REFRESHER COURSE FOR GENERAL PRACTITIONERS

BLOOD GROUPS AND DISPUTED PATERNITY

BY

GILBERT FORBES, M.D., F.R.F.P.S., F.R.C.S.Ed.

Senior Lecturer in Charge of the Department of Forensic Medicine in the University of Sheffield

The paternity of a child may be disputed either when the child is born in wedlock and the husband denies paternity or when the child is born out of wedlock and the man named as the father denies paternity. The first set of circumstances may lead to divorce proceedings, while the second results in an action by the woman to obtain a court affiliation order against the man in respect of her child. In actions of the latter sort, the sympathy of the court is usually with the woman, who presents a rather pathetic figure, and, though the Bastardy Laws Amendment Act, 1872, requires that the woman's story be corroborated "in some material particular," the degree of corroboration demanded by the average bench of magistrates is not exacting. Consequently gross injustice might be done to a man wrongfully accused by a mendacious woman, and he would be left almost defenceless. The attitude of some courts is shown by the evidence given by a well-known Metropolitan Police magistrate before a Parliamentary Committee considering the Bastardy (Blood Tests) Bill, 1938. He said that he would hold as the father of the child any man who had had intercourse with the woman about the probable time of conception. His evidence was based on the assumption that "the man who may be the father must pay," whether he is in fact the father or not.

This state of affairs is being rectified in many parts of the world as the value and reliability of blood grouping in determining blood relationship become more widely appreciated. In 1924 blood grouping in affiliation proceedings was introduced into the German courts, and it was estimated that by 1929 the tests had been applied in Germany in more than 5,000 cases. Other countries which followed suit include Austria, Denmark, Norway, Lithuania, Czechoslovakia, Holland, Sweden, Japan, Russia, Italy, Belgium, and the United States of America. In many jurisdictions not only is the result of the test accepted in evidence but the courts have the power to compel the parties involved to submit to a blood examination. In Great Britain there is, as yet, no statutory power of compulsion, as the Bastardy (Blood Tests) Bill had to be shelved to make way for more urgent legislation just before the outbreak of war in 1939.

History

The first observations of the agglutination of human red cells by serum belonging to the same species were made by Landsteiner in 1900. The discovery that the blood groups were inherited characteristics increased their importance. In 1910 von Dungern and Hirsfeld established the Mendelian inheritance of the groups, but it was not until 1924 that the exact manner of their inheritance was worked out by Bernstein. In 1926 Landsteiner and Levine discovered the MN system and the P system. In 1939 and 1940 Levine and Stetson and Landsteiner and Wiener discovered the Rh groups. Within the last six or seven years five more systems have been discovered: these are the Lutheran, Kell, Lewis, Duffy, and Kidd.

Genetics

Before a characteristic can be used in the study of inheritance it must be shown that it is free from the influence of age, diet, disease, or climate. The blood groups fulfil these requirements. Further, they remain constant from birth till death.

The characters of the body, in the widest sense, are controlled by hereditary factors called genes, which are present in every cell. They are passed from parents to offspring in the gametes. The genes exist in pairs called "allelomorphs," whose members are derived one from each parent. The two genes comprising an allelomorphic pair may be either similar ("homozygous") or dissimilar ("heterozygous"), and the persons possessing such allelomorphs are called "homozygotes" or "heterozygotes." When two genes are brought together in a cell they retain their identity and do not fuse.

In man each somatic cell carries 24 pairs of chromosomes in which the genes are located, but during meiosis the number is reduced by half. Each gamete is really only half a cell from the genetic point of view, and fertilization is an additive process during which the number of chromosomes is restored to 24 pairs. This theory of heredity was enunciated by Mendel (1822-84). Thus, if a person belongs to the blood group AB, one germ cell will carry the A gene and its fellow the B gene; therefore at fertilization the new being will receive either an A gene or a B gene from that parent, but not both.

