THE ROLE OF THE Rh BLOOD FACTOR IN THE ETIOLOGY OF STUTTERING, SPASTIC SPEECH, APHASIA AND DELAYED SPEECH

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CHAPTER I

INTRODUCTION – THE INCENTIVES FOR THIS STUDY

For several years preceding this study the examiner worked With children who were not up to the normal development of Speech. During that time, a similarity in the case and family Histories of delayed speech and stuttering cases was noticed – A history including such symptoms as anemia, jaundice, rickets, Poor deciduous teeth, feeding problem, lack of strength, list – Lessens, predisposition to respiratory infections, nervous Afflictions, and cyanosis at birth or in the neonatal life. Sometimes the indisposition was still present. Another Similarity was also noticed. Usually, the mother said the child Was bottle fed. No record was kept of the exact answers, not Of the reasons for the bottle feeding: in fact, the examiner felt that the reason for the difficulty with speech might possibly be lack of muscular strength and coordination which would have been gained by the sucking process in breast feeding, It was thought that training in muscular strength and coordination might be less in bottle feeding than in breast feeding. Although no two children had exactly the same expression, they All showed a peculiar resemblance in a set, almost immobile Facial expression, especially around the mouth, and an arrhythmic Breathing pattern. At the University of Wisconsin speech Clinic it was noticed again that stutterers and children with delayed

speech had a sluggish, immobile expression in the region of the mouth; and an arrhythmic breathing pattern; and mothers gave the same general histories. In response to the question of breast or bottle feeding, their answer was almost generally “bottle fed”. Dr. West agreed that the nursing experience of the child entered into the problem, but believed that the process of bottle feeding alone could not be the cause and that possibly substances peculiar to the mother’s milk might be responsible. Since it was discovered that in cases of Rh in-compatibility of pregnancy. Antibodies were in breast milk. It seemed that a study of these factors and their relation to speech development would be profitable.

The first procedure in the attempt to discover the relation Of bottle feeding to speech defects was to visit each room, from Kindergarten through the sixth grade, of one of the public Schools of Ft. Atkinson, Wisconsin. The pupils did not know They were being tested. While the teacher called on each pupil By name to read and speak, the examiner took down the names of Children thought to have been bottle fed, the judgment being made on the basis of facial expression, breathing and speaking. The classroom teacher and previously listed the ones she considered “speech cases”; and it was found that the lists of the teacher and the examiner were in remarkable agreement. The nurse was to get certain information in regard to infant feeding and health of the children selected and also of a control group. This control group was chosen by the teachers and the examiner, and was composed of the children (in the same rooms).

whose speech was considered the best. Again, the criteria used by the examiner were facial expression, breathing and Speaking.

At a later date, the examiner returned to the Ft. Atkinson Schools with apparatus designed to test the comparative abilities Of the two groups to use the chewing, sucking and swallowing mechanism. The apparatus is not reported since at the completion of the experiment the examiner decided that, though there was a great discrepancy between the two groups in the use of the mechanism for chewing, sucking, and swallowing, the results had no bearing on the examiner's immediate study. The study group tended to have an unrhythmic hypertonicity of ceratin muscles, especially those of the mouth and face, and unrhythmic breath control. The control group tended to have pliable mouth and face muscles, and rhythmic breath control. Although in the early grade levels the latter group had some difficulty with the apparatus, the children became more accurate and forceful in its use as age advanced; however in the former group all at great difficulty as to accuracy, strength and rhythm in the use of the apparatus. In the former group there was some improvement with age, but the degree of improvement was less than that in the control group and some children in the upper grades were never able to develop rhythm and strength in the use of the apparatus. The majority of the first group tended to breathe in and then try to use the apparatus while holding the Breath. Before information could be obtained in regard to infant feeding and health, the nurse at Ft. Atkinson had a prolonged illness and it was decided some other study must be made to de-

termine the relationship between bottle feeding and speech.

Since the distinguishing characteristic seemed to be something different in body contour (especially of the face),Somato typing was investigated; but it was seen that the somatotyping, like the test attempted at Ft. Atkinson, would merely show a superficial characteristic of stuttering and delayed speech.

The clue to the area for investigation of the “clause” of Stuttering and delayed speech came one Thursday afternoon when Dr. West interviewed Dr. Guyer on the “Our Children” program Of the Wisconsin School of the Air on W.H.A. The Rh factor In the blood was mentioned in such a manner as to suggest that It might be the “missing link”. After further investigation, It was decided to make a study of the etiology of shuttering And delayed speech on the basis of their relation to the Rh Factor in the blood and bottle feeding. The idea occurred that cleft palate might also be caused by the Rh incompatibility causing, not an incomplete synergy of muscle, but, incomplete union. Information in regard to cleft palate could easily be obtained as the state of Wisconsin has a special cleft palate program and a clinic each summer.

For the delayed speech and stuttering cases, cooperation Was requested of those who come to the University of Wisconsin Speech Clinic. All who volunteered to cooperate were asked to Fill out the following blank.

NAME:_____AGE:_____

ADRESS:_____

BIRTH DATE:_____

I. Child's History:

How long was the child breast fed? _____

How long was the child bottle fed? _____

If bottle fed, Why? _____

At what age did he (she) learn to walk _____, talk _____, eat _____, toilet habits _____?

II. History of pregnancies:

was the term of pregnancy normal _____, difficult _____?

Was delivery normal _____, caesarean _____, instrument_____, premature _____?

Was there trouble of any kind with any of your pregnancies Or at birth of any of your children, such as:

Anemia _____ stillbirths _____

Jaundice _____ Miscarriages _____

Dropsy _____ Transfusions for Mother _____

If any of these conditions were in a pregnancy from a Previous Marriage, indicate which ones.

III. Family History:

In the family history are there:

P R E S E N T F A M I L Y	A N C E S T O R S (Both sides of family as much as is known)
Stutteres _____	_____
Mongols _____	_____
Cleft palates _____	_____
Left hander's _____	_____
Children with delayed speech	_____
Erythroblast sis _____	

III. (Continued)

P R E S E N T F A M I L Y	A N C E S T O R S (Both sides of family as much as is known)
Missing toes or fingers	
Twins	
Sinus	
Allergies	
Asthma	
Migraine Headaches	

IV. Nationality:

I should like to known as accurately as possible single Nationality or combination of nationalities.

Father		Mother	
Father	Mother	Father	Mother

V.

1. Are the grand parents available and are they willing to give blood samples if needed (same as those requested in No. 2 below)?

2. Are you and your wife and child willing to give a Blood sample for an Rh test in studying the conditions Causing such speech difficulties?

Parent’s signature _____

Date _____

No. V-2 of the blank says,

> “Are you and your wife and child willing to give a blood sample for an Rh test in studying the conditions causing such speech difficulties?”

Dr. Stovall of the State Laboratory of Hygiene consented though Have his laboratory make 100 tests. This limited the number of cases studied since three tests were needed for each individual studied. The results from the testing of such a small group were not to be considered as conclusive evidence, but as an indication of whether or not there would be any areas for further study of blood incompatibility of pregnancy in the phenomena of the inheritance of the etiologies of the types of cases studied.

Since the plan for doing the blood testing proved to be very unsatisfactory, the study resolved itself into a critical Study of the literature. As the literature was studied, it Was seen spastic speech and aphasia should be included, and that Cleft palate, while not being ruled out as having blood in-Compatibility of pregnancy related to its etiology, should be a different study. The findings in regard to cleft palate, however, will be summarized at the end of Chapter VI.

The Rh blood factor has definitely been related to pregnancy and neonatal life (48,52,151,171,173,177,227,269, 327) and the speech pattern is definitely set, if not during pregnancy, during the neonatal period. Therefore, in discussing the incompatibility of the Rh factor as related to pregnancy and the development of speech, the pathologies and symptomatologies as shown by the fetus and infant are the main items of interest. In 1940 a blood incompatibility was discovered

which, in pregnancy, caused erythroblastotic fetalis (151)and this erythroblastotic fetalis is related to another pathology ong known to be related to pregnancy – nuclearicterus (kernicterus). However, These are not the only pathologies related by investigators.

