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HUMAN GENETICS AND ITS RELATION TO MEDICAL PROBLEMS.

(Continued)

by

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CHAPTER IX.

THE GENETICAL VALUE OF CERTAIN SEROLOGICAL CHARACTERS.

Thus far the suggested value of applied genetics has been mostly of the speculative type, but we are now to discuss one aspect of the subject where genetics has already proved itself to be of definite and unquestioned value, and that is in cases, many of them medico-legal in nature involving the establishment of identity mostly personal or paternal, but in a few cases even maternal.

If and when we know how certain definitely measurable characters in man are inherited, it will be possible in many cases to predict the possibilities regarding such characters in the offspring of any two known parents. And similarly in many cases, knowing the character in question exhibited by one parent and child it will often be possible to state definitely the character that the other parent must have exhibited.

The former type of case may arise in maternity hospitals where occasionally a question arises as to which of two babies belongs to a certain mother. In such a case it may often happen that genetical investigations may be able to prove without a doubt the identity of one, and thus also of both infants. It may also arise where it becomes necessary to identify a body found, for example, drowned, or in a burnt building, or after an aeroplane accident. In such cases if the parents are available it may be possible to prove definitely that the dead person was not their child; in other cases it may be possible to prove nothing further than that it is quite compatible with the finding that the deceased may have been their child.

The latter type of case mentioned above most frequently occurs in efforts to prove paternity of a child born or suspected of being born out of wedlock. In a percentage of such cases genetical investigation of certain characters can definitely establish the innocence of the suspected father; in the rest of the cases it can merely prove as above, that the findings are consistent with the assertion that the putative father is the real father, i.e., we cannot as yet prove paternity, all we can do is to prove in such cases that the putative father and the real father both belong to the same subdivision of the population at large. Such evidence when considered along with other relevant evidence may be enough to convince a jury, but considered alone, never. The more characters we investigate, the more certain would be the proof of non-paternity in the former type of case mentioned above, and the smaller would become the section of the population containing both the suspected and the real father, in the latter type of case.

The more human characters therefore which we can study and whose methods of inheritance we can solve, the more value will the science of genetics be to legal medicine. Characters such as haemophilia etc., are too rare to be of any service, and colour blindness, hair colour, eye colour, are not yet amenable to that exact quantitative treatment which is necessary to allow them to be used with any great certainty. But in certain serological characters we have been able to obtain such clear cut results that the method of their inheritance has now placed beyond any shadow of doubt their use and trustworthiness for such purposes. So important are these characters that their descriptions and their methods of inheritance will be dealt with at some length in the following pages.

ISO-HAEMAGGLUTINATION.

The opening days of this century witnessed the discovery of the phenomenon of iso-haemagglutination which lead up to the description of the so-called 'Blood Groups'. It can be stated without any fear of contradiction that the prime importance of this discovery has been the perfection of the operation of transfusion, an operation which must save and will continue to save thousands of lives annually. But there is another side of this discovery which has of late received much attention, and that is the genetical side, and it is with this aspect that the writer now wishes to deal at some length. Within quite recent years the genetical behaviour of the blood groups has been so definitely elucidated that their study rightly forms one of the most important and valuable foundations of Human Genetics; it is therefore here proposed first of all to give a brief sketch of the discovery of the groups and their behaviour, and then to bring forward the data collected by the writer and discuss its value.

The Landsteiner Reaction and the Four Blood Groups.

As in many other scientific advances it is very difficult to state exactly when the discovery was made. Furuhata (10) states that the phenomenon was described in old Chinese writings, but whether it is true that it was actually agglutination of corpuscles that was described or not, it can hardly be seriously maintained that the matter was the subject of scientific investigation, at any rate in the modern sense.

Shattock (33) in 1899 was attempting to discover whether there was anything in the blood of fever patients to differentiate them from healthy people. He noticed that the serum of the former, especially those suffering from pneumonia, caused an abnormal rouleau formation of red blood corpuscles. He states: 'On adding one loop of normal human blood to one loop of pneumonic serum, an immediate result was obvious to a naked eye, the drop appearing as if it held a deep red precipitate in suspension.'

There has been much discussion as to whether it was real agglutination Shattock saw. From his statement quoted above, it really sounds as though it was, but the fact remains that he did not recognise the reaction as what we now know it to be, nor did he realise its import. He was on the track without realising it and therefore the credit for the discovery must go to Landsteiner who has ever since remained one of the central figures in the field of haemagglutination.

In 1900 Landsteiner (20) discovered that the serum of some individuals had the power of causing the corpuscles of other individuals to coalesce into clumps. It had previously been known that the serum of one species had the power of thus agglutinating the corpuscles of other species, and this phenomenon was known as *'haem-agglutination'* to distinguish it from the phenomenon of agglutination of bacteria. Here were now two kinds of haem-agglutination, and in order to distinguish them they were called *'hetero-haemagglutination'*,—the phenomenon of agglutination of red cells of one species by the serum of *another* species, and *'iso-haemagglutination'*—the phenomenon of agglutination of the red cells of one individual by the serum of another individual of the *same* species. This latter phenomenon has also been justly designated, *The Landsteiner Reaction.*

Landsteiner at first examined the blood of 22 individuals and found by means of this reaction that he could classify his subjects into three groups. The corpuscles of the group which he called C were not agglutinated by the sera of either of the other groups, though the C-serum caused agglutination of the cells of both the other groups. His A-group cells were clumped by both the other sera, but the A-group serum clumped only the third group cells, it being without action on the cells of the C and its own groups. His third, or B-group cells were

clumped by both the other sera, but the B-serum was active towards the A-cells only.

Landsteiner explained these results by postulating the phenomenon to be an antigen-antibody reaction, and later additions to our knowledge have completely justified his explanation. It will be noticed that he did not come across any case in which the serum was completely inactive towards all cells, but a year or so later such cases were found by Sturli, a co-worker of Landsteiner's and von Decastello. The cells of people belonging to this group were found to be clumped by all sera except those obtained from the same group, and the sera of people in this group were completely inactive to all cells. Landsteiner's theory fitted the new findings. It was that there were two independent and specific agglutinogens which were capable of being present together or singly on, or both absent from the corpuscles, and that corresponding to these two agglutinogens there were two specific agglutinins. Each agglutinogen and its corresponding agglutinin were reciprocal in regard to their presence, that is to say that if an agglutinogen were present on the corpuscles of a certain individual's blood, the presence of the corresponding agglutinin in that person's serum was precluded, and conversely, the absence of a certain agglutinogen from the corpuscles demanded the presence of its corresponding agglutinin in the serum.

In 1907 Jansky (19) in Austria made the first complete classification of these groups, and he distinguished them by the numbers I, II, III, and IV. Unfortunately his results were published in too obscure a journal for such an important piece of work, and although his classification was used largely by hospitals in middle Europe, those in the west and in America followed a grouping published by Moss (27) in 1909, which grouping very unfortunately did not coincide with that of Jansky. Moss also called his groups I, II, III, and IV, but although his II and III happened to be the same as those of Jansky, the I of one classification corresponded with the IV of the other. This has resulted in dangerous confusion, and the necessity cannot be too strongly stressed of all clinicians making themselves aware of the situation.

In view of Janksy's prior claims all American scientific journals prefer his grouping, and those in Europe should do the same. At least all writers should state, and all clinicians and workers should know, which classification they are using. A further nomenclature has been recommended by the Health Committee of the League of Nations (generally referred to as the International Nomenclature) and is now in almost universal scientific use. Here the groups are designated **O, A, B,** and **AB,** the capital letters **A** and **B** indicating the presence of agglutinogens A and B respectively, the cells of group **O** having no agglutinogens at all. The agglutinins corresponding to the agglutinogens A and B are known as α and β respectively, and hence according to Landsteiner's theory, the complete blood formulae of the four groups

are as follows, the capital letters, representing the agglutinogen value preceding in each case the Greek letters representing the agglutinins:—

Group **O** = O, α and β
„ **A** = A, β
„ **B** = B, α
„ **AB** = AB, o.

The nomenclatures of the three classifications are shown in Table I, and it will be seen that the order **O, A, B, AB** correspond with Jansky's I, II, III, IV. Throughout this paper the International Classification will be used.

Jansky	International	Moss
I	**O**	IV
II	**A**	II
III	**B**	III
IV	**AB**	I

Table I, *showing the three ways of designating each blood group.*

From Table I the characteristics of cells or serum of any blood group can be readily made out.

Group **O**—The cells, having no agglutinogens, cannot be clumped by any serum, but its serum, containing both α and β will clump the cells of all the other three groups, since these cells contain either A or B or both.

Group **A**—The cells are clumped by sera of Groups **O** and **B** (which both contain α) while its serum clumps the cells of Groups **B** and **AB** (since this serum contains β).

Group **B**—The cells are clumped by sera of Group **O** and **A** (which both contain β) while its serum clumps the cells of Groups **A** and **AB** (since this serum contains α).

Group **AB**—The cells are clumped by the sera of all the other groups since these sera contain either α or β or both; but its serum, having no agglutinins at all, can clump no cells whatsoever.

Since the first important thing in testing bloods for transfusion is that the donor's corpuscles should not be clumped by the recepient's serum, it is readily understood why people belonging to group **O** are sometimes called 'Universal Donors' (their cells cannot be clumped by sera because they have no agglutinogens) and those belonging to group **AB** are known as 'Universal Recipients' (their sera having no agglutinins cannot clump any cells whatsoever). It should be noted that it is with these two important classes that the danger of ambiguity arises if numbers are used to indicate the groups without stating the classification,

for the universal donor belongs to group I in one case and to IV in the other.

The study of blood groups remained for some years merely a scientific exercise, until the Great War unearthed it and made it the foundation of the operation of transfusion. But the same war gave the opportunities for its further application in the realm of Anthropology. On the Salonika front, two doctors named Hirszfeld took advantage of their easy access to large numbers of many different races, and they set to work and grouped 500 members of a number of races. Two peculiarities immediately stood out in their results and they were:

(1) that the four groups, although present in every race, were never present in equal proportions (i.e., 25% of each) in any one race,

and (2) that the percentage of the four groups differed in different races.

This second finding has formed the basis of the application of blood grouping to Anthropology and Ethnology, for it was further noticed that the percentage of two closely allied peoples correspond much more closely than those of two widely different peoples. There was also a definite correlation between the **A** and **B** percentages on the one hand and the geographical situation of the race on the other, for, in general, while the percentage of **O** nearly always ranged about 40 and that of **AB** below 10, **A** was relatively high and **B** low, amongst peoples from the north-west of the Eurasian land mass, and as one passed in a south-easterly direction the **A** percentage fell and that of **B** rose till the latter reached its maximum in India. Then on up to Japan, **A** again rose at the expense of **B**.