In an individual, homozygous with regard to a particular pair of genes, the characteristic attributable to these genes will show strongly. If in a heterozygote the same characteristic manifests itself equally as strongly as in the homozygote, then that characteristic is said to be "dominant" and the alternative characteristic, which is obscure in the heterozygote, is called a "recessive." This is Mendel's first law. Dominants therefore may be of two genetic types—homozygotes or heterozygotes—while recessives can only be homozygotes. If two different allelomorphic genes both express themselves and can be detected together then they are co-dominants—for example, M and N. A true recessive characteristic is not detectable in the presence of the dominant characteristic, other than by breeding experiments or family studies.

Mendel's second law is to the effect that when two or more pairs of genes segregate (separate) simultaneously the distribution of any one of them is independent of the distribution of the others. But in man, although there are probably many thousands of allelomorphs (pairs of genes) in each cell, there are only 24 pairs of chromosomes. Therefore the individual pairs of human genes cannot assort entirely independently: as they travel in the same vehicle (the chromosome) they may consequently be expected to reach the same destination. This tendency for pairs of allelomorphs to assort together because they are carried in the same

pair of chromosomes is called "linkage." All the genes in a particular pair of chromosomes are linked together, but no genetic linkage has yet been detected between any of the eight blood group systems.

The position of a gene upon a chromosome is called its "locus." The closer the loci, the closer the linkage between the genes, and those genes with loci exactly opposite one another in a pair of chromosomes are, of course, allelomorphs. It is sometimes possible for a particular gene to exist in more than two phases, so giving rise to a series of "multiple allelomorphs," but not more than two members of such a series can coexist in the same individual.

The type of an organism judged by its characters is designated its "phenotype," while its "genotype" is its genetic constitution. Thus, to say that a person's blood belongs to group A is to give its phenotype; but two group A bloods may be genetically different—that is, either A/O or A/A. Therefore, a *phenotypical* description of some character is incomplete from the genetic point of view. The blood groups are controlled genetically by a system of multiple allelomorphs. For example, group O is due to the operation of a pair of allelomorphous genes O/O. Groups A and B are each produced by a different gene substitution (A or B). Both A and B genes are co-dominant with the gene O. It will be evident from the above that, from the point of view of blood grouping, the results of any mating can be predicted within certain limits once the mode of inheritance of the blood factor in question is established with certainty.

The Blood Group Systems

It is now known that there are at least eight systems identifiable in the blood of man—that is, ABO, MNS, P, Lewis, Rh, Lutheran, Kell, and Duffy. The mode of inheritance of some of these factors has now been established beyond reasonable doubt, one might almost say with absolute certainty, and only these groups can be safely used in excluding paternity. As more and more family studies accumulate, doubtless the mode of inheritance of the others will be elucidated, and they also will be utilized.

ABO Groups

In 1900 Landsteiner, who discovered this initial system, showed that, according to whether the red cells contain one or other, both, or neither of the two antigens A and B, the blood of human beings could be classified into four groups—that is, four phenotypes—A, B, AB, or O. He also demonstrated that the serum of an individual does not contain the antibody for an antigen present in his own red cells. In 1911 the subgroups A_1 , A_2 , A_1B , and A_2B were described. Next was reported the occurrence of individuals of group A whose blood gave only feeble reactions even with the most potent anti-A sera; this was attributed to a third sort of A agglutinin named A_3 . The existence of the agglutinin A_3 gives rise to two additional subgroups in group A—namely, A_3 and A_3B —but these subgroups are extremely rare. Recently it has been found that on testing unknown cells with anti-A, anti-B, and serum from an O person (anti-A + anti-B) the O serum on rare occasions agglutinated cells which did not react with anti-A or anti-B or with mixtures of both. This reaction was due to the presence of the agglutinin A_4 . Such bloods would have been

mistakenly regarded as O blood if the O serum had not been used. This subgroup is also very rare.