A review of the literature up to the present date shows a great discrepancy in the nomenclature, (74,140,197,234) pathology and symptomatology of Rh incompatibility of pregnancy. Erythroblastotic fetalis, as first attributed to the Rh in-Compatibility, designated a disease in which there was an hemolysis of the red blood cells accompanied or unaccompanied by interus and/or anemia. (36,62,135,315) The child (310)was born dead, died immediately or within a few days after birth. (69B) Some researchers still hold to this nomenclature which makes erythroblastotic fetalis a bread designation of several proven pathologies – namely,hemolytic anemia of the newborn, icterus gravis neonatorum, jaundice, kernicterus, splenomagely and hepatomagely and other symptomatologies and pathologies over which there are still controversies. By others erythroblastotic fetalis has been classified as severe (kernicterus), moderate (icterus gravis neonatroum), and mild (hemolyticanemic of the newborn). (8,155,259) Some consider it a distinction without a difference to make any division, and consider that all pathologies involving hemolysis of the red blood cells in the fetus and newborn where incompatibility of the blood is shown constitute the same hazard. Macklin (197) and others

think that when clarification of terminology is established, when the Rh and other atypical agglutinins have been segregated and clarified, and when pathologic finds have been standardized, it will result in the conclusions that erythroblast sis fetal is as a disease entity includes all endogenous acute hemolyticanemias of the newborn with or without erythroblast sis. Wiener(342) thinks the distinction is important because he thinks the different symptoms are cause by different incompatibilities and that the different evident symptoms show a need of different treatment of the pathology as found.

For the purpose of speech research, a definite classify-cation is not needed as it is out of the scope of this study to classify symptoms, etiologies and pathologies. The facts important in this correlation are:

1. That the etiology of the symptom is related to in-Compatibility of blood between mother and fetus.

2. That the symptom has familial characteristics which have been associated with the etiologies of stuttering, spastic speech, aphasia and/or delayed speech.

3. That the symptom and its etiology have been associated With defective speech development as demonstrated in Stuttering, spastic speech, aphasia and/or delayed speech.

A good name to use is <u>blood incompatibility of the newborn</u>, and this term will be used to designate any and all symptoms as shown related to such incompatibility. In the discussion, it will be assumed that reference is to speech cases which have unaccountable or unconvincing etiologies as it is well known there are many causes of aphasia and spastic speech and there

may be other causes of stuttering and delayed speech. Just so, neonatal hemolysis of the blood may be caused by congenital syphilis, ingestion of certain drugs, etc. (11B) as well as by blood incompatibility. This study is an attempt to find any existing relation between blood incompatibility and the causes of those speech disorders which have unknown etiologies or etiologies which are not obvious and completely convincing. In order to search for these relationships, the literature in Regard to blood incompatibility will be reviewed and critically compared with the literature in regard to stuttering, spastic speech, aphasia and delayed speech. Since the literature in regard to the speech disorders has been competently reviewed by others. their studies will be taken as bases upon which to make the comparisons.

CHAPTER II

THE RH BLOOD FACTOR – ITS DISCOVERY AND RELATION TO PREGNANCY

To understand clearly the comparisons being studied, a Brief review of the discovery of the Rh blood factor must be given.

The Discovery

Hundreds of years ago blood transfusions were made from Animal to man; but as there were as many deaths attributable To the operation, in 1675 a papal bull banned the performance. (36B) In 1818, an English obstetrician, James Blundell, reasoned that, since transfusion from animal to man was fatal but from animal to animal was not, transfusion from man to man should not be. He, therefore, used intra species transfusions in pregnancies. (36B) Fatal results from such transfusions caused Landois and Bordet to study the specificity of blood in the same species and it was found that blood transfused between the same species was usually satisfactory but between different species was fatal. (69B) There were many unaccountable fatalities from transfusions in the same species, however, until, in 1900, three important events occurred. (1.) The Mendelian laws of heredity, which had been unnoticed when Published thirty-five years before, were rediscovered. (61B) (2) Isoimmunization was demonstrated in goats. (61B) (3)The A-B-O blood groups were discovered by Landsteiner. (69B) Since that time, there have been discovered the M-N-P factors

and subdivisions of groups and factors. (69B) The substances, Which characterize the four A-B-O blood groups and M-N-p Factors, are present, not only in the red blood cells, but in almost all the tissues and body fluids of 80% of the individuals who are known as secretors. (61B) Those who have the sub stances confined to the red blood cells are called non- secretors. (61B – 167) But with all this knowledge of the capabilities and incapability of the blood to combine, there were still unaccountable, untoward reactions in blood trans-fusions. (167)

Then in 1940 Landsteiner and Wiener (151) discovered a Factor in the blood of the rhesus monkey unrelated to the A-B –O blood groups and M-N-P factors accountable for at least some of this untoward reaction. This factor they called Rh because it was first discovered in the blood of the rhesus monkey. Back as far as 1936, Zacho attributed the reaction encountered in transfusion in pregnancy and the post-partum period to an abnormal agglutinin which he found in the blood of the patient, (58) Levine and Stetson (177) postulated, in 1939, that the antigenic substance causing the formation of the agglutinins, which Zacho had found, was due to some fetal product. Before Rh studies were made, Darrow (52) said hepatio changes in the Newborn might be explained by an antigen-antibody reaction due To trans-placental immunization in the mother. Reisner (259) had maintained that hydrops, icterus gravis neonatorum and Congenital anemia of the newborn represented severe, moderate And mild antigen antibody reaction. (52,155,259) A review

of the literature (21,43,103,110,114,229,227) showed that the unaccountable reaction occurred usually in women who were or who had been pregnant or who had had blood transfusions. (13,30,216,37) The Rh antibody in those women who had been or were then pregnant or had had a transfusion of blood of a homologous blood group produced agglutination and hemolysis.(345) Levine and Katzin also observed that those women who had intra-group transfusion reaction because of isoimmunization in pregnancy gave obstetrical histories of spontaneous abortions, miscarriages, stillbirths and neonatal deaths. (229) In this Way it was shown that the reaction between mother and fetus, Which Zacho, Darrow, Reisner and Levine and Stetson and suggested as taking place, was caused by the incomparability of the blood that Landsteiner and Wiener had discovered – namely the Rh factor. (48,52,150, 173,269,327) This reaction was associated with erythro-blastosis in infants born of mothers whose bloods proved to be Rh negative. (170,173,178)

Putting all these facts and theories together Levine (157,161,170,175) in 1941 worked out a hypothesis to explain the reaction found in pregnancy. That theory is: If the mother is Rh negative and the fetus Rh positive, inheriting from the father the dominant antigen which is absent in the mother’s blood, and if due, probably, to some defect in the placenta, (114,347) the fetal blood escapes into the mother’s circula-tion, the production of anti-Rh agglutinins may be stimulated in sensitive mothers. Since only minute quantities of blood

are required, these antibodies may form, even though the placental lesion is microscopic. (173) It is normal for proteins to filter though the placenta from the maternal circulation to the fetal circulation; therefore, the protein Rh antibodies, set up in the mother because of the abnormal passage from the fetus to the mother, pass back into the fetal blood. (69B,26,161,173,347) If the concentration in the fetal circulation is suitable, an hemolysis of the fetal red blood corpuscles may occur which is likely to cause a hemolytic fatal or congenital disease. (17,22,26,45,113,344) If antibodies are once produced by the mother, either as a result of transfusion or of incompatibility of pregnancy, the sensitivity lasts ---- sometimes for years. (59,146,297, 323,354) Even though antibodies are not always found in the blood, in-Compatibility can sometimes be shown. (180,333) All of this must remain theory since human lives cannot be sacrificed in experiment; however, as the reasoning behind the hypothesis seems to have a strong physiological and statistical basis,(32) it may be accepted as a workable theory to be modified as later statistics are presented.

Occurrence and Inheritance of the Rh Factor

Unrelated to any other known characteristic of human blood, the Rh factor appears to be inherited as a Mendelian dominant not sex linked, (150,344) transmitted by two allelic genes,

Rh positive dominant and Rh negative recessive. (150,318)It was originally thought not to be sex influenced, but Halparin, (109) Wiener, (350) et al think probably there must Be a search for a sex influencing factor). Of the white Population of the United States, about 85% of the individuals Have this agglutinogen in their blood and the remaining 15% do not have it. (17,18,66,150,151,164,345) Potter Compailed figures based on studies made in different countries. (238)

Incidence of Rh

Rh+	Rh-	Country	Source of Testing Serum	Tester
85.15	14.85	England	Human	Boorman et al
84.6	15.4	Wales	Animal	Hoare
84.0*	16.0	Germany	Animal	Dahr
86.6	13.4	United States	Human	Levine
86.6	14.4	United States	Human and Animal	Wiener
85.0*	15.0	United States	Animal	Fisk and Foord
83.0*	17.0	United States	Animal	Gallangher and Jones
85.0*	15.0	United States	Human	Gallangher and Jones
84.2	15.8	Total		

*Potter calculated from author’s data.