Two peoples with curious group distributions have been found namely, the Australian Aborigines, who are all either Group **O** or Group **A,** and the pure North American Indians who also all belonging to these two groups, but with a higher Group **O** percentage than the Australians. The theory put forward to explain these findings is that the A and B agglutinogens arose as separate mutations, the former in the west and the latter in the east of Eurasia, and the blood group percentages found amongst present day races are the result of more or less complete mingling of these two prime races. The genetical ideas involved in this theory have been discussed at length by the writer elsewhere (38). And now with the word 'mutation' is inroduced the genetical side of this study.

The history of the study of the inheritance of blood groups is briefly as follows:—

In 1908 Epstein and Ottenburg (8) published some hereditary data, but it was von Dungern and Hirszfeld (7) who, in 1910 really began to get at the root of the matter when they showed that the groupings

themselves were not inherited, but that the iso-agglutinogens were. From the 72 families which they studied, they showed that these agglutinogens A and B behaved as Mendelian dominants, and the complete blood-formulae of the different groups as given above were explained by the assumption that the agglutinogens were dominant each to its respective agglutinin. In other words a blood grouping character was due to the the inheritance according to Mendelian laws of two independent pairs of factors *A, a,* and *B, b.* The presence of the factor *A* caused the appearance of the A iso-agglutinogen on the cells, and it was dominant to its allelomorph *a*, which in the absence of *A* caused the appearance of α agglutinin in the serum. A similar relationship existed between the dominant factor *B* and its corresponding recessive *b* in the causation of the appearance of the *B* iso-agglutinogen and the β agglutinin.

In the next table are set out the various genetical formulae (genotypes) of the individuals belonging to the different blood groups, and the possible gene combinations which could result in each case in the gamete formation according to this theory of Hirszfeld's, details of which were given in an earlier chapter. (It has always seemed to the writer that we need a different term to describe the blood phenotype of an individual. While it is perfectly correct to state that a person belongs to Group **A** or Group **B** as the case may be, it is not literally accurate to say he is Group **A.** While the term is correct and sufficient in describing the group, it is not correct when applied to the individual; we need a term to describe the blood phenotype, and for this purpose from now on we shall use the term '*haemotype*' which has the advantage that it can be used in connection with any new groupings that may be necessary,

Haemotype	Genotype	Possible Gamete Formulae
O	*aa bb*	*ab*
A	*AA bb* *Aa bb*	*Ab* *Ab, ab*
B	*aa BB* *aa Bb*	*aB* *aB, ab*
AB	*AA BB* *AA Bb* *Aa BB* *Aa Bb*	*AB* *AB, Ab* *AB, aB* *AB, Ab, aB, ab*

Table II. Showing the possible genotypes and gamete formulae of each haemotype according to Hirszfeld's theory.

e.g., the sub-groups of Group **A,** or Landsteiner's M & N. Individuals therefore who belong to Group **O** will be described as being haemotype **O,** those belonging to Group **A** as haemotype **A** and so on).

Although recent work has proved this theory no longer tenable, it is important to understand it fully, because the knowledge acquired in order to test its validity is almost as important an addition to this study as the result of its disproof. But before dealing with that question, we must note from the above table that an agglutinogen cannot appear in a child unless it was present in at least one of the parents. (For an agglutinogen to be present in a child, the child must have inherited the corresponding gene from either its maternal or paternal gamete, and from the above table it is seen that no gamete can carry this gene unless it is also present in the genotype.) This law still holds, and, as we shall see later, is the whole basis of the medico-legal application of this work.

It must be noticed that the converse is not necessarily true, namely, that if an agglutinogen is present in a parent it shall also be present in the offspring. That all depends on the genotype of the parent as seen also in the above table. Let us consider a parent of haemotype **A.** He or she belongs to Group **A** because the factor *A* is carried on one of the chromosomes of the pair involved. Now as the chromosomes are doubled, it does not matter whether *A* is carried on both of them or whether one carries *A* and the other *a,* for since *A* is dominant to *a,* in both cases will *A* produce its effect, i.e., in both cases will the individual's corpuscles show the *A* agglutinogen reaction. In the former case the genetical formula for this character will be *AA* (in which case it is said to be homozygous), and in the latter *Aa*—heterozyous. When therefore the parent is heterozygous, the gamete going to the offspring may carry either *A* or *a* and in this case the offspring may or may not be of the same haemotype as this parent. We thus now understand how it is that the haemotype itself is not inherited in the usually accepted sense of the term.

Naturally, as time went on, a large amount of family data was collected which put this theory to the test. From the last table we can construct another one showing the possible haemotypes of children resulting from matings between members of any two of the blood groups. Such a table is the following.

When family data involving all these matings are examined, it is noticed that most of the haemotypes of the children agree with the expectation according to this theory except where one of the parents is haemotype **AB.** Especially marked is the difference between expected and actual haemotypes resulting from the mating **O × AB.**

As stated previously, perhaps the greatest genetical advance since the rediscovery of Mendel's work, has been Morgan's Theory of the Gene, which has opened up the study of this science to statistical treatment. By using such methods Bernstein (2) in 1925 showed there was

Haemotype Mating	Gamete combinations possible	Possible resulting genotype	Possible resulting haemotype
O × O	*ab* × *ab*	*aa* *bb*	O
O × A	*ab* × *Ab*	*aA* *bb*	A
	ab × *ab*	*aa* *bb*	O
O × B	*ab* × *aB*	*aa* *bB*	B
	ab × *ab*	*aa* *bb*	O
O × AB	*ab* × *AB*	*aA* *bB*	AB
	ab × *Ab*	*aA* *bb*	A
	ab × *aB*	*aa* *bB*	B
	ab × *ab*	*aa* *bb*	O
A × A	*ab* × *ab*	*aa* *bb*	O
	ab × *Ab*	*aA* *bb*	A
	Ab × *Ab*	*AA* *bb*	A
A × B	*ab* × *ab*	*aa* *bb*	O
	Ab × *ab*	*Aa* *bb*	A
	ab × *aB*	*aa* *bB*	B
	Ab × *aB*	*Aa* *bB*	AB
A × AB	*ab* × *AB*	*Aa* *bB*	AB
	ab × *Ab*	*aA* *bb*	A
	ab × *aB*	*aa* *bB*	B
	ab × *ab*	*aa* *bb*	O
	Ab × *AB*	*AA* *bB*	AB
	Ab × *Ab*	*AA* *bb*	A
	Ab × *aB*	*Aa* *bB*	AB
	Ab × *ab*	*Aa* *bb*	A
B × AB	*ab* × *AB*	*aA* *bB*	AB
	ab × *Ab*	*aA* *bb*	A
	ab × *aB*	*aa* *bB*	B
	ab × *ab*	*aa* *bb*	O
	aB × *AB*	*aA* *BB*	AB
	aB × *Ab*	*aA* *Bb*	AB
	aB × *aB*	*aa* *BB*	B
	aB × *ab*	*aa* *Bb*	B

Haemotype Mating	Gamete combinations possible	Possible resulting genotype	Possible resulting haemotype
B × B	*aB* × *aB*	*aa* *BB*	B
	aB × *ab*	*aa* *Bb*	B
	ab × *ab*	*aa* *bb*	O
AB × AB	*AB* × *AB*	*AA* *BB*	AB
	AB × *Ab*	*AA* *Bb*	AB
	AB × *aB*	*Aa* *BB*	AB
	AB × *ab*	*Aa* *Bb*	AB
	Ab × *AB*	*AA* *bB*	AB
	Ab × *Ab*	*AA* *bb*	A
	Ab × *aB*	*Aa* *bB*	AB
	Ab × *ab*	*Aa* *bb*	A
	aB × *AB*	*aA* *BB*	AB
	aB × *Ab*	*aA* *Bb*	AB
	aB × *aB*	*aa* *BB*	B
	aB × *ab*	*aa* *Bb*	B
	ab × *AB*	*aA* *bB*	AB
	ab × *Ab*	*aA* *bb*	A
	ab × *aB*	*aa* *bB*	B
	ab × *ab*	*aa* *bb*	O

Table III. Showing the possible haemotypes and genotypes resulting from all the different haemotype matings.

a significant difference between actual and calculated frequency of occurrence of certain haemotypes. E.g., if the frequencies of haemotypes **A** and **B** in a population are known, it is possible to calculate the expected frequency of haemotype **AB,** and this expected frequency was always much larger than the actual frequency found. On mathematical grounds, therefore, Bernstein enunciated another theory of the inheritance of the haemotypes, and by means of this theory he has shown that the expected and actual results agree very closely. The theory is one of *triple allelomorphs,* that is to say, that there are three genetical factors concerned in the haemotype formation, involving only one pair of chromosomes. Corresponding loci on each of these chromosomes will contain between them two of the three possible genes. These three factors are designated *A*, *B*, and *R*. The presence of the *A* factor causes appearance of the A agglutinogen on the corpuscles, independently of the other factors, and the presence of the *B* factor causes the appearance

of the B agglutinogens, this also being unaffected by the presence or absence of either *A* or *R*. When the two loci are each occupied by an *R* gene, the result is a recessive condition in which neither A nor B agglutinogen is present giving the haemotype **O.** If *A* is present and not B, the haemotype is **A,** if *B* is present and not A, the haemotype is **B**, and if both are present they both cause the appearance of their respective agglutinogens independently, thus resulting in the haemotype **AB.**

We are now in a position to construct a table along the same lines as table II showing, according to this new theory, genetical formulae of the genotype corresponding to each phenotype, and the possible gametes to be obtained therefrom.

Haemotype	Genotype		Gamete Formula
	Homozygous	Heterozygous	
O	*RR*	—	*R*
A	*AA*	—	*A*
	—	*AR*	*A*, *R*.
B	*BB*	—	*B*
	—	*BR*	*B*, *R*.
AB	—	*AB*	*A*, *B*.

Table IV. Showing the possible genotypes and gamete formulae of each haemotype according to Bernstein's theory.

We may now proceed to demonstrate as before the haemotypes possible in children in all the different matings and this is shown in Table V where, in the last column, the possibilities according to the two independent factors theory are given for comparison.

The points of similarity which should be noticed are:—

(1) No child can possess an agglutinogen unless it is also present in at least one of its parents.