Inheritance.—Bernstein's theory of the mode of inheritance of the ABO antigens is now accepted as the correct one. It postulates three allelic genes—A, B, and O. Under this theory there is only one locus for the genes determining the blood groups in a single pair of chromosomes, at which locus any one of three genes may be situated. As each germ cell contains only one of the three genes A, B, or O from combinations of the three possible kinds of sperms with one of the three possible kinds of ova, six different genotypes result. Furthermore, the genotypes corresponding to each of the four blood groups are:

Phenotype	Genotype	
	Homozygous	Heterozygous
AB	—	A/B
A	A/A	A/O
B	B/B	B/O
O	O/O	—

The agglutinogens A and B cannot appear in the blood of a child unless present in the blood of one or both parents. It follows that if either parent is A/B the child cannot possibly be of the genotype O/O. Similarly, if either parent is genotype O/O, the child cannot be A/B. According to Bernstein's theory group O blood is not just A—B— blood, and therefore persons of genotype O/O would be expected to possess the agglutinin O in their red cells; the discovery in 1948 of the existence of sera which selectively agglutinate group O blood supports the theory. With regard to the heredity of the subgroups of A it is postulated that instead of the three allelic genes A, B, and O there exist four allelic genes A_1 , A_2 , B, and O. This extension of Bernstein's theory is supported by many family studies.

Source of Antisera.—As group A blood serum naturally has the antibody α (anti-B), and group B blood serum naturally contains the antibody β (anti-A), by suitable selection sera of a sufficiently high titre for testing purposes can be obtained from routine blood donors. All anti-A sera (from group B donors) contain two anti-A antibodies α and α_1 . Anti-A agglutinates the cells of all the subgroups A_1 , A_2 , A_1B , and A_2B , while α_1 (anti- A_1) agglutinates A_1 and A_1B cells only. The simplest method of subgrouping A and AB blood is with the aid of so-called "absorbed B serum." This is based on the fact that all group B sera contain two sorts of agglutinins—namely, the common anti-A agglutinin, which acts on both A_1 and A_2 cells, and anti- A_1 , which reacts only with A_1 cells. When the common anti-A agglutinin is removed from such sera, only that isoagglutinin remains which reacts with A_1 cells, so that in tests with such a reagent, if agglutination occurs the blood belongs to subgroup A_1 (A_1B), and if no agglutination occurs the subgroup is A_2 (A_2B). The anti- A_1 serum is prepared by absorbing selected B serum with A_2 cells. This removes the anti-A which acts on A_1 and A_2 cells, leaving the anti- A_1 in pure form.

MNS Groups

In 1927 Landsteiner and Levine showed that the M and N factors in the erythrocytes were independent of the ABO system. There are three types of blood—M, N, and MN—and no blood lacking one or other has ever been found. There are two allelomorphous genes—M and N—either of which determines, in single or double dose, the presence of the equivalent antigen in the red cells. An additional factor N_2 has been described, but it is very rare.

The pleasing simplicity of the MN system was complicated by the discovery in 1947 of a new antibody associated with M and N. This was called anti-S, and its association with the MN system was proved

statistically, though no association between S and the other systems could be found. It is possible that S is a separate gene having an allelomorph s.

Inheritance.—The agglutinogens M and N cannot appear in the blood of a child unless present in one or both parents, and M type parents cannot produce an N child, and vice versa. Not a single proved exception to this theory has been encountered in studies on a total of more than 15,000 children. Anti-S is a relatively recent discovery, and consequently extensive family studies have not yet been possible. One would therefore be on unsure ground in excluding paternity on the strength of the factor S alone. However, in a study of 123 English families none of the children was an exception to the theory of inheritance. Statistical analysis further bears this out.

Source of Antisera.—Anti-M and anti-N sera occur naturally in human blood, but not often enough or of sufficient potency to provide for routine testing. Normally these sera are prepared by the injection of OM and ON cells into rabbits. The rabbit serum is absorbed with cells from an A₁N person or an A₁M person respectively, to get rid of species specific antibodies and of the naturally occurring anti-A₁ which is sometimes present in rabbit blood. The correct preparation of these sera is technically difficult and requires much experience. Anti-S has been found only in human serum. It may be of natural or immune origin and is not in plentiful supply.

P Groups

In 1927 Landsteiner and Levine discovered that, when the sera of certain rabbits which had been immunized with human red cells had been absorbed to eliminate the species agglutinins, the rabbit serum could agglutinate some samples of blood and not others. The distinction could not be explained on the basis of the ABO or MN systems, and the two types of blood were called P+ and P-. These workers later found anti-P in a human being. The P factor is independent serologically of all the other known blood groups.