As the comparative strength of the sera is not known, any comparisons made would not be without inaccuracies.

As shown in the tables, the animal serum seems never to test as

high as the human serum. However, the average of all seems to run close to 85% Rh+ and 15% Rh-. (87% and 13% are considered accurate by some with the strongest sera.) (261) of the 85% having the Rh agglutinogen, approximately 37% will be homozygous and 48% heterozygous. The frequency of the occurrence of the Rh+ gene is approximately 39% and the frequency of the occurrence Of the Rh- gene is approximately 61%. In random mating, the Calculated frequency of mating of Rh- women with Rh+ men is Approximately 13%; with homozygous Rh+ men the frequency is 5.6% And with heterozygous Rh+ men 7.4%. The frequency of random Mating between Rh+ men and Rh- Women is approximately 75.7%; And between Rh- men and Rh- women approximately 1.7%. (288)

Scheme of the 8 Rh Blood Types

Clinically Rh- individuals (15%)				Clinically Rh+ individuals (85%)			
Designation of Types	Reaction with antiserum			Designation of Types	Reaction with antiserum		
	Rh'	Rh''	Rh^o		Rh'	Rh''	Rh^o
Rh	-	-	-	Rh^o	-	-	+
Rh'	+	-	-	$Rh^1(Rh^{o'})$	+	-	+
Rh''	-	+	-	$Rh^2(Rh^{o''})$	-	+	+
$Rh' Rh''$	+	+	-	$Rh_1Rh_2\ (Rh^o)$	+	+	+

The above table is explained as follows: of the three varieties of sera used at the present time, (150, 197, 256, 325, 332,49) the one corresponding to the original anti Rh serum, which re-acts with approximately 85% of the blood of the Caucasian ace, has been called anti Rh^0 or the standard anti Rh serum. (325,

256, 254, 332, 336, 349) The one which reacts with 70% has been called anti Rh′ (17, 256, 325, 332, 336, 349) and the one which reacts with 30% has been called anti Rh″. (197, 256, 307, 325, 332, 336, 349) These three anti sera detect the presence or absence of three corresponding Rh factors, Rh^0, Rh′ and Rh″. Rh^0, which is by far the most antigenic factor and therefore the most important clinically, (333) divides blood into + and – (really Rh^o+ and Rh^o-). This is called Rh testing. (698, 169, 325, 336)Rh′ and Rh″ are responsible in only a small percent of the clinical cases.) (336, 333)

AS anti Rh′ and Rh″ are related like anti A and anti B, tests made with them differentiate four types of blood analogous serologically and genetically to the four blood groups. Since every individual is either Rh^o+ or Rh^o-, each of the four types may be further subdivided giving the eight types in the table above. This is called Rh typing. (171, 328)

The names of the types are determined by the antisera with which the blood reacts. (235, 328) Rh^o blood is so called because it reacts with anti Rh^o serum but not with anti Rh′ and anti Rh″. The type which does not react with any anti Rh serum, called rh, is not identical with Rh- blood, Rh- refers to the blood which gives a negative reaction when tested with standard anti Rh (anti Rh^o) serum alone. This blood which for clinical purposes is Rh- (Rh^o-), actually includes four types: rh, Rh′, Rh″ and Rh′Rh″.Universal recipients would be those bloods which test Rh-; but universal donors would be those bloods of type rh because if Rh- were used, types Rh′ and Rh″ might

cause reactions. The designation Rh_1 ($Rh^{o\prime}$)and Rh_2 ($Rh^{o\prime\prime}$) show that Rh′ and Rh″ combine with Rh^o respectively to form the two types. (328)

The following table made up from information Synder (278) and from Wiener, Sonn and Belkin gives a summarized (350, 351) story of the Rh factors as founds in the blood.

Rh Factor

Type	**Frequency%**	**Antigen**	**Antibodies for**	**Genotypes**
Rh^o	2.4	Rh^o	Rh′Rh″	Rh Rh Rh rh
$Rh_1(Rh^{o\prime})$	54.3	Rh^o, Rh′	Rh″	Rh_1Rh_1, Rh_1rh, Rh_1Rh, Rh_1Rh′ Rh′Rh
$Rh_2(Rh^{o\prime\prime})$	13.7	Rh^o,Rh″	Rh′	Rh_2Rh_2, Rh_2rh, Rh_2Rh, Rh_2Rh″, Rh″Rh
Rh_1Rh_2 ($Rh^{o\prime}$Rh″)	13.2	Rh^o,Rh′, Rh″	None	Rh_1Rh_2 Rh_1Rh″
Rh-	13.3	None	Rh^o,Rh′ Rh″	Rhrh
Rh′	0.5	Rh′	Rh^o,Rh″	Rh′Rh′ Rh′rh
Rh″	0.2	Rh″	Rh″, Rh′	Rh″Rh″ Rh″rh
Rh′rh″	000	Rh′,Rh″	Rh^o	Rh′Rh″
Rh(not a type	none	none	none	none

(From Snyder & W-Sonn & Belkin)

The Percentage of Evident Incompatibility

At times there have been doubts as to Verity of Rh

incompatibility because the evident percentage of incompatibility was not equal to the expected percentage. Although, as said before, about 13% of all families (280) offer opportunity for isoimmunization, only about 1 in 200 births has been shown to have any symptoms resulting from incompatibility. (278) Both Javert (132, 133) and Potter, (238) in 1942 and 1943, set the figure at 1 in 400 but they were considering the classical symptoms as the only results of such incompatibility. (Potter (240) found the incidence in births subsequent to the first to be 1 in 245.) Levine (163) in 1942 set the figure at 1 in 400, but said “milder forms may have been overlooked.” And then Snyder (278) and Yannet, (363) by studies among the feeble minded, changed the established incidence to 1 in 200 births. As the cases of stuttering, Spastic speech, aphasia and delayed speech, and as there are so many evidences of “sub-clinical” symptoms, could it bee that disorders of speech which cannot be demonstrated in neonatal life are the only results of blood incompatibility of the newborn in some instances? Crothers (158) says of the human body “a good deal of damage, some of which may be permanent, can exist without modifying behavior in early infancy”.

Snyder says (278) that in about 23% of all births the infant carriers some antigen which is absent in the mother’s blood. But since only Rh^{o} seems to be important clinically and the incidence of an Rh- mother carrying an Rh+ child is

approximately 9%,(107, 241, 278) and since the proved incidence of Rh incompatibility is approximately 0.5%, there is a great discrepancy between the possible occurrence and the proved incidence. (162, 278) This discrepancy has been accounted for in several ways:

1. Only the 9% mentioned above are clinically significant. (278)

2. First children are seldom affected, probably because it takes more than one pregnancy for a mother to build up antibodies she has had a previous transfusion of incompatible blood. Potter (240) found the incidence of erythroblastosis to be 1 to 2380 in first births and 1 to 245 in subsequent births. In the American population about 31% of all births are first born. This would reduce the expected incidence from 9% to 6%.(107)

3. Some mothers do not seem to have the ability to produce antibodies; (246) or, in some mothers, the antigen does not permeate the placenta. This is indicated by the fact that there seems to be a tendency for the incompatibility to be grouped in families. (325)

4. Some infants do not seem to be affected by antibodies. (325)

5. the effects on the fetus are different from those usually recognized as classical (erythroblastosis). (103, 245, 254, 278, 324)

6. Race and Taylor think the majority of the fathers of erythroblastic infants are homozygous and only about 43% of the individuals are homozygous. (254)

In addition there would be a reduction in the discrepancy if the findings of Yannet’s (363, 364, 365, 366) and Snyder’s (598, 278, 279) studies of atypical results and the findings of Levine’s (162) study of full term births were related to the other studies. But, as can seen, there is a discrepancy for which some account must be given.