(2) When neither parent has as agglutinogen, none of the children can have that agglutinogen (e.g., if neither parent has agglutinogen A, i.e., belongs to neither Group **A** nor Group **AB,** then none of the children can belong to Group **A** or Group **AB.**)

Haemotype Mating	Possible Resulting Haemotypes According to the	
	Triple allelomorph Theory	Two Independent Factors Theory
O × O	O	O
O × A	O, A.	O, A.
O × B	O, B.	O, B.
O × AB	A, B.	O, A, B, AB.
A × A	O, A.	O, A.
A × B	O, A, B, AB.	O, A, B, AB.
A × AB	A, B, AB	O, A, B, AB.
B × B	O, B.	O, B.
B × AB	A, B, AB	O, A, B, AB.
AB × AB	A, B, AB	O, A, B, AB.

Table V. Showing the comparison between possible haemotypes resulting from the different haemotype matings according to the two theories of inheritance.

The points of dissimilarity are found where haemotype **AB** enters into one or both of the matings. According to the triple allelomorph theory, a child of haemotype **O** cannot be born of parents one of whom is haemotype **AB,** and further where the mating is haemotype **O** with haemotype **AB,** neither of the parental haemotypes can appear amongst the offspring, they all must be either haemotype **A** or haemotype **B.**

These matings, **O** × **AB,** are important therefore in testing the validity of the theories of inheritance of agglutinogens, and data by the writer on this point will be given later, suffice it here to say that all the work done since 1925 has cast the vote overwhelmingly on the side of the triple allelomorph theory, which in the main is the almost universally accepted theory at the present day.

Before going on to mention some of the results of our genetical knowledge of blood groups, one must here mention the theories of Furuhata (11). In 1925, he also came to the conclusion that the then existing theory was inadequate, and he published his theory of triple allelomorphs which practically and statistically yields exactly the same results as that of Bernstein, but there are certain important theoretical differences between the two which should be mentioned here. Bernstein postulated three factors *'A,' 'B,'* and *'R,'* the last being recessive in character and therefore only causing the appearance of its particular phenotype when in the homozygous condition. His theory asserts that blood cells belonging to group **O** are not characterised by

the absence of A and B agglutinogens only, but also by the presence of a recessive agglutinogen R which is specific for that group. But this R agglutinogen is peculiar in that for some unknown reason its presence cannot be demonstrated by methods similar to those which detect the A and B agglutinogens. Hooker and Anderson (16) in 1921 recorded that they had proved the presence of a specific group-agglutinogen in bloods of haemotype **O** but subsequent work has not confirmed their results. Any theory of inheritance of haemotypes to be adequate must also explain the occurrence of agglutinins in the serum, and to supply this need Bernstein assumed that there were three agglutinins α, β and ρ corresponding to the three agglutinogens A, B, & R, and that these agglutinins were universally present in human serum, but that the complete blood formula of each haemotype was the result of certain agglutinogen-agglutinin bindings. It is a well established fact that the agglutinogens are present and demonstrable on the red cells as soon as the cells are formed during intra-uterine life, but that the agglutinins are not demonstrable in full strength until any time up to about the 9th month of post-natal existence. Bernstein's hypothesis assumes that as the three agglutinins are formed each is bound and rendered inactive by its corresponding agglutinogen if it be present. Thus cells of haemotype **O** having only agglutinogen R, absorb out agglutinin ρ and leave β & α which we can demonstrate in any serum belonging to a group **O** person. Cells of haemotype **A** (homozygous for A) would absorb out α and leave β & ρ, while those heterozygous cells of this group (genotype AR) would absorb α & ρ leaving only β. Similarly the constitution of haemotype **B** serum may be either $\alpha\rho$ or α and that of **AB** would be ρ. Now just as an agglutinogen R is peculiar in that its presence is not demonstrable by ordinary methods, so is the agglutinin ρ unable to exhibit its presence by ordinary serological procedure.

It must be understood that this is pure hypothesis, the above mentioned work of Hooker and Anderson being the only experimental evidence in its favour. The fact that the period of such a projected agglutinogen-agglutinin combination corresponds with the period when infantile convulsions are most common, has been brought forward as circumstantial evidence in favour of the theory, but this cannot be said to carry much weight. It neglects the fact that corpuscles are being manufactured throughout life and so presumably are agglutinogens and agglutinins, and this absorption combination must therefore be a continuous occurrence.

Furuhata on the other hand in his first hypothesis of 1925 assumed the presence of two agglutinogens only, A and B; and whereas Bernstein's haemotype **O** is characterised by the presence of an undemostrable group-specific agglutinogen R and by the absence of agglutinogens A and B, Furuhata's haemotype **O** is characterised by the absence of agglutinogens A and B only. The presence of these

agglutinogens is assumed to be due to two genetical factors *A* and *B* respectively, and the haemotype **O** to be due to the presence of a recessive factor *O* in the homozygous state. The factors *A* and *B* are each dominant to *O*. But again this does not take the serum into account and in 1927 Furuhata remedied this with his second theory in which he assumes the three allelomorphic factors to be *Ab*, *aB*, and *ab*.

Each is thus a double factor, but with each member of the gene pair being so closely located to its companion on the same chromosome that the possibility of crossing over between them is extremely small. They are therefore always inherited together—completely linked. *A* and *B* are dominant to *a* and *b* respectively, *A* being responsible for the appearance of the A agglutinogen, *B* being responsible for the B agglutinogen, while the recessive condition *aa* is responsible for the appearance of α agglutinin, and *bb* for the β agglutinin in the serum.

In table VI is set forth the genotype and possible gamete formulae of the various haemotypes according to Furuhata's two theories, along with each complete blood formula.

Haemotype	Blood Formula.	1st Theory		2nd Theory	
		Geno-type	Possible Gametes	Genotype	Possible gametes
O homozygous	*O*, α β	*OO*	*O*	*ab* *ab*	*ab*
A homozygous	*A*, β	*AA*	*A*	*Ab* *Ab*	*Ab*
A heterozygous	*A*, β	*AO*	*A* & *O*	*Ab* *ab*	*Ab* & *ab*
B homozygous	*B*, α	*BB*	*B*	*aB* *aB*	*aB*
B heterozygous	*B*, α	*BO*	*B* & *O*	*aB* *ab*	*aB* & *ab*
AB heterozygous	*AB*, *o*	*AB*	*A* & *B*	*Ab* *aB*	*Ab* & *aB*

Table VI. Showing the genotype and possible gamete formulae of the various haemotypes according to Furuhata's two theories. The blood formulae are given in each case, that of the agglutinogen being followed by that of the agglutinin.

The thing to note about Furuhata's two theories and that of Bernstein is that they are mathematically and in practice similar, having triple allelomorphs as their basis in each case. By some mysterious mechanism yet undiscovered, we cannot have A agglutinogen and α agglutinin in the blood of the same individual and therefore any theory that explains the inheritance of the agglutinogens will also be found to fit in with the agglutinins. That is why these theories are similar in

practice. But theoretically they are fundamentally different. Bernstein assumes cells of haemotype **O** to possess group-specific agglutinogens and the serum of haemotype **AB** to possess group-specific agglutinins, both for some reason not being capable of demonstration. Furuhata absolutely denies the presence of these group specific elements; but further than that, he attempts to give a direct genetical explanation of the presence and the inheritance of agglutinins as well as agglutinogens. It should be noticed that his second theory may also be considered as one of two independent pairs of factors almost completely linked. A and B are thus never on the same chromosome and hence a child can never inherit both of these from one and the same parent unless there is a crossing over, and the close proximity of the characters on the chromosome makes this extremely improbable; it does however open the door to a genetical explanation of cases of children of either haemotype **O** or **AB** being born from mating of haemotypes **O** and **AB.** It would seem that illegitimacy would be a much more probable explanation of such cases, but that question is one which only a large amount of accurate data will settle.

The value of this work then lies in the fact that knowing the blood groups of two parents we can predict the possible groups that may occur among the children. If a haemotype appears which is impossible for that mating, the child cannot be the true child of those two people.

TABLE VII.

Mating	Haemotype Possible	Haemotype not Possible
O × O	O	A, B & AB
O × A	O & A	B & AB
O × B	O & B	A & AB
O × AB	A & B	O & AB
A × A	O & A	B & AB
A × B	O, A, B, & AB	all possible
A × AB	A, B, & AB	O
B × B	O & B	A & AB
B × AB	A, B, & AB	O
AB × AB	A, B, & AB	O

Table VIII. Showing the haemotypes that are (*a*) possible and (*b*) not possible as a result of each of the different haemotype matings according to the now generally accepted triple allelomorph theory.

This is set out in Table VII, and such a table is of use in attempts to establish whether an infant is the real child of two parents. More often than not there is no dispute about the mother's child but the identity of the father is in question. The blood grouping evidence in such cases is set out in Table VIII.

TABLE VIII.

Haemotype of Mother.	Haemotype of Child.	Haemotype of true father.	Non-paternity can be proved if putative father is haemotype.
O	O	O, A or B	AB
O	A	A or AB	O or B
O	B	B or AB	O or A
A	O	O, A or B	AB
A	A	O, A, B or AB	cannot be proved
A	B	B or AB	O or A
A	AB	B or AB	O or A
B	O	O, A or B	AB
B	A	A or AB	O or B
B	B	O, A, B or AB	cannot be proved
B	AB	A or AB	O or B
AB	A	O, A, B or AB	cannot be proved
AB	B	O, A, B or AB	cannot be proved
AB	AB	A, B or AB	O

Table VIII. Showing (*a*) the possible haemotypes of one parent when those of the other and a child are both known and (*b*) when non-parentage can be proved in each of these cases.

Thus in a certain proportion of cases, the proportion depending on the frequencies of the four groups in the community, non-parentage can be proved by the application of our genetical knowledge to the occurrence of agglutinogens in the blood of mother, child and putative father.

HETERO-HAEMAGGLUTINATION.

In 1928 Landsteiner & Levine recorded the discovery that human red blood corpuscles may possess other agglutinogens in addition to those already known as "A" & "B." The three new types which they discovered were named "M," "N" & "P" and Furuhata has recently discovered a fourth in Japan which he has named "Q."

