number of cross-matching tests; for example, the albumin test, the saline test, the high protein test, and the anti-globulin (Coombs') test. See Wiener, Nappi, and Gordon, Studies in Rh Sensitization, 8 Blood 1024, 1025-27 (1953). A new and highly sensitive cross-matching test, but which is still in the trial stages, is the papainizing test. Interview with Dr. R. W. Koucky, supra note 41.

68. Interview with Dr. R. W. Koucky, supra note 41.

69. See 3 Gray ¶ 304.25. Some hospitals do routinely test for hr' (c). E.g., University of Minnesota Hospital, Minneapolis, Minnesota. Interview with Dr. Newell R. Ziegler, Director.

70. See note 21 supra.

71. See note 22 supra.

72. Interview with Dr. R. W. Koucky, supra note 41.

73. Ibid.
cross-matching tests, a failure to use a cross-matching test that demonstrates Hr incompatibility may indicate lack of due care.

Severe hemolytic reaction may also result from incompatibility as to the M-N factors, the Kell factor, the Duffy factor, and others. These factors, however, are not routinely tested for in donor’s blood. Reaction due to incompatibility of one of these new factors (by the process of sensitization) is a very rare occurrence. To require that hospitals and blood banks test donor’s blood for each and every one of these newer and less common factors would create an overwhelming burden upon them. This would be particularly unreasonable in view of the fact that the hospital’s cross-matching tests will almost always detect such an incompatibility. Furthermore, since it is the judgment of almost all medical authorities concerned with blood that tests for the M-N factors, the Kell factor, the Duffy factor, and other new factors should not be made a part of the routine tests for the typing of a donor’s blood, courts should not sustain findings of negligence based on a failure to tests for these factors. Because of this reliance on the cross-matching tests, however, failure to use all the proper cross-matching tests might be a proper basis for a negligence action.

**Mislabeling**

In *Mississippi Baptist Hospital v. Holmes*, the hospital technician mixed up the decedent’s blood sample with the blood sample of another patient on the same floor. Thereupon, he accurately typed both samples but mislabeled each sample as a result of his original error. Because of this mislabeling, the decedent, while undergoing surgery, was transfused with incompatible blood and died. The Supreme Court of Mississippi, while overruling that state’s charitable immunity doctrine, held the hospital liable in damages for the negligence of its technician.

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74. See Wiener, Grant, Unger, and Workman, *supra* note 2, at 1437.
75. *Ibid.* See note 67 *supra*.
76. The University of Minnesota Hospital’s Blood Bank has adopted this preventive technique. They use elaborate and comprehensive cross-matching tests prior to every transfusion. Interview with Dr. Newell R. Ziegler, Director.
77. 214 Miss. 906, 55 So.2d 142 (1951).
78. The court did not state what the particular incompatibility was. The death certificate, however, showed that death was due to “acute hemolytic anemia, due to blood transfusions of wrong type.” 214 Miss. at 921, 55 So.2d at 147.
79. The change in the financial character of “charitable institutions” and the effect of insurance (which most “charities” carry today), have caused most courts to abandon the archaic charitable immunity doctrine. See, e.g., Ray v. Tucson Medical Center, 72 Ariz. 22, 230 P.2d 220 (1951); Wheat v. Idaho Falls Latter Day Saints Hospital, 78 Idaho 60, 297 P.2d 1041 (1956);
Apparently almost all legal scholars and experienced medical men would agree that a hospital's or blood bank's error in labeling a bottle of blood should constitute sufficient facts for a court to sustain a finding of negligence.80

Administration of blood transfusions

Another type of error arose in Necolayff v. Genesee Hospital.81 A nurse and an interne administered a blood transfusion to the patient notwithstanding her protests. This blood was, in fact, intended for another patient, and as a result the plaintiff suffered headaches, chills, high temperatures, and later was hospitalized at the state mental hospital for a period of time. Although the court's opinion makes no reference to such fact, the injury apparently was caused by the incompatibility of her blood to that of the transfused blood. The New York court, although still laboring under the medical-administrative dichotomy, held the hospital liable, reasoning that since this negligent act was in the nature of an assault or a trespass it could not have been a professional "medical" act.82