Also there are many discrepancies in the report of re-action. Among the main causes of differences in evidence of

reaction which have been considered by investigators are:

1. Other types than Rh^o may be more important clinically than has been thought. (11, 103, 170, 80, 109)
2. Wiener terms as “intermediate” those undiscovered factors which account for the reaction not traceable to any of the know factors. (68, 698, 251, 333)
3. “Blocking” antibodies which Wiener thinks are univalent instead of bivalent, like Rh agglutinins, complicate the picture causing only 50% of Rh- mothers to show agglutinins. (333) However, the effect of the reaction is the same as if the agglutinins could be demonstrated (251)
4. Factors in the blood other than Rh may be incompatible. Incompatibilities of blood groups in pregnancies called heterospecific pregnancies have been seen to occur mainly between A_1 and A_2 sub groups of A; but have been seen to occur also between groups 0 and A. (55, 103, 106, 140, 193, 196, 236, 237, 243, 340)
5. The Rh antigenic property is active at 37^0C while the A-B-0 is active at 20^0C. Therefore, the former is called a “warm” agglutinin and the latter a “cold” agglutinin. (146, 174)
6. The blood group substances are water soluble in 80% of the people (called secretors) and are, as a results, found in all body tissues and fluids of such people. In the remaining 20% (called “non secretors”), the quality is transmitted as a Mendelian recessive gone. (166, 174) At first it was thought the Rh agglutinogen was confined to the red blood cells but Boorman (19) found it in the liver, spleen, kidney and salivary glands though it is not very water soluble. (309) (Because of Boorman’s findings, Potter (243) disagrees with Levine’s theory of “non-secretors”).
7. Levine (388) and Halparin (109) think there is probably an inherited influencing factor, which might be at least partially sex linked, causing blood incompatibility to be grouped in families.
8. Some maternal organizations are incapable of producing antibodies. (246)
9. There may be effects different from the classical picture of erythroblastosis, anemia of the newborn and icterus gravis neonatorum since any tissue of which the antigenic substance is a protoplasmic content, may be affected by the antibody.(163, 278)
10. Effects may be mild or unrecognized. (37, 109, 161, 163)

11. Isoimmunization may develop without producing signs of erythroblastosis in the infant but sufficient to produce anti Rh agglutinins in the mother's serum. (2, 73, 212, 292)

12. If the mother has, in the past, had a transfusion or injection of incompatible blood, the result of the incompatibility is more severe than it would otherwise have been. It is often severe enough to produce an effect in a first pregnancy. Levine (157) thinks the neonatal injection of blood in the past has been very common.

These twelve modifications in reaction are considered as the influencing factors which produce various effects and degrees of the disease. And these same influencing factors are the possible links between blood incompatibility of the newborn and speech disorders. The reports of blood incompatibility vary as to symptoms just as reports of speech cases vary as to symptoms; but through them both run similar threads. It would be an impossibility to quote cases that "match" because there are none, but a review of reports will give an impression of similarity to speech cases. The study of blood incompatibility is too "new" to have definite classifications of symptoms and pathologies except in the "classical" (erythroblastosis) picture. Since the hypothesis of this study is that the speech problems may be found in the cases having symptoms and pathologies which are not classical, those "similar threads" must be watched. It would be impossible to give references for each similarity noticed but a very complete bibliography will be listed as a final chapter. Just as anyone wishing to realize the beauties of nature must do so "first-hand", anyone wishing to realize the likeness of the pathologies of blood incompatibilities of the newborn to the pathologies related to

speech disorders must review the literature “firsthand”.

The Ten percent Atypical Reactions

It is seen, then, that there are several factors affecting blood incompatibility reaction and the evidence of blood incompatibility reaction in addition to the Rh+ and Rh- reaction.

The following factors may fill a great part of the gap between observed and expected reaction and will be the ones considered in this study. All or some of them may cause the incompatibility to have a “mild” effect and go unrecognized.

1) The sub factors of Rh.
2) A B O blood groups and factors.
3) The Hr factor.
4) The “non secretor” recessive gene.
5) “Intermediate” factors.
6) Blocking antibodies.
7) “Warm” end “cold” agglutinins.
8) Inherited influencing factor.
9) Mildness of effect.
10) Lack of demonstration of incompatibility.
11) Other effects than the classical ones.
12) Familial indications:
    a. Some maternal organizations are incapable of forming antibodies.
    b. In some pregnancies antigens do not enter the maternal circulation.
    c. Some infants are not affected by antibodies.

The statement “while 15% of all white women in this country are Rh-, of the women giving birth to erythroblast infants 90% are Rh-“, (246) has been upheld by other (160, 305, 323, 340) investigators. The other 10% are Rh+ (32, 32, 45, 163, 170, 176, 268, 315) Potter (240) thinks Schwartz and Levine’s (268) figure substantiating this incidence are unfounded because they assumed that if the child died and the

mother was Rh-, the child could be classed as erythroblast due to incompatibility. It seems that not only the above mentioned but most of the other studies of Rh have left out some **essential factor** needed to give a **complete picture** of evidence of blood incompatibility, theory producing one picture in one study and another picture in another study. If all the studies considered all factors, probably the reported results would be more nearly the and same of the discrepancy between observed and expected incidence would be accounted for.

According to Levine's original theory of isoimmunuzation, as given in the first of this chapter, erythroblastosis could not occur in an Rh- infant born of an Rh+ mother and heterozygous rh+ father, but such reaction has been found. (17, 19, 32, 89, 158, 137, 163, 170, 245, 323, 324) (234)

As previously stated, approximately 90% of all mothers of erythroblastosis infants are Rh- in tests with standard Rh serum and the infants are Rh+. It is supposed that in the cases where the infant is Rh- antibodies are not formed because infants produce antibodies very poorly. Evidence of isoimmunization in the remaining 10%of the mothers who are Rh+ may involve at least one of three different blood substances. Sc:

1. Properties of A and B fetal blood – "non secretor" type.
2. Hr factor.
3. Finer divisions of Rh factor.

Properties of A and B Fetal Blood

Ever since blood groups have been known, it has been thought heterospecific pregnancy (a pregnancy in which mother and infant are of different blood groups and the infant is a “non secretor”.) might be responsible for pathologic conditions of mother and child at birth. (698, 201, 224) Dienst, in 1905, was the first person to recognize that when a mother’s blood contained an Alpha or beta agglutinin incompatible with the fetal cells, (7) the agglutinin titer of her cells was frequently high for four to eight days after delivery. He thought the rise was from antigenic action of fetal cells which had gained entrance to the maternal circulation and the cause of eclampsia. Since the possibility has been discussed freely. (1, 2, 16, 40, 42, 52, 62, 63, 90, 110, 201, 224, 340,345) Darrow (52) postulated a difference in fetus through the placenta and after birth through the breast milk. She and co-workers reported an antiserum which differentiated fetal and adult Hb. There were other similar studies made, but the subject was dropped because no proof of isoimmunization could be found. According to the theory of heterospecific pregnancy, the infant may be affected by antibodies that react with: (32, 166, 235, 238, 266)

1. Blood of sub group A_1 and A_1B.
2. All groups 0 blood and less intensely with group A_2 bloods.
3. All p negative bloods.
4. Bloods containing unclassified irregular agglutinogens.

This heterospecific pregnancy, which can only occur if the infant is a “non secretor”, is seen about 20 in every 100 pregnancies. (32) Levine (160) thinks there is a higher incidence of abortions and fetal deaths of unknown cause in heterospecific pregnancies than normal. However, Potter (238) does not think this is the case. Also stillbirths, not due to erythroblastosis, may still be due to incompatibility because there is a contrasting distribution of Rh and AB, A or B between the mother and a “non secretor” fetus. (32, 161, 166, 235, 238) (If the fetus is a secretor, it is supposed the deposit of agglutinogen in the tissues and fluids of the body acts as a “buffer” preventing reaction from taking place.)

Earlier in this chapter it was stated that certain atypical affects would be reported in this study. The consideration turns now to references which give reason for some of this “sub-clinical” consideration.