These differ from the "A" & "B" in this curious important fact that while there can be found in human sera specific agglutinins α & β which will agglutinate the A & B agglutinogens respectively, no naturally occurring human sera containing anti-M, anti-N or anti-P agglutinins have ever been found. Such anti-sera can only be made by injecting human red blood corpuscles into a rabbit and using the rabbit immune serum as an anti-serum for the type M, N or P corpuscles which were used for the injection. This type of agglutination where the reaction is between the agglutinogen of one species and a specific agglutinin formed in another species is known as *hetero*-haemagglutination, in contradistinction to the ordinary blood grouping reaction of *iso*-haemagglutination. Owing to the difficulties involved in working with the P agglutinogen and its relatively indecissive reactions, most of the work on these new substances has been confined to investigations concerning M & N.

These agglutinogens are inherited absolutely independently of the A & B agglutinogens and their manner of inheritance constitutes the second marked point of contrast between them and the A & B. The appearance of the M & N characters depends on a single pair of genetical factors, *M* which causes the appearance of M agglutinogen and *N* which causes the appearance of N agglutinogen.

The third great difference between the AB & MN types is that although we can demonstrate bloods containing, either both A & B, or either A or B, or neither A nor B, in the MN series we can only demonstrate blood of type MN, of type M, and of type N. No individuals have as yet been definitely shown to be without both the M and the N agglutinogen. The genotypes and phenotypes of the three groups are thus as set on the Table IX.

TABLE IX.

Phenotypes	Genotypes	Possible Gametes.
M+ N+	M N	M & N
M+ N−	M M	M
M− N+	N N	N

Table IX. Showing the genotypes of the three haemotypes M, N, & MN and the gametes that each can form.

Refering to Table IX we can see that this phenomenon of hetero-haemagglutination is also going to be of important medico-legal use.

It should be noticed that the heterozygotes are phenotypically distinguishable from the homozygotes and thus the genotype can be written down directly from the phenotype. The following table shows the possible haemotypes of the progeny in all the possible matings involving M & N agglutinogens.

TABLE X.

Mating	Possible Gamete Union	Haemotypes possible in children.	Haemotypes not possible in children.
M × M	$M+M$	M	N or MN
M × N	$M+N$	MN	M or N
M × MN	$M+M$ $M+N$	M and MN	N
N × N	$N+N$	N	M or MN
N × MN	$N+M$ $N+N$	MN and N	M
MN × MN	$M+M$ $M+N$ $N+N$	M and MN and N	all possible

Table X. Showing the haemotypes that are (*a*) possible and (*b*) not possible as a result of each of the different hetero-agglutinogen haemotype matings.

And now just as we did for the ordinary blood groups, we can devise a table for the application of this knowledge to paternity disputes. Such is Table XI.

The value of this evidence is twofold (*a*) it may confirm the evidence of non-paternity as deduced from the ordinary blood groups and (*b*) in some cases where this latter evidence is not able to establish non-paternity, the hetero-agglutinogen evidence may be able to do so and thus simultaneous examination of iso- and hetero-agglutinogen content of blood is able to establish non-paternity in a larger proportion of cases than either alone.

TABLE XI.

Haemotype of Mother	Haemotype of Child	Haemotype of True Father	Non-paternity can be prove if putative father is haemotype.
M	M	M or MN	N
M	MN	N or MN	M
N	N	N or MN	M
N	MN	M or MN	N
MN	M	M or MN	N
MN	N	N or MN	M
MN	MN	M, N, or MN	cannot be proved.

Table XI. In which is shown how knowledge of inheritance of heteroagglutinogens M & N may be of use in establishing non-paternity.

CHAPTER X.

METHODS OF ADVANCING THE STUDY OF HUMAN GENETICS.

No country, whatever be the order of its material prosperity, can have an ultimate value greater than that of its people themselves, whether that value be measured by standards moral, mental or material, and hence no country that has its future at heart can afford to neglect the future of its people. In other words the Eugenic aim of Galton should be one of the first aims of every wise human community. Just as the chemical characters of a solution depend on the molecules that give origin to the active ions, so the characters of the human population depend on the family unit which gives rise to the individuals responsible for human actions. The family is the ultimate unit on which the future of the state depends, and it is only through the family that this future can be controlled, and it is therefore on the family that interest biological and sociological should be centered. It is only through the study of *Human Genetics* in its broadest sense that we shall be enabled to understand, control and improve the reaction of the family unit to the environment, an environment which our experience has shown can change so markedly even from one generation to another.

How then may this study of human genetics be pursued to the greatest advantage of the community? It is obvious that the two main headings under which this question may be discussed are:—

(*a*) Methods of Investigation and

(*b*) Methods of Education.

(*a*) *Methods of Investigation.*

For the sake of argument and clarity we shall suggest that these should be divided into three main classes,

(*i*) studies of the inheritance of human characters as shown by individual families,

(*ii*) statistical studies of human characters as shown not only in families but in larger population groups, the size of the group depending on the type and frequency of the character being studied, and

(*iii*) comparative racial studies of inherited characters.

In many countries a scheme such as this is already being followed, but in very few is the importance of the organisation of this type of investigation under one advisory and controlling institution recognised. It should be the policy of every modern state to foster the organisation of such an institution, a policy in which Holland and Sweden are so ably setting a lead. A department concerned with the studies of class (*i*) would collect its data almost entirely through specialists in one or other of the main medical sciences such as physiology, pathology, clinical medicine both general and special, immunology etc., and the value of such observations would be greatly increased if the experts had a working knowledge of the principles of genetics. The main function of such a department would then be, not so much to carry out such investigations themselves, but to get in touch with investigators in the various hospitals and research institutions already in existence, with private clinicians who have the facilities for recording valuable and rare conditions, to stimulate the recording of valuable observations, to help to obtain the funds necessary for such work, and, from time to time, to correlate the findings thus collected.

The department responsible for studies of class (*ii*) would consist mainly of trained statistical geneticists who would deal with the correlation of the data derived from the investigators in class (*i*) and at the same time be responsible for the field work necessary for such investigations which are more purely genetical e.g., twin studies, linkage studies, consanguinity studies and those depending on more complicated methods of inheritance.

In the British Empire the third department should be exceptionally well developed, since here exists the opportunity of making observations both physiological and pathological on so many types of the human race and under almost every known climatic condition. We should like to stress here the value of comparative racial psychological studies, especially if they are capable of being followed up by similar studies in mixtures of these same races. Racial psychological differences are just as marked as morphological ones and the great advances recently made in quantitative estimation of psychological characters and the modes of

their inheritance, give this branch of the study every prospect of being one of the most fruitful in all the field of human genetics. We have already dealt rather fully with the question of anthropological physiology, and it just remains to point out again that these studies should not rest with the investigation of the reaction of different peoples due to hereditary forces, but should also include the consideration of the effect climate has to play on such reactions. In this way the study of anthropology may be made not only of sociological but of great medical importance as well.

(*b*) *Methods of Education.*

From the above suggestions we can see how greatly we shall be dependent on the clinician for the elucidation of our problems, and on the co-operation of the individuals of the community at large, and the help that they can give will only be forthcoming if they are educated sufficiently to appreciate its value. This end may be achieved to a certain extent through the admirable activities of eugenic organisations, but it must ultimately come through the individual members of the medical profession themselves. Our educational problem thus resolves itself into how the physician is to get this extra genetical knowledge which at present his medical training does not supply. We shall discuss this question under the two headings (*i*) for the clinicians of later generations, and (*ii*) for the clinicians of the present and immediate future.

(*i*) The only hope for the clinicians of the more remote future is to introduce into the medical curriculum of our medical schools and hospitals more instruction on human genetics. That suggestion will certainly meet with a very cold reception in these days when every specialist is pressing for more time in the curriculum for his special subject, *but the study of genetics is destined to form such an important and valuable part of medicine in the future that the sooner its introduction is considered, the better will it be for the profession and the community.* In Northern Europe it already forms a *compulsory* part of the medical course. In our shorter courses that would perhaps overemphasise the comparative value of the subject to the general medical man, but the need can be met without the formation of a new subject of study, by introducing such instruction into appropriate places in the existing curriculum.

The genetical work covered in the zoology and botany classes should again be brought to the notice of the students during their anatomy and physiology period, not only to make them realise that the study of genetics does not begin and end with peas and flies, but also to prepare them for work on those clinical states which we already know are mainly genetical in aetiology. In modern times, no physician can treat lightly the chance that he may need to know something of the inheritance of blood groups either in transfusion or in medico-legal

work. In some schools where the anatomist is interested in genetics the course could be given as a short but none the less important introduction to embryology. In other schools it may be found better to give it in the physiology course during either elementary work on the cell or, which is perhaps better, when iso-haemagglutination is being discussed. Whatever be the actual method of choice, the student should enter on his clinical period with a definite knowledge of genetical principles as applied to man, and the realisation that it may at any time be of use to him in his future work. The large percentage of practitioners will be completely engrossed in the practical side of their profession, and for them the amount of instruction thus obtained will be sufficient, and further genetical training would be an uneconomical use of their time; even if these had the training they would not make use of it, for their interests lie in other directions. But there will always be a small percentage of practitioners interested in genetics, and it is from the ranks of these men that we must look for the human data which are going to establish genetics as an applied science. These men must therefore be given further opportunities during their student days of becoming more familiar with the exact position and the field of human genetics. For these reasons every medical school should provide a voluntary course of 6-10 lectures on the strictly clinical side of genetics for the senior students; thus would the instruction be given only to those interested enough to attend, the very people who in after life would be able to apply their knowledge and collect data, and thus would those interested in the more immediately practical side of their profession not have their time taken up with what to them are less important matters. It goes without saying that these lectures should be given by a medical man; even the non-medical geneticists who now give these courses would be the first to admit the wisdom of this step, regarding themselves not as permanent teachers, but rather in the light of missionaries eagerly looking forward to the day when the medical profession will be enlightened enough in this direction to carry on the work by members from its own ranks.

(*ii*) The only way of doing something for the present and the immediate future is by reaching the present practitioners. Isolated lectures to medical societies are definitely better than nothing, but the subject really needs a series of consecutive lectures to do it justice, and busy practitioners have time for only one or two such meetings a month and then it is only reasonable to expect that these should be planned to cover many of the phases of medical practice and not be limited to just one aspect. It is true that a number of recent books have tried to supply this demand, but the only efficient method to adopt would be one similar to that which we suggested for students; all general and post-graduate schools should provide a course of voluntary lectures on applied genetics. By this means the opportunity of acquiring this knowledge would be given to the very people who could use it best,

those who, by personal experience have come to be impressed by its importance and value, and who are eager to take back to the scenes of their labours any new learning which may be of use to them in their unceasing war on disease.