There are a multitude of additional errors that, when committed, may cause injury. For example, the serums used to type or cross-match the blood samples may be interchanged; patients with similar names may each be given blood intended for the other; or there may be an error in reading the labels accurately. These errors are all mechanical in nature, and proof of such errors, when shown to have caused injury to a patient, should be sufficient to sustain a finding of negligence.83

Res ipsa loquitur

It is possible that the doctrine of res ipsa loquitur84 should receive wider application in negligence actions based on injuries caused by blood transfusions.85 To date, in only one case concerning

82. 270 App. Div. at 653, 61 N.Y.S.2d at 836.
84. Res ipsa loquitur means, "the thing speaks for itself."
85. Although res ipsa loquitur has been applied in Minnesota in a malpractice action, Jones v. Tri-State Tel. & Tel. Co., 118 Minn. 217, 136
a blood transfusion has this doctrine been applied. In *Sherman v. Hartman*\(^\text{86}\) the needle injecting the blood into the patient’s arm slipped out of the vein and approximately 200 c.c.’s of blood\(^\text{87}\) entered the tissues of her arm. The California court ruled that the case should have gone to the jury under instructions of res ipsa loquitur.

Res ipsa loquitur may be particularly useful in the area of blood transfusion injuries since the injured patient is seldom in a position to discover the specific cause of the injury. As to the applicability of the doctrine, it would seem that the prerequisites for its use may be met in many cases arising out of a blood transfusion injury. By way of illustration, if a person with type A blood receives a transfusion of type B blood, a fairly strong argument may be made for the application of res ipsa loquitur. First, although expert medical testimony may be required, the plaintiff should be able to establish that type B blood is not normally given to a type A recipient in the absence of negligence. Secondly, if the action is against the hospital, it should be fairly easy to demonstrate that it was the negligence of some employee of the hospital which caused the injury. Even if the blood bank had mislabeled type B blood as type A, the error should have been caught in cross-matching tests. Again, if the donor’s blood was properly labeled, the hospital’s technician probably failed to make the routine A-B-O typing tests on the recipient’s blood and failed to make proper or any cross-matching tests. Lastly, it should not be difficult to establish that the plaintiff was free from contributory negligence. In any blood transfusion case the recipient hardly has an opportunity to be negligent.

**Emergency situations**

An obvious exception to the entire foregoing discussion on legal liability is that different requirements of due care must be applied in cases of emergency. The degree of the emergency probably should determine how far from normal standards the hospital

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Medical experts testified that it is a frequent occurrence for a needle (injecting the blood) to come out of the vein. They stated, however, that rarely does even 10 to 25 c.c.’s actually get into the body’s tissues. *Id.* at 594, 290 P.2d at 897.
or blood bank should be allowed to stray without being considered negligent. For example, a badly injured person is brought to the hospital in need of an immediate blood transfusion. Delay in getting the blood might cause death. The technician hurriedly types the patient's blood with the anti-A, the anti-B, and the anti-Rh₀ serums, and uses one of the fast but less sensitive cross-matching tests. If this cross-matching test fails to detect some unusual incompatibility, such as to the Kell factor, the hospital would clearly not be negligent for not having pursued more sensitive cross-matching tests. However, even in emergencies there must be a minimum standard, and patent errors in typing or labeling should possibly still be proper bases for a cause of action.

II. TRANSMISSION OF DISEASE

There are several diseases that one human can transmit to another through a transfusion of whole blood or pooled blood plasma. The viruses or organisms of such diseases may be carried in the donor's blood, and may pass with the blood to enter the blood stream of the recipient. Three diseases which may present this problem are homologous serum hepatitis, malaria, and syphilis. Of these three, the transmission of homologous serum hepatitis occurs most frequently and because the resultant illness is serious and sometimes fatal, this entire problem is an important one in the field of blood and plasma transfusions.