Polayes (236) reports nine cases in which children were group A and mothers group 0 and neither Rh nor Hr was responsible for the erythroblastisis. Kennedy (140) reports cases of irregular isoimmunization and adds in his discussion of the disease “those that survive may show brain deterioration due to kernicterus”. “In 95% of the cases of icterus precox there is incompatibility of blood groups.” (106) Gurevitch (103) maintains isoimmunization in cases of incompatible blood groups is possible, but thinks chances that it might be responsible for a hemolytic process in the child are indeed small; because,

since heterospecific pregnancy is so common an occurrence, if it could produce hemolytic effects in newborn, a greater incidence would be found. Smith (277) and Toney (298) find no evidence of harmful effects to children in cases of heterospecific pregnancy with signs of isoimmunization. Boorman (19) et al observed the titer of A and B antibodies rose highest 10 to 20 after delivery. The theory of heterospecific pregnancy may explain a few instances of incompatibility reaction found in Rh+ mothers; i. e., those in whom the fetus belongs to the non-secretor type as: Mother 0 Rh+ and infant A Rh- non-secretor type. (“Non-secretor” is inherited as a Mendelian recessive.) The mother’s blood could be isoimmunized and act on susceptible blood of the fetus because of absence of group. A substance in fetal tissues and fluids. (110, 166, 238) Boorman (17) makes the statement; “agglutinogens in secretors are able to absorb incompatible agglutinins crossing the placental barrier.” However, if any tissue of which the substance is a protoplasmic content may be damaged, those “buffers” may be damaged. (698) Potter (238) says this cannot be true because it has been shown that when A and B substances are not shown by ordinary test, they can be shown with alcohol and that the Rh substance is there, but not very water soluble. Wiener and Peters (345) say “there is as yet some un-described qualitative difference among the irregular antibodies”.

Thus it is seen that the intra-group incompatibility has been considered by many researchers, but the reports have not always been in agreement. (26, 32, 45, 60, 66, 150, 164, 170, 310, 313, 314, 323, 345)

The Hr Factor

The second blood factor listed as affecting the evidence of isoimminization is the Hr factor discovered independently by Race and Taylor (254) in England, and Levine and polayes (176) in this country. It is allelic to Rh and occurs with about as equal frequency as the Rh agglutinogen. Levine (158) thinks the Hr and Rh relationship is similar to group O. At any rate Hr is found in all Rh- bloods and heterozygous Rh+ bloods. (254) Levine (169) sees no basis for excluding the Hr genes from any theory of heredity of thee various subtypes of the Rh factors. Levine (169) also assumes that of the 9% of Rh+ mothers of erythroblastotic infants about 3% are Hr negative there by being immune to the Hr factor in the blood. Mc call Holds worth (187) think since 90% of the mothers bearing children with hemolytic disease are Rh-, it clear that either Hr isoimmunization occurs much less frequently or its effects are usually unrecognized. (Levine (169) thinks that Hr is much less antigenic than Rh)

It is important in a consideration of this type of blood to understand that Rh- blood is not blood characterized by the absence of agglutinogens for it includes agglutinogens of sub-types Rh’, Rh” and Rh’ rh” and in addition the agglutinogen which has designated Hr. (339) Though it is assumed Hr

has the same subdivisions as Rh, only two have been found. (328)

Finer Divisions of the Rh Factor

At third category to be considered, in the 10% atypical reactions of blood incompatibility, is the Rh factor itself. The subdivisions of the Rh factor may be incompatible when mother and child are both Rh+, but of different sub groups. It is thought isoimmunuzation in pregnancy due to Rh incompatibility occurs more often when the A-B-O groups are compatible than when they are incompatible because a competition of antigens a case of sub group incompatibility in which ha says the child recorded and is now all right. Levine (167) says there are sub clinical cases, but Weiner says they must be proved. (Haldane (107) and Weiner (324) say the subtypes come from mixture of races.) Many researchers have reported various symptoms; and from all, the conclusion is drawn that the significance of subtypes is not known, but that they must be watched and studies.

Summary

The reports in regard to the 10% atypical reactions are merely suggestions of the picture because like the consideration of pathologies and symptomatologies resulting from typical incompatibility, the exact reactions of factors and groups are out of the scope of this study. However, a few summarized statements are in order:

1. Heterospecific pregnancies occur about 1 in 4. (171, 277, 337)

2. In 26.5% of cases in a report of heterospecific pregnancies the children were normal (278)

3. Any tissue of which a blood substance is a protoplasmic content may be affected by the agglutinin (698)

4. There are reports of sub clinical cases. (245, 324)

5. It is thought subtypes come from mixture of races.

6. Cases of irregular agglutinins are reported. (103, 236, 245, 254, 298, 324)

7. Tovey (298) and Smith (277) find no evidence of harmful affects to the child in cases showing atypical agglutinins.

8. There are reports of child and both parents being Rh+. At eleven months the child did not sit up had spasticity of the legs. (74)

9. Gurevitch (103) thinks where there is incompatibility of subtypes and blood groups the reaction is less severe than if the incompatibility is only Rh+ and Rh-.

10. It is assumed that Hr reacts with the dominant Rh+ factor. (32, 255, 254, 331, 356)

11. If an infant has icterus precox due to A or B sensitization, transfusion may be withheld because such infants usually recover spontaneously. (337)

12. A long period of neonatal jaundice followed by sequelae in the C. N. S. is very suggestive of an atypical erythroblastosis fetal is and kernicterus. (74)

The whole picture is so complicated that only a serologic specialist could analyze it; and in addition, there is not at the present time sufficient amount of the necessary sera to make extensive studies. One reason the testing started for this study was so unsatisfactory was that it was seen that if blood incompatibility was an etiological factor in the causation of the types of speech disorders studied, it was probably due

to an atypical agglutinogen and such tests could only be made by serological specialists.

The twelve statements above, the summarized statements on pages 7, 8, and 9 and the brief reports on page 10 suggest that there are loopholes in the picture of blood compatibility which must be found and for which some account must be given. The hypothesis of this study is that as the review of the case histories of blood incompatibility presents such a similar picture to that presented by case histories of stutterers, aphasics and children with delayed and spastic speech, there must be a common denominator somewhere. The fact that speech does not manifest it self until a later period makes it impossible to test for speech in the neonatal life. Later it will be shown that some of the statements made in this chapter are closely related to speech disorders. Crothers (15) says that we “have to recognize that a good deal of damage, some of which may be permanent, can exist without modifying behavior in early infancy cerebral and basal ganglia lesions can go unnoticed until walking and talking are attempted”. This damage Crothers mentions may sometimes be due to blood incompatibility (and may be speech cases) and the victim may show speech difficulties as he develops.

CHAPTER III

KERNICTERUS AND ITS SEQUELAE

As far as the relation of the speech of the stutterer, spastic aphasic, and the child with delayed speech to blood incompatibility is concerned, it would seem, the most important consideration is that form of icterus related to isoimmunization in pregnancy known as kernicterus. The knowledge in regard to kernicterus has many incomplete links even though the disease, as such, was first described by Orth in 1875. (74) In 1903, to designate this severe type of jaundice of various nuclear masses and jaundiced pigmentation and degeneration of the brain, Schmorl coined the term “kernicterus”. (118)

The etiology of kernicterus has never been satisfactorily explained and as no one has been able to produce it in animals, (16) there can be no experimental study. Lack of normal brain development has been considered as the most tenable hypothesis of ischemia of cases involved with secondary staining by bile pigments. (16) In fact, the primary existence of maldevelopment to some degree may be the controlling factor in ischemia. (16) Immaturity of cerebro or cerebro vascular tissue may play an etiological role. (74, 85) Doctor (74) thinks the universal association of kernicterus with erythroblastosis would suggest a more specific etiological factor than can be explained on the basis of ischemia and jaundice even with maldevelopment. He thinks the pathologically important lesion is demyelinization and degeneration of nerve cells and the secondary

Lesion is pigmentation. Macroscopically, he found pigmentation to be greatest in the nuclear structures of the brain but microscopically cellular degeneration and pigmentation were seen scattered throughout cerebral and cerebellar cortices. And the areas of most marked pigmentation were not always those of most marked degeneration. “In kernicterus, there is no uniformity of symptoms and it hasn’t been found blood destruction is the cause of kernicterus except that it may lead to an increase of serum billirubin.” (238)

Since the basal ganglia, especially the corpus striatum, and the hippocampus have relatively poor blood supply, they would be expected to be the first to suffer in anemia or disease involving blood supply to the brain. A general summary of cerebral complications found (118, 16, 48, 49, 51, 66, 67, 74, 86, 89, 106, 142, 283, 3.9, 357, 372) in kernicterus groups them under four headings, namely: (1) structures involved, (2) areas congested, (3) hemorrhaged areas, and (4) other findings.