Inheritance.—P is inherited as a simple Mendelian dominant by a pair of genes P and p. Therefore a P+ child cannot result from the mating of P- parents.

Source of Antiserum.—This was originally obtained by immunizing rabbits, but it occurs naturally in human serum. It tends to vary in strength, and it may be difficult to distinguish between a weak P+ and a P-.

Rh Groups

In 1940 Landsteiner and Wiener, having immunized rabbits and guinea-pigs with the blood of the Rhesus monkey, found that the resulting antibodies in the rabbit and guinea-pig serum agglutinated not only the monkey red cells but also the erythrocytes of 85% of the sample of the white people of New York City tested. The former were classed Rh+ and the remaining 15% Rh-. The next discovery was that apparently the same antibody (anti-Rh) as that made by the rabbit against the monkey cells was present in the serum of persons who had had transfusion reactions following transfusion of blood of the correct ABO group. It was then shown that incompatibility of these groups between mother and child was the cause of erythroblastosis foetalis, and it became evident that the stimulation of blood group antibodies in the circulation of a person lacking the equivalent antigen could occur in at least two ways, either by transfusion of blood containing the antigen they lack or by sensitization of a pregnant woman by a foetus possessing an antigen (inherited from the father) which she does not have. At first

people were simply divided into Rh+ and Rh-, but it was ultimately shown that this system was more complex.

Five antigens have been discovered—namely, C, c, E, e, and D (named by Wiener rh', hr', rh'', hr'', and Rh₀). According to Fisher's theory there are six Rh antigens in three pairs, Cc, Dd, and Ee, and each of these antigens can in certain circumstances stimulate the production of its equivalent antibody, the relationship between the members of these pairs being one of genetic allelomorphism (see above). However, the antigen d has never, up to date, been conclusively demonstrated and Wiener doubts if it ever will. Wiener and Fisher disagree also about whether the three allelomorphic sites of Fisher are in one gene or in three genes, but the recent finding of a blood with the Rh antigenic formula —D—/—D— (no C, c, E, or e being present) seems to settle the dispute in favour of Fisher. The three genes C or c, D or d, E or e are carried in the same chromosome. This is proved because, if a person inherits say cde from one parent and CDE from the other he hands on to his children either cde or CDe. If the genes were in different pairs of chromosomes he would as often pass on Cde or cDe or CDe or cde. In addition to the genes being on the same chromosome, they are closely, or perhaps absolutely, linked.

A third antigen and gene allelomorphic with C and c is known; it is called C^w. Several examples of the specific antiserum anti-C^w have been found. The seven genes, C, c, C^w, D, d, E, e, can be combined in a chromosome in twelve different ways and therefore 78 different Rh genotypes are possible. As anti-d serum is rare or non-existent, and in any case is certainly not generally available, using the remaining six antisera, 28 distinct Rh types can be recognized.

Inheritance.—In the case of the Ee and Cc antigens, the agreement between the observed and the expected distribution of the children, based on Mendelian inheritance, is very close. In the case of Dd, when the antigen D is absent from the blood of both parents, it is not found in the children's blood. It is clear that a child cannot have an antigen D, d, C, c, E, e, if both its parents lack it. Further, the antigens are handed on in threes—for example, if a man inherits CDe from his mother and cdE from his father, then he, in turn, may transmit to his offspring either his three maternal antigens CDe or his paternal antigens cdE. If the exact genotype of an individual cannot be determined with certainty by the use of anti-D, anti-E, anti-e, anti-C, and anti-c, then a family study may make it possible to deduce what it must be. When the genotype is not certain, but alternative or probable, the alternatives are allowed for in assessing the children possible from various matings. These alternatives do not in any way invalidate the use of the Rh groups in cases of disputed paternity.

Source of Antisera.—The main source of Rh antibodies is the serum of human beings who have been immunized by transfusion or pregnancy. Intentional immunization of volunteers is another source.