A. Homologous Serum Hepatitis

Hepatitis is the inflammation of the liver. Viral hepatitis is the inflammation of the liver due to a virus infection. Homologous serum hepatitis, or the type of hepatitis contracted by blood transfusion, is defined by Blakiston as:

A form of viral hepatitis transmitted by the parenteral injection of the human blood or blood products contaminated with the causative agent.

The terms "homologous serum jaundice" and "transfusion jaundice" are also used to describe this type of hepatitis.

The virus of homologous serum hepatitis cannot be detected in donors by any known medical test. Neither can the virus be detected
in the blood of the donor when taken or in pooled blood plasma. This dilemma has been a subject of medical concern for many years, and a number of persons have been injured as a result of the transmission of this hidden virus. Formerly the only precaution the blood banks and hospitals could take was to warn prospective donors of these dangers and try to eliminate those who might have been exposed to such a virus. For a time, the medical profession thought a process of irradiation could kill the hepatitis virus in pooled blood plasma. It was soon shown, however, that irradiation was unable to completely destroy the virus. While the possibility of transmission of homologous serum hepatitis in whole blood is quite slim, the possibility of transmission in pooled blood plasma is multiplied by the number of units making up the pool, for a virus from a single donor can contaminate the entire pool of blood plasma, rendering every transfusion from such a pool highly dangerous.

In *Parker v. State*, the patient was given a transfusion of pooled blood plasma which unknowingly contained the hepatitis virus, and as a result the patient died. The decedent's administrator brought an action for negligence against the State (as distributor of the plasma) on the ground that the State should have warned the physicians of the danger of the hepatitis virus. The court affirmed a dismissal of the action on the ground that it was reasonable for the State to expect that any authorized person using the plasma would know of the danger. Although the physician on whose orders the transfusion was given was not a party to the action, the court


94. Reports of transmission of hepatitis in plasma transfusions vary considerably, from 3% up to 12% of those receiving plasma transfusions. See Albrecht *et al*, *supra* note 92; Madsen, *supra* note 93; Murphy and Workman, *supra* note 92; Wiener, Grant, Unger and Workman, *supra* note 2, at 1438. An additional problem is that even persons who have never had hepatitis can possess the virus and transmit it through their blood; such persons are called "carriers." See Barnett, Fox, and Snively, *Hepatitis Following the Use of Irradiated Human Plasma*, 144 A.M.A.J. 226 (1950).

indicated that he would not be considered negligent since there was a need for a fast transfusion, approaching an emergency.

In *Hidy v. State*, the decedent had been in the hospital 15 hours prior to his operation; no blood typing tests had been made; and there were ample supplies of whole blood available for transfusion. The court again ruled that the State, as distributor of the plasma, was not negligent. The court stated that the only way the State could prevent this type of injury would be to recall all the pooled blood plasma, but this is highly undesirable because pooled blood plasma has many advantages notwithstanding its dangers. The court, however, clearly implied that the physician might have been negligent in making his choice to use plasma rather than whole blood especially in the absence of an emergency.

A second basis of liability was tried in *Merck & Co. v. Kidd*. The plaintiff, injured due to transmission of homologous serum hepatitis, argued that the sale of the plasma containing the hepatitis virus violated the Tennessee Food, Drug and Cosmetic Act, thus constituting negligence per se. That statute states that a drug is adulterated if it consists in whole or in part of any "filthy, putrid, or decomposed substance" and that such adulterated drugs are prohibited. The plaintiff argued that the hepatitis virus in the pooled blood plasma was a "filthy" substance making the plasma "adulterated." The court, after pointing out the medical impossibility of detecting or destroying this virus, held that the virus was not a "filthy" substance within the "intendment" of the statute. The lone dissenter, apparently more logic-minded than policy-minded, argued that whether or not the virus could be detected in the plasma was irrelevant.