But whether any attempt is made or not to raise the general level of genetical knowledge in our profession, one thing is necessary now and that is to dispell the vagueness and uncertainty of meaning of certain terms now commonly used in medical literature. The terms referred to are "congenital", "hereditary" and "familial".

Congenital. A congenital condition is simply and solely one which is present at birth and therefore may or may not have a genetical foundation, and this term has thus no real place in exact genetical writings and should be reserved for conditions better suited for it e.g., congenital dislocation of the hip or congenital syphilis.

Hereditary. Any condition which has a genetical foundation is strictly speaking hereditary, but unfortunately this term has come to be used by medical writers as descriptive of a certain type of disease, viz., one which appears in the family of an affected parent. It is thus handed on from generation to generation, the child inheriting it from the parent. From the examples in the earlier chapters we now know this is simply one special type of inheritance where the abnormality is due to a dominant gene and thus affected children must always have at least one affected parent, and an affected parent will beget affected children—if the family be large enough—the proportion depending on whether the parents be homo- or hetero-zygous for the factor concerned.

There is yet another use of the term "hereditary" which needs consideration. Some clinicians, especially neurologists and psychiatrists consider that certain conditions are the result of the union of *impaired germ* cells and they thus call the resulting characters hereditary. From the foregoing pages it should be evident that the whole of the conception of modern genetics is that inherited characters are due to the reaction between the environment and certain factors which are transmitted from one generation to the other by carriers, many of which we have identified as genes on the chromosomes, and some of which we are prepared to be convinced may be demonstrated as being in the protoplasm. In true Mendelian inheritance every factor in the cells of one generation must have had its counterpart in the germ cells which went to form the two parents in the preceding generation. This is not so in the above mentioned examples. Here in the time that elapses between the fertilisation resulting in the formation of the parental zygotes and the formation of the germ cells in these individuals, something happens to affect the factors, so that different factors are handed on from those that were originally present. The new characters that appear as the result are thus not inherited in the Mendelian sense, but once they have been established

in one generation, they may be handed on and thus become inherited in the true sense in following generations.

This is really no new thing in experimental genetics; germ cells have been bombarded by X-rays with the result that their factor carriers are altered and new characters make their appearance in the next generation. Those new characters can hardly be said to have been inherited from the parents, but once they have been established, they may be transmitted and thus become hereditary if the change brought about in the genes is permanent and is not so great as to have a lethal effect.

Familial. This term is usually used to describe conditions which appear among several children of one family without being present in the parents. It does not appear to be "inherited" from the parents therefore, but it occurs "in families". It is however just as much hereditary as one of the last class. These diseases or abnormalities are examples of recessive conditions, the children being born of two heterozygous parents who are thus phenotypically normal.

In clinical work however it would be much better if the present use of the terms familial, congenital and hereditary was discontinued, the latter term being restricted to those conditions whose existence is capable of explanation by the ordinary principles of Mendelian inheritance, and the conditions at present referred to as congenital or familial should be given their own accurate genetical description. This is already done in cases of sex-linked recessive inheritance—of which there are a number of known examples in clinical medicine—and the method could well be followed in the other cases mentioned.

And here we must allow the case to be put to the medical jury. It is repeatedly being asserted that genetics has not yet reached the stage where it may be applied to human work. The defence has attempted to show what an exact science genetics of lower forms of life has now become; has attempted to explain that the reason that genetics as applied to human work is not generally accepted is because human findings do not seem to fit in with elementary genetical laws, but that, with a more intimate knowledge of advanced genetics, the grounds for this seeming inapplicability disappear, and with it disappears the only evidence to support the view that the mechanism of human heredity differs in any way fundamentally from that of any other forms of life, either plant or animal.

ANTHROPOLOGICAL STUDIES AMONGST NORTH AMERICAN INDIANS OF BRITISH COLUMBIA.

by

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Through the kindness of Dr. G. E. Darby of the R.W. Large Memorial Hospital, Bella Bella, British Columbia, Professor Furuhata of the Imperial University, Kanazawa, Japan, and myself were given the opportunity after the 5th Pacific Science Congress in 1933 of collecting some anthropological data amongst the coastal Indians of British Columbia. It was decided to make investigations similar to those which I had carried out in British North Borneo in 1931 and '32 and hence the work was subdivided so that Prof. Furuhata did the blood-grouping while I collected the family histories and data concerning palm prints, ear pits and hair whorls. This paper is a full report on all the data obtained and although the blood grouping results have already been published by Dr. Furuhata (3), they are given again here in order that a complete report of our investigations may be recorded and in fairness to Dr. Furuhata it must be stated that for the opinions expressed in the discussion of these results, and for the conclusions now published, the writer takes full responsibility.

BLOOD GROUPS.

In all, 204 individuals were grouped but since one of these had been included in data previously published by Dr. Darby and Prof. Ruggles Gates, our total is reduced to 203. The details are embodied in Table I.

TABLE I.

Tribe	Totals	O	A	B	AB
Kwakiutl	69	55	11	3	0
Tsimshian	48	33	15	0	0
Salish	32	24	8	0	0
Nootka	30	29	1	0	0
Kitamaat	24	11	13	0	0
Totals	203	152	48	3	0
%		74.88	23.65	1.48	0

TABLE I. In which are set out the results of blood grouping 203 individuals drawn from various British Columbia Indian tribes.

Prof. Ruggles Gates and Dr. Darby (4) in discussing the results obtained from their investigations and from ours, point out that the

greater Group A percentage in our figures may be due to the fact that children were included in our subjects, while their tests were made on the older generation as far as possible. They conclude "The greater frequency of A & B in their results indicates an increase of white blood in the younger generation". In view of this statement, it seemed advisable to sub-divide our data into two groups, one group containing individuals over 20 years of age and the other, those under 20. This grouping is set out in Table II.

TABLE II.

	Totals	O	A	B	AB
Over 20	107	79	26	2	0
Under 20	93	71	21	1	0

TABLE II. In which the 200 individuals whose ages were taken, are divided into two classes; one containing those over 20 years of age, the other those under 20. The blood group percentages found in each class is set out and are not found to be significantly different.

The chi-square test shows that these distributions are not significantly different and hence if the younger generation is different from the older, our numbers are too small to show it. It would be interesting if the above mentioned authors could treat their data in a similar manner and see whether the combined data show any difference between the generations.

Our high Group A percentage is mainly due to the Kitamaat tribe, amongst which, of the 24 tested, 13 were found to be haemotype A. It is possible that it may be due to the fact that this small total, 24, may be solely comprised of members of two or three haemotype A families. The fact is that seven out of the thirteen are unrelated (as far as our records show) while the other six come from two families, one an A father with two A sons, and the other an A mother with an A son and an A daughter. Similar investigations into the three B haemotypes show two of them to be supplied by a father and son and the other no relation.

This question of families in anthropological data is one which demands attention. The whole of our p, q, and r calculations is based on the assumption that our subjects are chosen at random. But most field data are taken in family groups and in order to reduce data from different peoples to comparable forms, a family correction will have to be devised and applied for this unavoidable departure from random sampling.

To return to the question of racial mixture, it was difficult to get information on this point, but one very frequently came across subjects looking "very European". Most of them gave a pure Indian history except two cases, (*i*) A Salish man, haemotype A, who said his father was European although notes made on the spot say he "looks Indian". He had two sons both haemotype O, the one with "brown eyes, looks Indian" the other with "grey eyes, looks European". (*ii*) A man from Cape Mudge, haemotype A, stated his father was European. He had grey eyes but no note was made of his appearance. He has no children. Thus the only two cases admitting racial mixture with Europeans both belong to Group A.

From these results it is extremely difficult to come to any conclusion about the effect of racial mixture on blood group frequencies, but they are sufficient to show that amongst the native population of British Columbia we have an excellent opportunity of gathering data relevant to this question. In considering data thus collected it is important that workers should keep the actual problem ever before their minds. During the spread of the North American Indians into that continent from elsewhere, they would take with them hereditary characters from parent people or peoples. If during the transit or after arrival these characters did not undergo any change, we should be able to recognise the parent races from their similarities to the present day American Indians as judged by such hereditary characters, (providing also that the characters of the parent races as shown by those of their known descendents have not changed either). In the blood groups we have excellent examples of recognisable and individually stable characters, characters with whose mechanism of inheritance we are definitely familiar. When we come to compare the blood group frequencies of the Indians with those of other peoples, we can find no resemblance close enough to warrant our asserting racial affinity, and the generally accepted method of escaping from this impasse is to assume that the American continent was peopled before the B mutation arose or else by a people who had not yet acquired an infiltration of the B character. But unfortunately for this easy method of escape, all other anthropological evidence seems to point to the fact that the migration into America took place in relatively recent times. If this be so, the emigrants must have started off with both A & B agglutinogen characters, and they must have therefore lost them, either on the way, or after arrival.

In another paper (6) the writer has discussed this possibility and has pointed out that such a change in racial character may be brought about in two ways (*a*) by selection and (*b*) by mutation, and that the migration along narrow land corridors provides the opportunity for selection by inbreeding, which may result in the increase in the frequency of recessive characters in the community.

With regard to mutation it is interesting to note that the work of Hansen (5) on *Drosophila* shows that physiological states such as starvation have a definite power of increasing the rate of artificially produced mutations.

This fact introduces very important possibilities into the study of racial physiology and morphology. A group of people in an accustomed environment may show a constant frequency of a certain character, but once that environment of the group is changed, as it may well be during migrations, we have the possibility of new character frequencies being set up, especially if the environmental change, as for example that produced by translation from one continent to another, is a radical one.

From these arguments it becomes obvious that genetical studies of the peoples of eastern Asia and of the land corridors leading therefrom to Australia and America will be of great value. In North America the problem is none the less important because in the mountainous north we have the continuation of the narrow confines to which the emigrants must have been subjected even after they arrived on the continent, and genetical studies of the peoples along the different routes of dispersion from the North West of Canada through the Continent should be undertaken as soon as possible and should throw much needed light on this problem.

From our data the value of $p=.126$, $q=.008$, and $r=.865$, and $(p+q+r)=.999$.

PALM PRINTS.

Palmar Formulae.

Palm and finger prints of 53 individuals were taken and the former analysed according to the author's method (7) and the results set out in Table III and the left and right hand values compared.