Other Blood Group Systems

Mention must briefly be made of other blood group systems which, though they cannot yet be safely used in excluding paternity because of lack of sufficient data regarding their mode of inheritance, will undoubtedly be established in this field of serology in the near future.

The Lutheran antibody was found by Callander and Race in 1946. It was of immune origin and appeared after a

transfusion of blood containing the provocative antigen. It has been decided to call the antibody anti-Lu^a; the phenotypes Lu(a+) and Lu(a-); the genes and antigens Lu^a and Lu^b. At present, the gene Lu^b can be recognized only as an absence of Lu^a, as an anti-Lu^b serum has not yet been found. Investigations to date appear to show that the antigen Lu^a is inherited as a Mendelian dominant.

The Kell blood groups (K) were discovered by Coombs, Mourant, and Race in 1946. Up to 1950, 16 examples of this antibody had been recognized. The corresponding antigen k was found in 1949.

The Lewis groups were discovered in 1946 by Mourant. The antibody anti-Lewis was considered to be of spontaneous origin. The antigen is independent of all other known blood groups. There are two agglutinogens in this system, Le^a and Le^b. The genotypes are Le^a Le^a, Le^a Le^b, or Le^b Le^b. If only one antiserum, anti-Le^a, is used, the phenotypes which it defines are called Le(a+) and Le(a-). If both anti-Le^a and anti-Le^b have been used, the phenotypes are called Le(a+b-), Le(a-b+), or Le(a-b-). The Lewis factor is inherited as a Mendelian recessive.

The Duffy blood group system was discovered by Cutbush, Mollison, and Parkin in 1950. The antigen was shown to be inherited by means of a gene expressing itself in single or double dose, and the notation adopted was Fy^a and Fy^b for the genes, giving rise to genotypes Fy^a Fy^a, Fy^a Fy^b, and Fy^b Fy^b. Nothing is known with certainty about the mode of inheritance of the Duffy genes.

A serum that identifies another new blood group antigen was obtained from the parturient mother of a baby with erythroblastosis foetalis by Allen, Diamond, and Niedziela in 1951. This was called the Kidd antigen and it is independent of the other systems. It is present in the blood of about 77% of Americans. The inheritance of this group is now under investigation. This new antigen has been called Jk^a. The phenotypes are Jk^a + and Jk^a -, with genotypes Jk^a Jk^a, Jk^a Jk^b, or Jk^b Jk^b.

Blood-grouping Technique

There are many variations in technique, each worker adhering to that which he finds most satisfactory. Only one will be briefly described.

It is most important that glassware, syringes, etc., should be clean, quite dry, and free from all traces of alcohol, ether, or other antiseptics. Contaminants may cause haemolysis and render the sample unfit for testing. If the parties can visit the laboratory personally, then a few drops of blood may be taken from the finger or lobe of the ear and suspended in several millilitres of normal saline in a test-tube. The sample may be used direct, or it may be washed once by centrifuging, pouring off the supernatant fluid, and resuspending the cells in fresh saline solution. It is preferable, however, to test both the cells and the serum, and therefore it is better to take a few millilitres of blood by venepuncture, place the blood in a test-tube, and allow it to clot. It is then rimmed and centrifuged, when some of the serum can be pipetted off into a separate tube. Free cells in the serum round the clot can be drawn off and suspended in saline. This is centrifuged, the supernatant fluid is removed, and the cells are resuspended in saline to make a 2% suspension. The cells are now ready for use. If the blood is to be sent from a distance, experience shows that it travels best as clotted blood, and on arrival it can be dealt with as described above. It is necessary to have available a supply of reliable antisera—namely, anti-A, anti-A₁, anti-B, O, anti-M, anti-N, anti-S, anti-P, anti-D, anti-C, anti-c, anti-E, anti-e, and anti-C^w. Also, to test the unknown sera, a 2% saline suspension of known A, B, and O cells must be available.