A somewhat surprising aspect of the *Merck* case is that the court, in 1957, stated that there was still no scientific procedure available to detect or destroy the virus of homologous serum hepatitis. This, however, is not true, for it has been known for several years that the homologous serum hepatitis virus in pooled blood plasma can be totally destroyed by a process of storing the plasma at room temperature for six and perhaps as few as three months. The Minneapolis War Memorial Blood Bank has used this storage process and irradiation for seven years and has encountered not

\[97. 242 F.2d 592 (6th Cir. 1957), cert. denied, 78 Sup. Ct. 15 (1957).\]
\[99. 242 F.2d at 596.\]
\[100. Interview with Dr. G. Albin Matson, supra note 60. See Allen, Enerson, Barron, and Sykes, *Pooled Plasma with Little or No Risk of Homologous Serum Jaundice*, 154 A.M.A.J. 103 (1954).\]
one incidence of transmission of homologous serum hepatitis.\textsuperscript{101} Since the virus-killing action of storage has been so widely accepted, failure to utilize such a virus-destroying process resulting in transmission of the disease might become a proper basis for a negligence action. This process is now widely known among persons dealing with plasma preparations and is not a burdensome, expensive or impractical requirement to utilize.\textsuperscript{102}

Unfortunately, this virus-destroying process is not effective for whole blood. Since medical scientists have not yet discovered a method of detecting or destroying the virus in whole blood, and the blood banks and hospitals must still rely on the donor's medical history and truthfulness, it would seem that failure to prevent the transmission of homologous serum hepatitis in whole blood transfusions—\textit{if stringent donor requirements are set up}—certainly is no indication of negligence of either the blood bank or the hospital.

B. Malaria

A second disease that may be transmitted from a donor to a recipient through a blood transfusion is malaria. Malaria may not be transmitted through a pooled blood plasma transfusion, for the malaria organisms reside in the cells of the blood, and plasma contains no cellular components.\textsuperscript{103} In whole blood, the only known method of possibly detecting this organism is through a detailed and extensive microscopic examination. Because of the impracticality of testing each sample of blood, as well as the inconclusiveness of the findings, blood banks are again forced to rely on the veracity or the memory of the donor himself. Storage of whole blood for more than five days may destroy certain types of malaria organisms.\textsuperscript{104} Although medical journals report not infrequent cases of transmission of malaria,\textsuperscript{105} no reported legal actions based on contraction of this disease could be found. Liability based on the transmission of malaria should be limited to instances of actual negligence. In practice, liability would probably be limited to failure to ask questions regarding malaria.

C. Syphilis

The transmission of syphilis occurred occasionally in the past when blood was transfused immediately from donor to recipient.

101. Interview with Dr. R. W. Koucky, \textit{supra} note 41.
102. See Allen, Enerson, Barron, and Sykes, \textit{supra} note 100, at 107.
103. See Wiener, Grant, Unger, and Workman, \textit{supra} note 2, at 1438.
104. \textit{Ibid.}
It was subsequently found that chilling the whole blood would effectively destroy the syphilis agent; therefore, failure to chill the blood resulting in transmission of syphilis should, in the absence of an emergency, constitute negligence on the part of the supplier of that blood.

III. Warranty and Strict Liability

A. Warranty

Another basis used in attempting to impose liability on suppliers of whole blood or pooled blood plasma was attempted in Perlmutter v. Beth David Hospital, that of breach of an implied warranty. The transfusion in this case was of whole blood. As a result of the transfusion the plaintiff became afflicted with homologous serum hepatitis. The plaintiff argued that since her hospital bill included a separate item of $60 for "blood," the transaction constituted a "sale" within the Sale of Goods Act, thereby entitling her to actions on the implied warranties of "fitness" and "merchantability." The New York Court of Appeals, in a four-to-three decision, reversed a denial of the hospital's motion for a dismissal, ruling that the administration of the blood transfusion, even though specially itemized on her bill, was merely one incidental part of her medical treatment, that is, medical "services." The court again pointed out the lack of means of detecting or destroying the virus, and referred quite clearly to the undesirability of making hospitals insurers of the products they administer.