1. The most commonly affected structures are:
   The basal nuclei, especially the corpus striatum, the hippocampus, caudette nucleus, lenticular nucleus, subthalamus, dentate, parts of cerebellar cortex and posterior horns of the spinal cord.

<u>Other structures affected are</u>:
The mammillary bodies, nuclei of crania nerves, corpus callosum, fornices, hippocampal and dentate gyri, pons, medulla, cerebella, cerebellar cortex of clocculus,

Pariental cortex, pariental white matter and surface of brain.

2. Congestion is found in the pia arachnoid. (“Modoerate congestion” with no location designation is mentioned.)

3. Humorrhages are reported as:
   Sub arachnoid, massive intracranial hemorrhage over the right cerebral hemisphere and within the right ventricle.

4. Other finding are:
   Clots around the base of the brain, developmental hypoplasis of corpus callosum and formic, purulent leptomeningitis, brain infarction and cystic degeneration of the choroids plexus.

Obviously, these complications do not cover all areas of degeneration, demyelinization and pigmentation, possible or probable, because the only reports would be of those brains examined at autopsy. Those who “recover” carry their stories of damage with them. And if the manifestation is not one previously classified as a sequela of brain damage it is not recognized as pertaining to the “damage”.

Often the kernicteric patient does not live but, if he does, there is evidence of damage to the basal ganglia i. e., extra pyramidal spasticity, choreathetoid movements, emotional instability and more or less mental retardation. (198, 371, 372) klingman and Carlson (142) found symptoms of disturbance of the nervous system in the form of convulsions, spasticity, apathy or lethargy, restlessness, increased crying and

difficulty In feedings. In other cases the symptoms referable to the nervous system were not recognized at time the child had the jaundice but appeared from a week to a month later in the form of delayed motor function, gradual progressive spasticity, incoordination, choreatherosis, staxia, hypotonicity or retarded mental development. (74) One report says, almost all cases showed definite mental retardation. (309) Yennet reports that some children, who recovered from icterus gravis neonatorum, subsequently developed mental deficiency, extra pyramidal spasticity and symptoms of injury to the C. N. S. (364) Also, autopsies were made which demonstrated clinical and anatation had faded as jaundice anywhere fades. (371)

Common delay after birth in development of signs of icterus suggest factors associated with delivery may have a great effect upon the development of erythroblastosis and kernicterus. The fact that kernicterus is common in infants who develop icterus only after birth, and fact that certain infants dying of kernicterus have little or no signs of long standing injury due to maternal with delivery may be of such significance as to accounts for kernicterus, at least in some cases. (197)

Potter associated kernicterus with blood incompatibility when she said “among other things associated with erythroblastosis is pigmentation and degeneration of nerve cells in the brain”. Doctor (74) says that possibly the Rh agglutinins have a specific affinity and toxic action on cells

of the basal Ganglia or in some way sensitize these cells to circulating bile pigment as the immediate cause is due to injury of brain cells by excessive bile salts in the blood. Yannet says, (363) “occasionally the maternal immune antibodies produce cerebral lesion instead of erythroblastosis and if anoxia due to red blood cell destruction occurs during a critical period of embryological development, lasting mental deficiency may result”. Necrosis of ganglion cells in the jaundiced parts of the brain have been regarded ad the cause of the nervous symptoms which occur frequently during erythroblastosis or later. (278)

After it should be stated, the literature must be reviewed to obtain a clear picture. Such review shows that kernicterus does not seem to have uniformity of sequelae, but it seems always to have an involvement of the C. N. S. to some degree. The erythroblastosis and kernicterus may seem severe and the evident symptoms mild or absent or vice versa; the damage to the C. N.S. may sow up after the jaundice has faded or the evidence of damage to C. N. S. may fade as the jaundice fades. Macklin (198) thinks the pathology is almost always accompanied by mental retardation. Only a small number of cases of kernicterus have a history of severe jaundice (74) and it is thought perhaps the reason for the damage to the C. N. S. is because tissues of the C. N. S. are notoriously susceptible to oxygen starvation. In asphyxiation, permanent injury to brain tissues takes place before the rest of the body is beyond repair. (278) Danger of permanent injury to brain through

anoxemia at the birth process is recognized as one of the birth hazards. (241) Oxygen deficiency at the time brain tissues develop may result in permanent damage: as a matter of fact, oxygen deficiency at any time can result in temporary or permanent damage according to the severity of the anoxia. (498) In blood incompatibility of the newborn, death has been, attributed by some to anoxemia and certainly the excess bile salts in the blood encountered in kernicterus can cause anoxemia. (498)

It has been show in this chapter that a disease, long known to cause damage to the brain, is one of the pathologies of blood incompatibility in pregnancy. The sequelae of the disease are various forms and degrees of damage to the C. N. S.; and these sequelae are highly indicative of the etiologies of certain speech disorders. (623, 648, 668, 231, 321, 363) Crothers (158) has called attention to the fact that residuae of damage to the C. N.S. may go unnoticed until the infant should begin to walk and talk. It is the hypothesis of the author that these residuae might be the “sub clinical” cases of disease of blood incompatibility of the newborn; and that, therefore, the form of blood incompatibility of the newborn known as kernicterus may be an etiological factor in speech disorders such as stuttering, spastic speech, aphasia and delayed speech.

CHAPTER IV

STUTTERING, SPASTIC SPEECH, DELAYED SPEECH AND APHASIA

The modern world may have moved far from the theory predestination, but genetics is still governed by it. Whom speech disorders, of certain types are considered, the examiner is always baffled as to what has caused the condition. Perhaps the father and mother of the patient have normal speech, perhaps his sisters; brothers, aunts, uncles and grandparents do also. But some where back among the ancestors there is usually a stutterer a spastic, an aphasic or a person with delayed speech, and the health histories have surprising similarities all along the line. (It must be kept in mind that the discussion is those cases with unconvincing or unknown etiologies.) Perhaps the earliest known inquiry in regard to the genetic effect was the question the disciples asked Jesus, “Master, who did sin, this man or his parents that he was born blind?” Jesus’ reply was, “Neither hath this man sinned or his parents, but that the works of God might be made manifest in Him.” Since that time there have been various conflicting opinions as to whether or not a deviation from the normal in the living organism meant “sin” in the previous generations. Would the sins of the fathers visited upon the children even un to the third and fourth generation be in the form of this deviation from the normal in neurological, physical, mental and/or emotional development? It is easier to prove familial tendencies and antenatal influence.

There is obvious proof that antenatal conditions cause cleft palate and there have been unsuccessful attempts to prove the antenatal conditions were caused by the “sins” of the fathers. Cleft palate obviously occurs in the same family line, but no one has been able to show why. One theory postulates heredity through a recessive gene (83, 358) and another suggest nutritional deficiency of the mother during pregnancy. (98, 316, 317, 318, 319) Could it be that both of these factors are linked in one causative factor: the deficient nourishment allowing the recessive gene to become active in blood incompatibility?

As stuttering, spastic speech, aphasia and delayed speech do not manifest themselves at birth, proof that they are caused by antenatal or neonatal conditions is not so obvious. However, again, as they tend to “run in families” many investigators, to no avail, have attempted to discover what “sin” of the father has been visited unto the third and fourth generations.

Cleft palate and spasticity have always been classed as deformities, but stuttering, aphasia and delayed speech have had many classifications and names. All have been enigmas as to etiologies. Eisenson (228) thinks Noses stuttered, but when he had a message which the Lord commanded him to deliver he could speak. “Lord, I am slow of tongue.” Might this not just as well refer to mild aphasia, or spasticity? What is the cause of the “slowness of tongue” manifested in speech disorder? To answer this question we will first make a survey of previous investigations of the causes of stuttering.