In a previous paper (8) the mean values of these palmar main lines in the hands of natives of British North Borneo were compared with the above results and using the difference of twice the standard error as the limit of significance this comparison gives a significant racial difference in every main line with the exception of the B-line on the left hand. In every case the difference is such that the Canadian values are greater than the Borneo ones, that is to say that the papillary ridges on the palms of British Columbia Indians are directed in a more transverse manner, than those on the palms of British North Borneo natives. A reference to Table III shows that the palmar asymmetry previously recorded viz., that right hand values are greater than left, is also exemplified in the Canadian hands except in the C-lines where the difference is not significant.

TABLE III.

Line	Mean	S. E. of Mean	Difference of Mean	S. E. of Difference	Remarks
D_L	3.430	.076	.348	.116	S
D_R	3.778	.087			
C_L	2.785	.069	.144	.106	N.S.
C_R	2.929	.081			
B_L	1.910	.033	.286	.066	S
B_R	2.196	.056			
A_L	1.258	.033	.297	.039	S
A_R	1.555	0.30			
K_L	5.022	.031	.211	.032	S
K_R	5.233	.008			

TABLE III., in which is set out the mean values of palmar main lines as calculated from 53 British Columbia Indians. The right and left hand values are compared, and the conclusions given in the last column.

S = significant difference.

N.S. = difference not significant.

Thenar Patterns.

One of the outstanding characters of these palm prints was the frequent occurrence of thenar patterns, and the data concerning these are set out in Tables IV & V.

TABLE IV.

	+ +	+ −	− +	− −	Totals.
Males	13	16	3	27	59
Females	24	22	4	32	82
Totals	47	38	7	59	141

TABLE IV. Showing the frequency of thenar patterns on left and right hands in male and female British Columbia Indians. + + means pattern present on both hands, + − present on left and absent on right, − + present on right and absent on left and − − absent on both hands.

Applying the chi-square test to these figures we find the value of P lies between .8 and .9 and hence there is no significant difference in the occurrence of these patterns in the two sexes. With regard to the left and right hands however, we find that a pattern occurs on a left hand in 53.19% ± 4.20 of the cases and on a right in only 31.21% ± 3.30. The differences between these frequences is 21.98% ± 5.7 which difference is significant and hence we have here another palmar asymmetry viz., that the frequency of incidence of a thenar pattern on the right hand is less than on the left as shown by these data.

TABLE V.

	L +	L −	R +	R −
Males	29	30	16	43
Females	75	36	28	54
Totals	46	66	44	97

TABLE V. Showing the frequency of occurence of thenar patterns in the two sexes on left and right hands treated separately and not combined in pairs. L + means present on left hand, L − absent on left hand and so on.

The chi-square test when applied to this table again shows the sex difference to be insignificant and hence we see that the occurrence of patterns with reference to single hands is not different in the two sexes, nor is there any difference when considered in combination of pairs in left and right hands.

Thenar patterns occurred in either one or both hands in 82 individuals out of 141 or a frequency of 58.16% ± 4.15.

EAR PITS.

This developmental abnormality of the external ear is relatively frequent in the people of the Far East. (I propose to continue using the term "ear pit" until the embryological studies establish the correct anatomical name to be given to these abnomalities). What the frequency of occurrence amongst Europeans is is hard to say. Congden (2) states that "sinuses, fossae and scars have been described in several hundred Europeans by clinicians." Wood Jones (10) quotes Eyle as stating their frequency in white people to be 2 per 10,000. For over five years now I have been observing this abnormality and have come across it in only 4 white people (total number of individuals observed unknown). Congden has recorded 267 cases of its occurrence amongst Asiatics but

unfortunately the percentage frequency is not given. Wood Jones states that Stannus found them in 5.2% of females and 3.6% of males among African Negroes of Nyasaland, but not having access to the original paper for the actual numbers it is impossible to compare these results with mine. I have observed the frequency of occurrence amongst Borneo Natives, as well as amongst Chinese in Hong Kong, and when these figures are published, the racial data will be compared.

Amongst these Canadian Indians they occurred in 4 individuals out of 204, i.e., 1.96% ± .97. Of these four cases three were single—on the left in one case and on the right in two—and one was double.

HAIR WHORLS.

In a previous article (9) a description of occipital hair whorls was given. The frequency of the different types of whorls was also investigated in British Columbia and the data recorded in the same manner as previously, i.e., the whorls where they occurred were described (*a*) according to number—whether they were single or double, (*b*) according to direction—whether clockwise or anti-clockwise, (*c*) according to position on the head—whether situated to the left or the right of the middle line, or in the middle line itself.

The results are shown in Table VI.

TABLE VI.

SINGLE					DOUBLE		OTHERS	
	Left	Centre	Right	Totals	Cl A	8	Star	3
Cl	28	58	42	120	A Cl	1	Unable to find	20
A	8	28	17	53	Cl Cl	2		
Totals	20	86	59	173	Totals	11	Grand Total	207

TABLE VI. Showing the frequency of the different types of hair whorls recognised amongst 207 British Columbia Indians. Cl = clockwise, A = anti-clockwise, Cl A = clockwise whorl to the left of an anti-clockwise one etc.

It is not intended to discuss these results fully because the above method of description has now been modified for these reasons; (*a*) the description of the whorl position as being on the left, centre or right gives subdivisions which are not strictly comparable, because a whorl

to be classified as lateral may fall in any position out of the middle line, whereas a whorl to be classified as central must fall in one plane and one only; such a method of classification is therefore biased in favour of the lateral groups; (*b*) there is no valid reason why one should make a whole group of whorls lying in the medial sagittal plane any more than any other sagittal plane; (*c*) it is very difficult to decide what is the exact mid-line of the body and how to designate a whorl bordering on this line, and hence the accuracy of one's result depends greatly on one's mental state. Repeatedly on expeditions one notices that early in the day when one is fresh and more critical, whorls lying close to the mid-line are placed in one or other of the lateral groups, while later in the day when critical faculties are dulled by fatigue such types tend to be all classified as central; (*d*) even if one could detect the mid-line of the body accurately there is no reason to suppose that it corresponds with the embryonic mid-line and in the genetical consideration of these data this is important.

In short, the positions occupied by whorls on the head, form a continuous series and not a discreet one, and in our efforts to classify these positions we must choose a method which employs the same standard of accuracy for each position. I have therefore changed my method of describing the position of a whorl, no longer describing it in terms of its relation to the mid-line, but in terms of the ratio of its distance between that point and the corresponding point on the right of the head measured through the whorl. The chosen point from which the measurement is made is the upper margin of the tragus and the proceedure is as follows:—A millimetre tape is passed across the head from the left to the right tragus through the whorl and the inter-tragal distance recorded, as well as the distance of the whorl from the left tragus and this latter distance is expressed as a percentage of the former. One case recorded is as follows:—The distance from the upper margin of the left tragus to that of the right tragus was 350 m.m. The distance from the former point to the clockwise hair whorl was 180 m.m. The whorl and position are thus recorded Cl. $51\left(=\frac{180}{350}\times 100\right)$. Thus the location of every point on the head is subject to the same constant error of measurement and the locations are thus able to be arranged in a continuous series which can easily be dealt with statistically; by this means also to every position on the head can be applied a value, which value is characteristic of all whorls in that same sagittal plane, and in that same plane only.

Despite the fact that this change in method was made after the above data were collected we can discuss the results to a certain extent. Thus out of the 187 individuals in whom the whorls were diagnosed,

173 had a single type, i.e., 92.51% ± 1.92 Cl, 67.17% ± 3.51
A, 28.34% ± 3.50

11 „ „ double „ „ 5.88% ± 1.72

3 „ „ star „ „ 1.60% ± 2.14

Of the 173 single whorls,

120 were clockwise, i.e., 69.36% ± 3.50 and

53 „ anti-clockwise, „ 30.64% ± 3.50.

The only other data on hair whorls to which we have access is that of Bernstein quoted by Bauer Fischer and Lenz (1) and those of British North Borneo Natives (9). Unfortunately the former data do not include standard deviations and hence their comparative value is lost.

TABLE VII.

People	Number	Cl	A	Double	Star
British Columbia Indians	187	67.17% ± 3.51	28.34% ± 3.30	5.88% ± 1.72	1.60% ± 2.14
British North Borneo Borneo Natives	649	59.17% ± 1.93	28.12% ± 1.78	11.71% ± 1.26	—
North Germans	—	74%	19%	7%	—

TABLE VII. Setting out for comparison available racial data concerning hair whorls.

From Table VII it is seen that the differences in the percentage frequencies of various hair whorls found among the natives of British North Borneo and the Indians of British Columbia are as follows:—

Cl, 8.00% ± 4.005; A, 0.78% ± 3.749; Double, 5.83% ± 2.132

thus taking a difference of twice the standard deviation as the limit of significance, all we can say is that the difference these races show lies in the double whorl, which is more common amongst the Borneo people than those of British Columbia.

HEREDITY.

It is intended to leave to a later paper the full discussion of the inheritance of the various characters described in this article, but there is one interesting family which it is intended to describe since it shows