The effects of the Perlmutter decision, especially because of the closely divided court, have been far-reaching. The Legal Department of the American Medical Association has recently recom-

106. See Wiener, Grant, Unger, and Workman, supra note 2, at 1438; Trumbull, supra note 105, at 256.
107. The only reported case based on negligent transmission of syphilis is Giambrozi v. Peters, 127 Conn. 380, 16 A.2d 833 (1940), a malpractice action against a physician who failed to test the donor's blood for syphilis. The negligence action, however, was barred by a two-year statute of limitations.
110. This case was followed in Gile v. Kennewick Pub. Hospital Dist., 48 Wash.2d 774, 296 P.2d 662 (1956).
111. "The art of healing frequently calls for a balancing of risks and dangers to a patient. Consequently, if injury results from the course adopted, where no negligence or fault is present, liability should not be imposed upon the institution or agency actually seeking to save or otherwise assist the patient." 308 N.Y. at 107, 123 N.E.2d at 795.
mended that hospitals refrain from itemizing a specific charge for "blood," at least to eliminate the patent resemblance of the trans-
action to a "sale." The Department also suggested that hospitals
adopt a form of contract, or of consent, in which the patient would
be warned of the inherent dangers of both blood and plasma trans-
fusions, and contain a disclaimer of the warranty of fitness. In
Minneapolis, however, neither of these proposals have been
adopted. The reason that specific charges for blood have not been
discontinued concerns the policies and practices of procurement of
blood for transfusions. The charge of $60 in the Perlmutter case,
and $30 charge used in Minneapolis, is not the "cost" of that
blood, and the recipient is not, strictly speaking, buying the blood.
The fee is in the nature of a deposit. If the patient arranges to have
a friend or relative donate a unit of blood, that is, replace the unit
of blood given the patient, the "charge" is refunded. The "charge" is
deliberately made high in order to provide a substantial induce-
ment to replace the blood. If the blood banks and hospitals do not obtain
replacement of blood from these reliable (or non-indigent) patients
or their families, they will have to open their doors to the indigent
skid-row donor whose medical history may be extremely unreliable.
Thus, the blood banks and hospitals argue that their primary pur-
pose in using this "charge" for blood is the protection of the
community in general, and that the "charge" for the transfused
blood should not indicate a "sale" but a "service."

The Minneapolis community blood bank, the Minneapolis War
Memorial Blood Bank, formerly helped alleviate the crowded
hospital dilemma by providing a service within the bank itself of
administering blood and plasma transfusions, thereby making it
unnecessary for the recipient to occupy a hospital bed. As a result
of the close Perlmutter decision, the Blood Bank discontinued this
service for fear that such an isolated transfusion might be con-
sidered a "sale" and not an incident to medical "services."

Possibly an argument could be made that the blood banks that
supply the blood and plasma to the hospitals should be held for
breach of warranty because the replacement "charge" is partly
shared by them if the blood is not replaced, and that this should
constitute a "sale." Assuming that the patient could prove this

112. See Leading Article, Blood Transfusions—Medicolegal Responsi-
113. The Department provides suggested forms for both whole blood
and plasma transfusions. Id. at 286-87.
114. Interview with Dr. R. W. Koucky, supra note 41.
115. Interview with Dr. G. Albin Matson, supra note 60.
direct relationship resembling a sale, the blood banks may contend that they should have the same protection the hospitals have under the Perlmutter rule. The argument is that the collection of blood from a multitude of donors is a complicated task, and that it was recognized that it was much more desirable, from a community standpoint, to relegate this responsibility of collecting blood and making plasma to a central agency—the community blood bank. Therefore, the community blood bank is not an independent organization selling blood to the many hospitals; rather it is simply executing one of the multitudinous aspects of the hospitals' "services."