Hereditary Theory

The hereditary theory of stuttering has been in the foreground for some time among authorities. Orton and Travis (223, 628) seem to think the disorder is at least partly inherited. Boome and Richardson, (108) the English workers, say they “incline to the view the child inherits peculiar neuropathic tendencies which predispose him to stammering rather than the view that the actual stammer is inherited”. Frank Bryant (248) says it is caused by “a germinal trait which produces an inherited predisposition”. Froschels (268) says, “We can hardly go astray if we think of a constitutional predisposition which may be transmitted by inheritance, as we must assume, in general, a constitutional sensitiveness of the sufferer for the malady even though no forbear was afflicted with this speech disturbance.” None of these people has given any idea of the cause of the “predisposition”, “condition”, “nouropathic tendency”, etc. However, “If one may judge from the symptoms of stuttering itself (i. e., from the speech manifestations of dysphemia) the condition underlying the stuttering is transmitted from parent to offspring by biological heredity stuttering “runs in families”. (668)

Biological Heredity Studies

This theory of biological heredity has been studied from the viewpoint of attempting to discover the significant

physiological difference the stutterer has in his make up. Twitmeyer (304) and Trumper (301) related stuttering to a hemato-respiratory imbalance. Starr (285) maintains the pH of the blood of stutterers is disturbed as shown by the high alveolar CO_2 determination; but Kopp (143, 144) in his studies of blood, alveolar air and urine of stutterers could not uphold Starr's findings. In his work with the substances in the blood, however, he found that the relative balance between the in organic phosphate, sugar, potassium, calcium and protein of the blood is not maintained. This difference in ratio seemingly is unimportant in the function and Whealdon (6) tested for the ABO blood grouping in stutterers and found a alight difference in the incidence of the B group which they did not consider significant.

Neurological Studies

Travis (300) says of stuttering that it is a "neuro-muscular derangement secondary to general reduction in cortical lead control". A temporary neuromuscular block of different forms has been investigated by Travis, Strother, Blackburn, Seth and West; and after reviewing their investigations, Van Riper (638) agrees that the temporary inability is due to the lack of simultaneous or similar volleys of nerve impulses sent from the brain to the paired speech musculatures. West, (321) because he noticed that certain cases of what resembled dysphemia brought on by an organic lesion due to syphilis showed very sluggish movement

of the muscles of the face and in non speech movements as well as speech movements, made a study of the maximum speed to which the facial and mouth muscles could be forced in purely repetitive acts of simple nature. The sluggishness disappeared when the infection was cured. The results of the test showed that the stutteres had a much slower rate of diadochocinesis than the normals and the slowness of the rate was in proportion to the subject's possession of "this certain quality or condition that characterizes dysphemia". (321) the lower quarter of the normals overlapped the upper quarter of the stuttereres making possible divisions into:

1. Those who, even though their emotional adjustment was poor, would not stutter.
2. Those who, even though their emotional adjustment was good, would stutter and
3. Those whose stutter would vary with their emotional adjustment.

Children, both stutterers and controls, demonstrated much slower rate of diadochoconesis than adults, but were consistently in a ratio as to age and sex.

It was felt that the factor being tested was probably more significant in the etiology of dysphemia than any other one factor, and that slow diadochocinesis of certain face and mouth muscles is either a cause of or is related to a cause of the disorder. "The test may be largely a test of ability to maintain an efficient control over the lower motor neurons of the face, mouth and throat in opposition to the sub-cortical control of these same neurons". (321) This is shown by the fact that sometimes the spasms disturbing speech seem to be of the musculature of the face, sometimes only the lips, sometimes the tongue, and at other times the larynx or respiratory apparatus

causing a practically spastic condition of the throat accompanied by the “fight or flight tune up of the cardiovascular, respiratory and muscular systems. As the muscles of the mouth and face are more directly under the control of the centers which control the emotional responses of the motor system than the rest of the musculature of the body, there could easily be a confluent between the thalamus and the cerebral cortex for the control of the L. M. N. If the shift from one to the other center in rapid enough, there is “a decided tremor of the articulatory musculature symptomatic of an emotional state”. (321) Just before the stuttering spasm begins, such a tremor very often is observed in dysphemia. (321)

The neuropathology predisposing the person to dysphemia might be one of two types: (66B)

1. an interference of the inhibition of the cerebrum over sub cortical centers
2. a hyperactivity of the sub cortical centers causing their impulses to break through the cerebral inhibition even though it is normal.

“This hypertonic condition may be due to toxic condition systemic in nature or to specific stimulation of the centers as in chorea.” (668 “Also apparently during stuttering, the symmetrical action of analogous muscles on the two sides of the face is disturbed, thus indicating a rather fundamental disorganization of the integrating centers of the C. N. S.” (62B)

The dysphemic person’s lack of vocal inflection may be an evidence of the same spasticity which West (321) found caused a sluggish muscular response in repetitive movements. The normal speaker glides through a vowel, but the stutterer finds

that the more nearly level he keeps his tone, less likely he is to stutter. He seems to find it impossible to shift the control as quickly from one muscle group to the other as is needed for change of inflection. The stutterer sounds very much like the spastic when he uses this scanning speech. In the spastic, scanning speech is a phase of intention tremor known to be due to partial impairment of the motor tract of speech. It demonstrates the effort of the brain to be coherent and is comparable to the slow, careful gait of the ataxic. (4) when the stutterer uses scanning speech, he may be removing cortical control entirely and placing the control in the same area the spastic uses. Then, stuttering speech may be a speech which has mixture of the qualities of normal speech on one side and spastic speech on the side on which the balance of the scales is maintained.

The slowness of diadochocinesis of the spastic is very much like the slowness of diadochocinesis of the stutterer except that the lack of energy displayed by the stutterer has a more even tenor than that of the spastic. It seems that any factor which would interfere with the inhibitory control of the cerebral cortex over the L. M. N. could also interfere with the inhibitory control over the basal ganglia which is the center for emotional control of movements causing the phenomenon of choreathetoid spasms. Only if the spasm occurs in a muscle group which controls speech, will speech be affected.

Like the stutterer, the spastic displays emotional

involvement of varying amounts. However the stutterer the emotional involvement does not seem to be paramount to the spasticity. Like the speech of the stutterer, the speech of the chorea tic has spasmodic speaking and blocking. And when he becomes conscious that his speech is different from that of those around him he develops a spasmodic hesitancy which West thinks may have the same psychogenic aspects as stuttering. (66B) How many time have speech therapists thought of a stutterer, “If he would relax – especially the face, neck, shoulder, back & chest musclea” Dr. Phelps (71B) says, “If the athetoid could learn conscious relaxation, he wouldn’t need speech training”. An article in the June, Journal of Speech Disorders calls attention to the fact that “Some of the speech problems of the very young children reasonable speech blocks or delayed speech”. The author has observed this in cases other than those of young children. However, such cases may be limited, at least usually, to cases of congenital origin. This chorea of children and birth (Syndenham’s Chorea or St. Vitus’ dance) has “involuntary movements, semi purposeful in character, involving muscles of limbs and face. As death is rare, little is known of the neurophathology, but in the few fatal cases degenerative changes in the corpus striatum, substantial nigra and the body of Luys have been described”. (7B)

Medical Studies

Stuttering has been considered from hereditary, physiological and neurological points of view. Now it will be considered from a medical standpoint. Gordon (98) found that large doses of thyroxin given to persons who had not previously stuttered initiated the staggering mechanism. Browning (13B) associated stuttering with lack of thymus involution of the ductless glands. (Davidsohn (57) thinks perhaps blood incompatibility may be a disturbance of the endocrine system.)

Berry (14) made a through study of the medical history of stutterers and found a high incidence of

1. Diseases peculiar to infancy.
2. Convulsions.
3. Cerebral hemorrhage due to birth injury.
4. Malnutrition and anemia.
5. Diseases later than the birth period which could have caused brain damage.
6. Allergic symptoms.
7. Respiratory manifestations.
8. Cardiac disturbances.
9. Involvements of nose and ear.
10. Diseases of nervous system (exceedingly high).
11. Twins.
12. Males.
13. Stutterers in families containing both twin and single siblings as compared to families where there were only angle siblings.
14. Retardation in the acquisition of motor skills as evidenced by walking.
15. Retardation in the development of intelligible speech based on the standards set by McCarthy.

Of the above factors, which have been related to stuttering,diseases peculiar to infancy, convulsions, malnutrition and anemia, diseases of the nervous system (exceedingly high).

and retardation in the acquisition of motor skills as evidenced by walking, have definitely been related to blood incompatibility; cerebral hemorrhage due to birth injury, allergic symptoms, respiratory manifestations, cardiac disturbances, twins, males, and stutterers in families containing both twin and single siblings as compared to families where there are only single siblings, are factors which the author found prominent in the literature on blood incompatibility of the newborn. Any disease later than the birth period which could cause brain damage could be an etiological factor if kernicterus is an etiological factor.