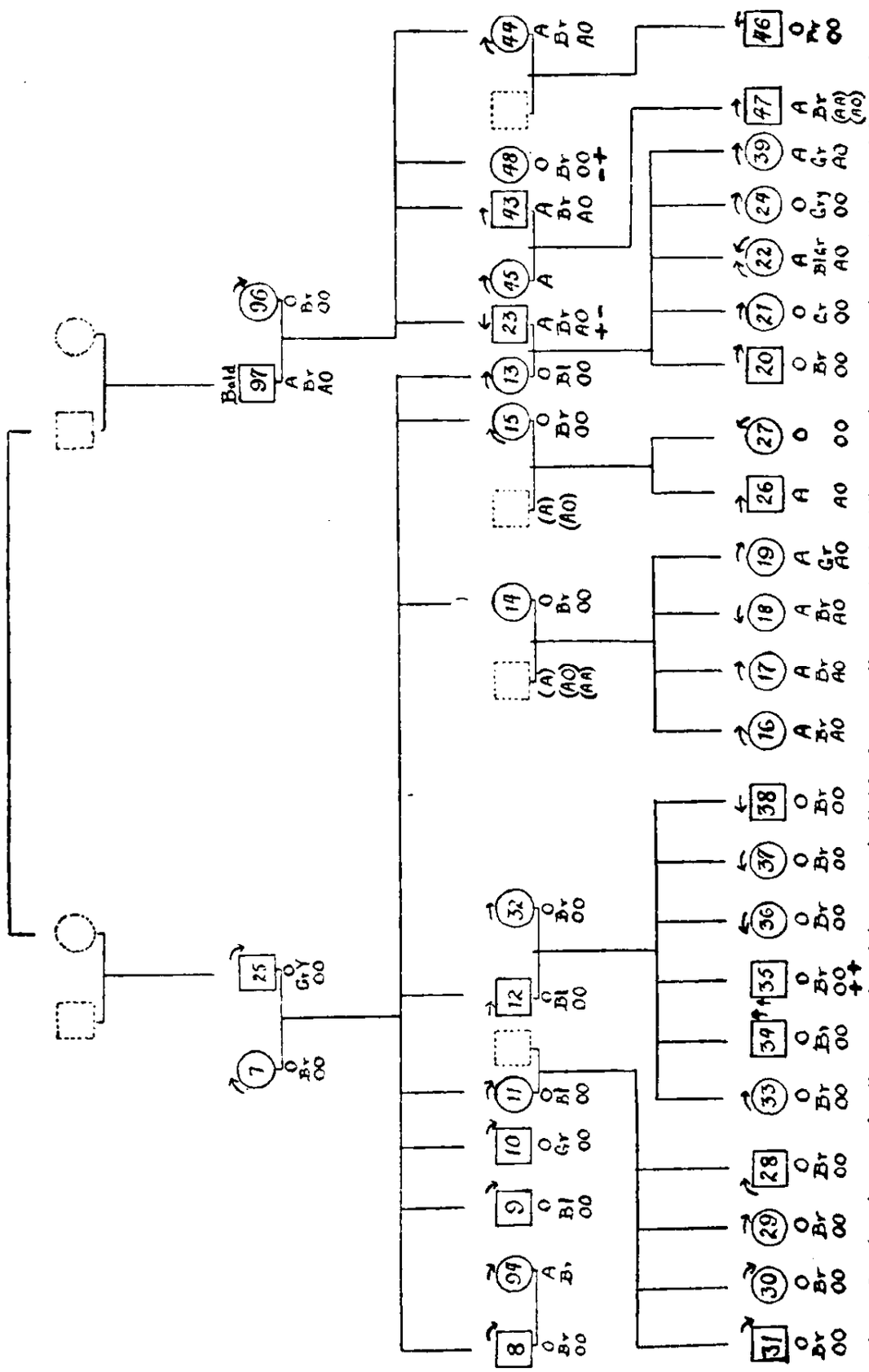

Chart I, showing a family tree involving 42 individuals actually examined. The numbers are the writer's serial numbers, males being indicated by squares, females by circles, the letter immediately underneath being the blood group, under that is the given eye colour, under that again the blood group genotype. In three cases (23, 35 and 48) ear pits were present, (+ +) meaning present on both sides, (+ —) present on the left and (— +) present on the right. The arrows indicate the type and situation of the occipital hair whorl, the clockwise whorl being indicated by an arrow directed in a clockwise manner, etc., and the position of the head of the arrow roughly indicating whether the whorl is on the left or right of the midline or in the centre. Letters in brackets indicate estimated values only. Br = Brown, Gr = Green, Bl = Blue, Y = Yellow Gry = Grey.

this fact that of the 4 cases of ear pits found in 204 individuals, 3 can be traced back to common parentage, two being members of one family and their father being the cousin of the grand-father of the other case. The fourth case comes from a different part of the country and no relationship was established. The fact that a rare character in a community tends to be found more commonly amongst relatives is evidence that the character is in some way inherited as a recessive condition, but the exact method of inheritence of ear pits will need much more family data for its elucidation than that which is given in this article. (See Chart 1.)

SUMMARY.

1. The blood group distribution amongst British Columbia Indians, as shown by examination of 203 individuals, is given, and the anthropological value of this data is discussed.

2. It is found that there is no significant difference between the distributions of the four groups amongst people under 20 years of age and those over 20, and hence these data do not support the contention that racial mixture is causing a change in group frequencies.

3. The effect of taking data in families is considered.

4. Statistical investigation is made of palmar formulae of 53 individuals, and left and right hand as well as sex differences are discussed, and racial differences between these and North Borneo natives pointed out.

5. The frequency of ear pits is given and racial comparisons made with other available data.

6. Hair whorl data is given and compared with data obtained from British North Borneo and Germany. An improved method of recording the character and position of the whorl is described.

7. A family chart involving 42 individuals is given showing inheritance of blood groups, hair whorls and ear pits.

I should like to take this opportunity of expressing my sincere thanks to (*a*) Prof. Furuhata, for joining in this work and giving us the benefit of his experience and knowledge, and the pleasure of his company, (*b*) the Superintendent, Dr. Darby, and also the Matron, of the R.W. Large Memorial Hospital, Bella Bella, by whose interest, hospitality and much appreciated help was this work made possible, and (*c*) Dr. Pitts of The Vancouver General Hospital who on very short notice so kindly provided us with all the group sera we needed.

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Clinical Notes.

A Case of Hernial Protrusion of the Great Omentum, appearing in the Anal Orifice, through a Peritoneo-Vaginal Fistula.

By T. K. LIEN (Surgical Unit).

Clinically, one does not often see a case of a hernial protrusion of any the abdominal viscera, prolapsing through the anal canal apart from traumatic injuries. Its true pathological condition and its causation are by no means easy to ascertain before an exploratory laparotomy. In this particular case here, the condition was not attributed to any obvious cause although she had a positive Kahn's test. As such a case when seen by any surgeon should be regarded as a surgical emergency, equally serious as any acute abdominal conditions, I therefore, think, it is perhaps worth recording.

Patient's name P— Y— M—, F. 37, Chinese, U. S. C. No. 98/35. She was admitted into hospital on 1/4/35 at about 10 a.m. by Dr. Thomas and she was transferred to the Surgical Clinic immediately after admission. Operation was performed at 11.30 a.m. Patient died on 5/4/35 at 6.45 a.m.

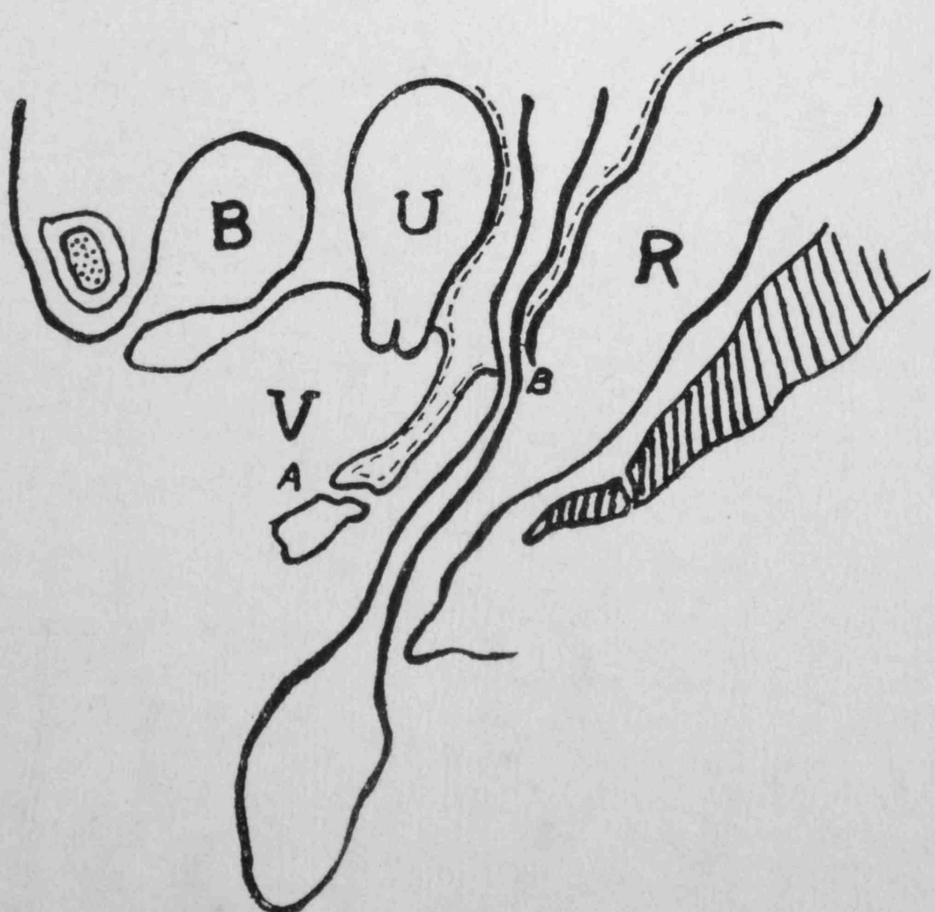

Diagram 1. Rectal examination.

History:

On the 31st. of March at 9 p.m. she went to stool and after that she felt that there was small mass protruding in the anus. It was accompanied by slight bleeding and pain. Towards midnight, the swelling became bigger in size and more pain was experienced, she could not sleep throughout the night on account of the discomfort. On admission, it was found that the prolapsed mass measured 8 inches long, presenting reddish, fleshy appearance but was covered by greenish yellow faeces. It was firm and soft to touch. There was oedema of the lower extremities. Patient looked pale and waxy.

Past-illness:—

She had been having bleeding after defaecation for the past 7—8 years (? piles). She had amenorrhoea for 4 years and also had incontinence of urine for 3 years.

Patient was married at the age of sixteen. Her menstruation started four months after marriage. Husband died 4 years ago. She had never conceived. Menses had been irregular. Her husband contracted both syphilis and gonorrhoea eight years ago.

Physical examinations:—

The lower fistula (A) was about 1 inch above the external sphincter. The upper fistula (B) could just be reached by the examining finger.

Urine:	albumin	—	+ +	
	sugar	—	absent.	
	pus cells	—	present.	
	Red blood cells	—	absent.	
	casts	—	none.	
Blood picture:	Hb %	—	17.	
	total white	—	39,8000	
	differential count:		Polymorphs	95.
			lymphocytes	4.
			mono-nuclears	1.
Kahn's test:	Positive.			
Blood urea:	105 m.gm. per 100 cc.			

Patient was operated upon on 1/4/35 at 11.30 a.m. i.e. one hour after admission. Patient was anaesthetised with warm ether. A sub-umbilical mid-line incision was made. After pushing the intestines away from the pelvic pouch, it was found that a piece of the great omentum passed through an aperture in the pouch of Douglas into the rectum. There was very little faecal contamination around the rent as it was partially sealed. The prolapsed omentum was then divided between clamps from above and the lower portion was then pulled

through from below. The opening was next sutured by close opposition of two adjacent peritoneal surfaces. Abdomen was closed leaving a drain down to the pelvic cavity.