But arguments about "sale" or "service" seem to lead nowhere in this area. The real question involved seems to be one of policy. Further, the question appears to be the same whether one is considering the transfusion of "diseased" blood or "incompatible" blood—although transfusion of "incompatible" blood may only come under the warranty of "fitness for a particular purpose."

Basically, a court, faced with this warranty question, should attempt to determine the impact of what would in fact amount to strict liability on hospitals and blood banks. Since the ultimate supplier usually bears the burden in breach of warranty cases, it seems reasonable to suppose that the ultimate burden in "bad blood" cases would fall on the blood banks. Other factors which should be considered in resolving the warranty question are the same or similar to the factors considered in the separate concept of "strict liability," treated below.

B. Strict Liability

Liability without fault may also be obtained through a modern device, separate from warranty, called "strict liability." This is a social device developed in the last 100 years, designed to shift certain inevitable losses to those best able to bear them—generally a substantial segment of society. In the area of blood transfusions, the doctrine of strict liability would hold the suppliers and those administering blood transfusions absolutely liable (no negligence need be proven) for all transfusion injuries. The hospitals, blood

116. The Minneapolis War Memorial Blood Bank supplies whole blood and plasma to all but four Minneapolis hospitals. The exceptions are University of Minnesota Hospital, Veterans’ Administration Hospital, General Hospital, and Swedish Hospital. Interview with Dr. G. Albin Matson, supra note 60.

117. The doctrine originated in England in Rylands v. Fletcher, L.R. 3 H.L. 330 (1868). The rule was early adopted in Minnesota, Cahill v. Eastman, 18 Minn. 324 (1872), and is still followed today, Bridgeman-Russell Co. v. City of Duluth, 158 Minn. 509, 197 N.W. 971 (1924).

banks, or manufacturers could then "shift" the burden of the loss over the entire group of persons receiving transfusions. Or, by means of insurance, shift the burden of loss upon all the hospitals and blood banks which would in turn shift the burden upon all the users of their facilities—a substantial segment of society.

Strict liability, however, is limited in its applicability. Prosser states that this doctrine has normally found expression "where the defendant's activity is unusual in the community, and the danger which it threatens to others is unusually great even though the enterprise is conducted with every possible precaution." He further states that the "courts have tended to lay stress upon the fact that the defendant is acting for his own purposes, and is seeking a benefit or a profit of his own from such activities." Viewed in this light, strict liability seems inapplicable to blood transfusions. It can hardly be said that blood transfusions are unusual in the community; on the contrary, transfusions are an everyday occurrence in a community of any size. Secondly, although some danger is admittedly involved, the danger, absent negligence, can hardly be classified as unusually great. Lastly, and possibly most important, the supplier of blood, at least the hospital, does not seem to fit into the category of one "acting for his own purposes." The latter category seems to imply primarily a profit-seeking defendant. Although some hospitals do, of course, show a profit, the tag of "primarily profit-seeking" would appear to be a misnomer.

**Conclusion**

Although it would seem clear that doctors and hospitals should be just as responsible for acts of negligence as other tort defendants, the wisdom of imposing liability in the area of blood transfusions, through use of negligence per se—under a Pure Food and Drug Act, implied warranty, or strict liability, is open to question. Strong arguments, however, may be advanced on either side. This Note has not attempted to resolve these questions, but rather to present the opposing views—with some leaning toward not imposing liability. Final determination of the questions raised calls for an extensive factual inquiry into the problems discussed in the sections of this Note on warranty and strict liability. Perhaps this inquiry could take the form of a cooperative research project conducted by lawyers, doctors, blood experts, and administrators of both hospitals and blood banks.

119. *Id.* at 317.
120. *Id.* at 318.