The tests are carried out in round-bottomed tubes of 6 mm. internal diameter, and the tubes are arranged in a series of Ministry of Agriculture and Fisheries blocks—6.4 by 3 by 1.5 in. (16.25 by 7.6 by 3.8 cm.)—with apertures for the tubes in five rows of ten. Each block bears a permanent number on one face, and that face is always the front of the block. The number serves for identification and orientation. Before testing begins, a plan of the tests is drawn up on a series of diced diagrams corresponding to the blocks in use. Each diagram is numbered to correspond to a block, and the sera and cells to be placed in each tube are indicated on the diagram. It is thus unnecessary to label the tubes in any way.

Block 244

	Cells				Serum			
	Miss S	Baby S	Mr. Y		Miss S	Baby S	Mr. Y	
Anti-A ..					Controls			
					A ₂ O A cells			
Anti-A ₁ ..					A ₁ A ₂ B cells			
Anti-B ..					B O O cells			
O serum (anti-AB) ..					A ₂ O			

Arrangement of tubes in block.

One drop of the appropriate antiserum and one drop of cell suspension are pipetted into each tube. The tubes are then allowed to stand at the appropriate temperature for the reaction for from one and a half to two hours. The reactions from the ABO groups, the MNS groups, and the P group take place best at room temperature (18–20° C.), whilst the tests for the Rh factors are best done at 37° C. A drop of the material in each tube is then pipetted off, spread gently on a microscope slide, and the reading made microscopically. The results are entered on the prepared charts.

It is imperative that each antiserum should be controlled to ensure specificity and potency by using both positive and negative controls. The positive control must be a weak-reacting cell so that if this cell is agglutinated by the antiserum used under test conditions the test cell will be agglutinated if it contains the antigen. The negative control cells should be strong reactors for the antibodies which should be absent from the antiserum. This controls the specificity of the serum. In the case of the ABO groups, as both cells and serum are tested, it is unnecessary to employ more than one set of antisera. However, in the case of the other groups it is advisable to use two or more different antisera as an additional check. In the case of very rare antisera this may not be possible. The various controls used are shown in the Table.

Antiserum	Controls		
	Positive	Negative	Absorption
Anti-A ..	A ₂	O	—
Anti-A ₁ ..	A ₁	A ₂	—
Anti-B ..	B	O	—
O ..	A ₂	O	—
Anti-M ..	MN	N	—
Anti-N ..	MN	M	—
Anti-S ..	S+	S—	A ₁ B.S—
Anti-P ..	P+	P—	A ₁ B.P—
Anti-D ..	CDe/cde	CDe/CDe	A ₁ B.cde/cde
Anti-C ..	CDe/cde	CDe/cDE	A ₁ B.cde/cde
Anti-E ..	CDe/cDE	CDe/CDe	A ₁ B.cde/cde
Anti-c ..	CDe/cde	CDe/CDe	A ₁ B.CDe/CDe
Anti-e ..	cde/cde	cDE/cDE	A ₁ B.cDE/cDE
Anti-C ^w ..	C ^w De/cde	CDe/CDe	A ₁ B.CDe/CDe

It should be pointed out that the Rh factor controls are possible controls, not the only ones. Serum which has had its anti-A and anti-B agglutinins removed by absorption, as in the case of anti-P, anti-S, and the anti-sera for the Rh factors, is apt during storage to regain its agglutinin content. This might lead to false positives amongst A, B, or AB cells. A further control in these cases is necessary to allow for this possibility. The absorption controls are A or B or AB cells (according to the agglutinins absorbed) lacking the antigen to the antibody in the test serum—for example, AB.cde/cde for controlling absorbed anti-D serum. When the reactions are being read the controls must be examined before the actual tests. With the completed charts it is possible to determine whether the child in question could result from the mating of the parents.

Chances of Exclusion

The putative father in a case of disputed paternity naturally wants to know what his chances of exclusion are if he is innocent and submits to the tests. The answer depends on which blood groups are examined. With the ABO groups alone a falsely accused man has one chance in six of establishing his innocence. With the subgroups A₁ and A₂ his chance is increased by only a further 2%. With the ABO and MN groups together the chances of excluding paternity are raised to 30%. When the ABO, MN, and Rh factors are taken into account the chance of exclusion of a falsely accused man rises to 55%. In actual practice that percentage of exclusion is never achieved, as many of the men accused are actually the natural fathers of the children in question. It must be remembered that paternity can never be absolutely proved by blood grouping. *If the groups of the parties are such that the putative father could be the father of the child in question, the tests are inconclusive and should not be regarded as incriminating the man.* He, or any other man of the same blood type, might be the father. If, however, the child has in its blood an antigen which could not possibly have been inherited from either the mother or the putative father, then paternity can be definitely excluded.