Post-operative Course.

Patient did not pick up well after the operation. She ran a swinging temperature ranging from 101—104°. She complained of much pain in the chest and was dyspnoeic. She passed scanty amount of urine average 8—10 ozs. in 24 hours. Her pulse was rising. She died 3 days after operation. Post mortum was performed on the 5/4/35.

Findings were as follows:—

Post mortem rigidity was present. There was oedema of both legs.

Abdomen.

Free fluid with purulent exudate was seen in the peritoneal cavity. Intestines showed congestion. The fistula in the pouch of Douglas was well closed by stitching peritoneum over it, this fistula communicated with the rectum. Another fistula was found communicating from the bladder to the vagina, (vesico-vaginal) and another one recto-vaginal fistula.

Liver.

Wt. 56½ozs. Enlarged, surface showed slight adhesions of the capsule. Cut surface showed typical "nutmeg" appearance. Gall bladder was distended.

Spleen.

Wt. 6 ozs. appeared normal.

Kidneys.

Rt.—wt. 6¾ ozs. Capsule was non-adherent. Cortex, thin and pyramids slightly enlarged.

Lt.—wt. 8 ozs. Cortex, thin, pyramids were congested. Capsule was adherent.

Suprarenals.

Both were enlarged.

Pancreas.

Wt. 2½ ozs. appeared normal.

Lungs.

Right—very oedematous, middle and lower lobes consolidated and congested. Patchy areas which appeared to be red infarcts being triangular in shape.

Heart.

Increase in pericardial fluid. Fatty infiltration. Valves normal. Wt. 13 ozs.

Brain.

Wt. 47 ozs. No abnormality seen.

Histological Examinations:—

Liver.

Showed chronic venous congestion with advanced fatty degeneration.

Spleen.

No morbid change.

Kidneys.

Right—The glomeruli were irregular in size, some were atrophied, being replaced by hyaline material. There were round cell infiltrations which were more marked in the interstitial tissue. The tubular epithelium exhibited cloudly swelling and in the tubules was hyaline material. The left kidney also presented a very similar picture.

Suprarenals.

Both showed congestion of the cortex with dark brown pigment in them.

Lungs.

Right—The alveoli were filled with cells consisting of polymorphs, lymphocytes and large mononuclears. Several pigment containing histeocytes were also present. The blood vessels were congested. The left lung-alveoli were filled with mucoid secretion. In one area, an early pneumonic patch was seen. Several pigment containing histeocytes were seen.

Fistula wall.

(Vesico vaginal) Showed granulation tissue with numerous new blood vessels, fibroblastic proliferation and lymphocytic and plasma cell infiltration.

Recto-vaginal fistula.

Also showed granulation tissue, covered by stratified squamous epithelium.

Diagnosis:

Lobar pneumonia and chronic parenchymatous nephritis.

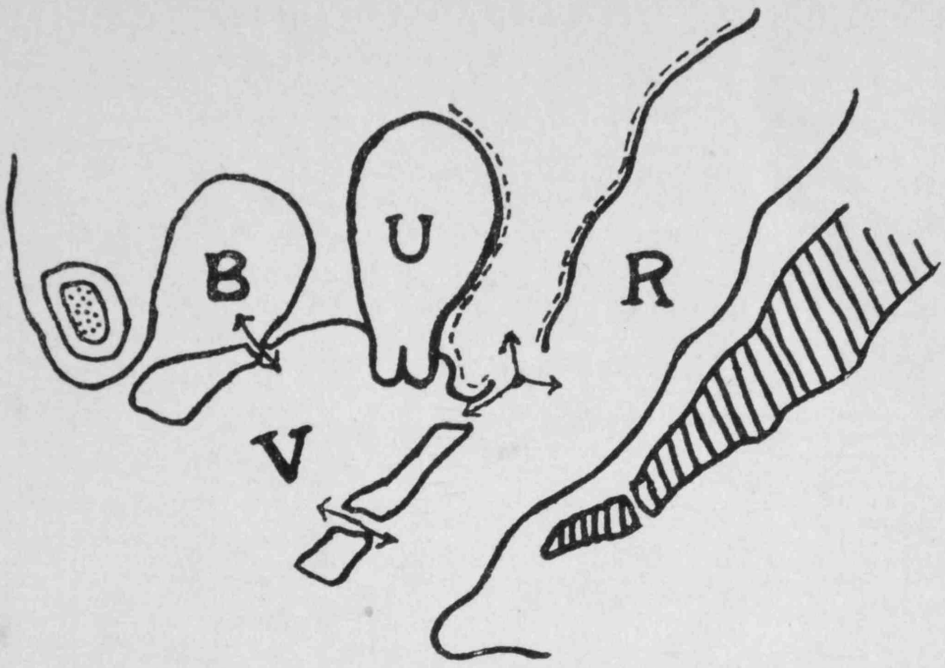

Diagram 2. Showing three fistulae confirmed at post mortum.

It is interesting to note that this patient had had no history of trauma, never had child-birth or any other cause to account for the multiple fistulae formation. One could only suspect that they might be the result of syphilitic ulcerations. The cause of death was evidently two folded; namely, from lobar pneumonia and from diseased kidneys.

Prof. Digby has kindly given permission for publishing this note.

POST-MORTEM NOTES ON A CASE OF HAEMORRHAGIC SMALL POX.

By Dr. K. H. UTTLEY,

Medical Offiier in Charge, Kowloon Public Mortuary.

The following notes on a case of haemorrhagic small pox give details of a condition which, if not very common, is important, and its occurrence has to be borne in mind.

The body of a Chinese male aged 23 years was sent to the Kowloon Public Mortuary during the small pox season with the following history :—

While he was working the previous morning as an earth coolie, he complained to his companions that he was suffering from intense lumbar pains, which soon became severe enough to compel him to cease work. Later on in the morning he developed sensations of heat and cold, (? rigors) and then acquired a generalised red discolouration of the skin. This must have been the prodromal vivid red erythema of the face, limbs and trunk which is often the forerunner of haemorrhagic small pox. He collapsed and died in the afternoon. I was unable to ascertain if there were any further symptoms previous to the day of death.

On examination at the mortuary, the body was that of a well built Chinese male of the coolie type. Rigor mortis was well marked, although the body was still slightly warm. There were the scars of three old vaccination marks on the left arm.

Rash. On the thighs, flexures of the groins, glans penis, trunk, face and neck, front of the arms and hands there was an extensive haemorrhagic rash, confluent in many places, the areas being up to 1½ inches in diameter. No evidence of commencing papulation could be seen.

On incising the skin and muscles, in various parts of the body there were numerous small haemorrhages. There were petechial haemorrhages on all the serous membranes of the body. The lungs were intensely hyperaemic as were the respiratory passages. The liver was a little enlarged, but otherwise normal; the kidneys were normal; the spleen was 20 ounces in weight, firm and dark.

The suprarenals were extensively invaded by haemorrhage, so much so that it was impossible to see any suprarenal tissue. The haemorrhage was limited by the gland capsule.

The other organs were normal.

I have to thank Dr. Greaves of the Bacteriological Institute for making microscope slides of certain of the organs, and for reporting on them. He stated that the suprarenal cortex and medulla showed extensive haemorrhage, largely destroying the parenchyma, especially

in the medulla, very few of the cells had been left in functioning order. There was no cellular reaction present.

The heart muscle showed an intense grade of passive congestion, all the small venous radicles being widely dilated and filled with blood. The lungs were air-holding, there being no exudate in the alveoli, the blood vessels and capillaries being greatly engorged with blood.

The kidneys showed a fair amount of congestion of the vessels. The spleen was normal.

Discussion.

Small pox is a common disease in Hong Kong. To judge by the mortuary findings, the fatal forms, which are mainly of the confluent type, occur much more frequently among infants and small children than among adults. The haemorrhagic form is distinctly rare, this case being the first that I have seen at the Mortuary for four years.

The distribution of the purpuric rash, especially in the groins, is typical of the haemorrhagic type of small pox.

The presence of vaccination scars usually renders the prognosis favourable if a patient falls a victim to an attack of the desease, but in this connection it should be remembered that the prophylactic value of infantile vaccination disappears to a large extent after the age of 15 years. This man was 23.

The available history confirms the rapid onset and course of haemorrhagic small pox.

Summary. A case of haemorrhagic small pox is described, as met with in the mortuary. A noteworthy feature was an extensive haemorrhage into the suprarenal glands.

Review of Books

AIDS TO SURGERY. Joll & Ledlie. Sixth Edition. Balliere Tindal & Cox, London.

The fifth edition of this excellent little manual appeared first in 1924. It was therefore quite time for revision and bringing up to date. This renovation appears to have been well carried out.

Forty-four clear line-diagrams by Mr. H. H. Greenwood have been introduced and add to the attractiveness of the book.

The reviewer ventures on a few criticisms.

Surely the old confusing names for hydrocele or hydrocaele (as this work pedantically has it)—idiopathic, infantile, encysted, congenital could now be dropped and replaced by a scientific terminology on an anatomical basis? The same applies to the naming of the varieties of oblique inguinal hernia. The chapter on affections of the ear caused the reviewer to give one or two dissentient gasps. Thus (p. 313) "syringing of the auditory meatus" and "inflation of the Eustachian tube by Politzer's method or the passage of a Eustachian catheter" are recommended for the treatment of chronic supperative otitis media. There is no mention of radiography of the mastoid process in chronic otorrhoea.

The B. N. A. terminology is not employed.

The treatment recommended for transcervical and intertrochanteric fractures of the femur does not appeal to us. There is no mention of Peterson's Nail operation or its later modifications in the hands of others.

Notwithstanding such criticisms this very handy little book is certain to appeal strongly to the student who is revising his surgical work, and on the whole, this popularity will be deserved.

K. H. D.

POCKET MEDICAL DICTIONARY. By LOIS OAKES. Assisted by T. B. Davie. E. & S. Livingstone. Edinburgh, 1935. Price 3/-

There are a number of interesting and valuable points about this small book. It's size is really what it claims to be, a pocket book; the amount of information given is amazing, and the addition of a large number of diagrams increases greatly its usefulness; besides the dictionary proper a large amount of information on many subjects is assembled in tables which make the book border on a medical pocket encyclopaedia; it is well bound so that it should stand the wear and tear of a pocket or hand bag. In fact it is suited in more ways than one to the pocket and needs of the student of medicine.

L. T. R.

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