Age of Development of the Blood Groups

The antigens ABO can be demonstrated in the foetus at 37 days and the sensitivity of the cells increases through antenatal and postnatal life up to the age of 20 years. The agglutinins are usually not developed at birth but rapidly increase in titre thereafter. The M and N factors are well developed in the newborn, and the same applies to the S antigen. That the Rh factors develop during intrauterine life is proved conclusively by the fact that an Rh-negative mother may develop antibodies to an antigen which is present in the foetus and which she does not possess. It is therefore safe to use the blood groups for medico-legal purposes when a child is in the early months of post-natal life.

Identification

In all medico-legal work accurate identification is vital, and attempts at impersonation must be prevented. If the parties attend at the laboratory together to have the samples taken, then they can identify each other. Usually they prefer not to do this, in which event they should produce some identification document such as a passport or a National Registration identity card, and

leave a signature. Wiener makes the mother and the putative father sign the report form, takes their thumb-prints, and takes a footprint from the baby. If the parties attend with a witness, then the name and address of that person should be noted.

If the blood is taken by someone other than the serologist for transmission by registered post, then each party should sign the label on the container in which the blood is put and complete a certificate, to be sent with the blood, certifying that the blood in the container bearing his or her signature is his or her blood. As an additional precaution the signed label may be used as a seal for the stopper, so that if the signed label arrives intact the possibility of the sample having been tampered with in transit is eliminated.

Presentation of Results

The results are usually presented in the form of a certificate giving the blood groups of the parties concerned in the case. There then follows an explanation of the results, stating whether the putative father could or could not be the natural father of the child in question. In the former event it is pointed out that the test is inconclusive and should not be considered as incriminating the man.

Nova et Vetera

THE TRAINING AND PRACTICE OF A LONDON PATHOLOGIST, 1637-46

BY

J. J. KEEVIL, D.S.O., M.D., F.S.A.

Library Keeper, Royal College of Physicians, London

Dr. Robert Wright was an exceptionally brilliant man, but in his lack of inherited wealth, dependence on patronage, and the brevity of his academic course he was the prototype of the London physician of his day. The qualifications M.D.Cantab., M.D.Leyden, F.R.C.P. are now obtained with such difficulty that it is hard to realize that in Wright's case they rested on no more than twelve days passed in a university. This indicates that the statutes of the English universities, requiring fourteen years of medical study before proceeding to the M.D., must be viewed with reservations.

At the Royal College of Physicians there are several biographical manuscripts, written in Latin, which are a guide to the actual procedure. The life of Robert Wright is recorded in one of these. He was born in London about 1600 and was left an orphan, without money, before he could be apprenticed to any calling. Presumably about 1615 "he offered his services to Dr. Robert Fludd . . . in order to be educated." In Fludd's house he worked "as a reader in the evenings and a scrivener in the small hours; in the daytime he sometimes did the work of an apothecary, and in his few hours of leisure he tirelessly followed the doctor's example and diligently read philosophy." Fludd was a Rosicrucian philosopher, and in spite of his D.M.Oxon. his progress in the College of Physicians was marked by a series of controversies. He succeeded, however, in becoming a candidate in 1607, a Fellow in 1609, and a Censor on four occasions. Wright, as amanuensis, must have been kept busy by his eccentric master, who produced a great mass of obscure works. His servitude continued until Fludd's death in 1637.

Simeon Fox's Patronage

Left once more destitute, Wright sought the help of Dr. Simeon Fox, son of the martyrologist, President of the College (1634-40), and noted for his generosity and for his encouragement of young scholars. Through the endeavours of influential men Wright secured his patronage; through his