Karl in and Kennedy (138) reporting on Flechsig's study of the embryology of the brain say later development of speech in the male may be caused by a later myelinization of nerve tracts in the speech pattern. These tracts may be in any part of the speech synergy: the receptor or the hearing mechanism, the association fibers in the cerebrum or in the efferent motor tracts to the muscles. Based on the same reasoning, the hypothesis may be made that delayed development of speech in any individual may be caused by delayed myelinization of nerve tracts in the speech pattern. (Kernicterus, as was shown in Chapter III is an icterus which damages the brain) and one form of damage found at autopsy of kenicteric patients is demyelinization of nerve tracts.

Retardation in the development of nerve tracts. Speech based on the standard set by McCarthy, has been shown by Berry (14) to be related to stuttering. All stutterers do not have this retardation, but delay in the development of

intelligible speech and stuttering seem to have something in common. If the degree of retardation in excessive, the disorder known as aphasia may be evidenced. Huber (125) found that aphasics have difficulty in changing from surds to sonants. This difficulty might represent a dystonia similar to the dystonia of stuttering.

Berry (14) found the significant difference between the normals and the stutterers in relation to the frequent acute upper respiratory infections was that a comparatively large number of the stutterers had an illness which took a severe course and was accompanied by high temperature (104^{o}F or higher): it was a qualitative rather than quantitative difference and she adds, “If we were to make a general summary we would conclude that specific infectious diseases of the respiratory tract and diseases of the nervous system are most immediately and most frequently associated with the onset of stuttering.” (14) This substantiates the author’s theory that the kernicteric damage is possibly, an etiological factor in the onset of stuttering. In summing up her discussion Berry (14) says, “Although the problem still is sub judice, the final interpretation of these infections diseases and of nervous disorders some day may be stated in terms of a demonstrable impairment of nerve cells or, in the phraseology of Schick and Pishkin of a ‘physiochemic disturbance in equilibrium which probably manifests itself through the nervous system’.”

Then delayed speech aphasia, stuttering and spastic speech

seem to have indications of a common etiological factor evidenced by a dystonia. That etiological factor seems to involve brain damage; and blood incompatibility has been shown to sometimes cause brain damage in the newborn. Since speech develops later than the neonatal period, speech disorders have not been associated with the symptoms manifested at birth; and Crothers (15B) says there may be a great deal of damage which will be unnoticed until the time the child should normally walk and talk.

The Psychogenic Nature of Dysphemia

Where stuttering is mentioned, the question of the psychogenic nature of dysphemia always enters the discussion. “Though psychogenic disorders are functional in nature, they are more prevalent among those showing poor physiological and neurological stability.” (321) The person we call “normal” is not perfectly adjusted socially and emotionally; he merely has an adjustment far enough above the danger line to allow him with his physiological and neurological stability to “get by”. Probably with another person’s poor physiological and neurological stability, he would manifest a stutter which has been evident because of a stable physiological and neurological pattern backing up his adjustment. Maybe some dysphemies, in spite of poor physiological and neurological stability, with the normal adjustment referred to above would

Not stutter. However, sometimes those with such an adjustment do stutter. The biological of the mechanism used for speaking may be in perfect order; only when the overlaid function of speech is required do the muscles innervated by the Vth, VIIth, IXth, Xth, XIth and XIIth cranial nerves, superior cervical, and phrenic nerves give evidence of abnormal response.

Then, stuttering though it may have a psychogenic factor in its etiology has also biological, physiological, neurological and medical etiological factors, thus when stutterers are spoken of, the terms psychogenic, physiological, biochemical, neurological, medical, hereditary and familial are interwoven in an effort to explain the speech picture. The present study is an attempt to relate all these parts of the puzzle to the picture of blood incompatibility as inherited in families, thereby influencing these interwoven etiological factors.

The other speech disorder being discussed have phychogenic symptoms but usually to a less degree than stuttering. All of those disorders which kernicteric qualities and psychogenic symptoms might be called dystonic speech.

CHAPTER V

POSSIBLE REASONS FOR CORRELATIONS BETWEEN BLOOD INCOMPATIBILITY OF PREGNANCY AND STUTTERING, SPASTIC SPEECH, DELAYED SPEECH AND APHASIA

The complete significance in obstetrics and pediatrics of the discovery of the Rh blood factor is not clear at the present time because of conflicting theories. However it is obvious that this discovery is so important that no stones should be left unturned to explore the subject. As speech development is closely related to pregnancy and neonatal life, anything which, as we have seen in Chapters II and III, so vitally affects such periods of possible aid for what Dr. Wendell Johnson has called, “The Million Forgotten Children.”

“Sub Clinical” Reaction

First there must be made evident the “sub clinical” view of the reaction of blood incompatibility as that view is the basis for assuming that such reaction could possibly have any relation to speech development.

When the fact was first established that Rh incompatibility between the blood of the mother and the fetus was related to pathology resulting from pregnancy, erythroblastosis fetalis was the only disease entity attributed to it. (17, 45, 51, 60, 89, 162, 163, 166) Then, researchers spoke of icterus gravis

neonatorum and hemolytic anemia of the newborn. (197, 218, 229, 234) Gradually, as the subject was studies more intensively, other pathologies were attributed to the incompatibility by some and denied by others as was shown in chapter II. Macklin (197) says all “acute anemia’s” should be considered; but, since other functions and organs are related, there would be no uniformity of symptoms and it hasn’t been found blood destructions is the cause of kernicterus except that it may lead to an increase in serum bill Rubin.” (197) Since the human machine works as an entity it seems plausible to assume that impairment of the functioning of any particular organ may impair the functioning of related organs of time as it is discovered which congenital pathologies of unknown etiology are attributable to blood incompatibility, it seems logical to consider all such pathologies as possible sequelae of such incompatibility. These pathologies are often the ones which the speech pathologist finds relates to speech discovers such as are being studied in this research. It was also shown in Chapter II that exact symptoms as shown by the pathologies attributable to blood incompatibility are still controversial. The term “sub-clinical” gradually crept into the literature in an attempt to explain some of the unaccountable seeming discrepancies in findings, and such phrases as the following were used:

1. Some of those with highest icteric indices showed no neurological signs at months.
2. (74)Some showed no signs of spasticity or convulsions until months or years later. (74)

3. Antibodies were evident but there was no sign of damage. (74)
4. Some who were spastic when jaundiced tended to improve as the jaundice faded. (74, 330)
5. Mild cases have gone unrecognized. (55, 171)
6. Transfusion is not needed if icterus precox is due to sub-groups because the child will usually recover spontaneously. (70, 324)
7. Milder forms may have been overlooked. (158)
8. The effect may be different from erythroblastosis fetalis. (57)
9. Spontaneous recovery may be made in mild cases before damage is done. (11)

As shown in Chapter II besides the Rh+ and Rh- incompatibility, there have been proved incompatibilities involving subgroups, sub-factors, Hr, and other as yet undiscovered factors. There are also other influencing agents. These differences are supposed to cause milder reaction and many reports, as stated above, say “sub-clinical” reaction. (19, 85, 102, 119, 137)

Because speech develops later than the neonatal period it could be possible that the “sub-clinical” cases do not “recover spontaneously” as reported, and do have damage which has not heretofore been associated with blood incompatibility. Crothers (15) says no decision can be made as to prenatal or neonatal damage to the nervous system because symptoms in infancy are not defined very clearly.

Speech Disorder and Brain Damage

It was shown in Chapter IV that stuttering, spastic speech and aphasia are due to damage to the central nervous system and

might be called dystonic speech. In the case of aphasia certain areas of damage are fairly well known. However, as the brain works as a whole there may be other areas than the ones which have as a whole there may be other areas than the ones which have been definitely located. Spastics have trouble with speech only if the mechanism for speech is involved in the spasticity. West (66B) showed similarities between spastic speech and stuttering and Berry (14) associated delay in speaking with stuttering. Delay in speaking is also associated with aphasia. (66B) Where will the line be drawn between the child who is merely slow in initiating speech and associating symbols and the child who has an impairment of such ability? When does he have delayed speech and when is he dysphasic? Snyder (278) has shown that acquired amentias are caused by blood incompatibility and aphasia is an acquired specialized amentia. (66B) As will be shown in the next chapter, there are many comparisons between “sub-clinical” blood incompatibility symptoms and dystonic speech symptoms.

Kernicterus and Speech Disorders

In Chapter III it was shown that one form of the disease of blood incompatibility of pregnancy is an icterus which damages the brain — kerniterus. It was shown also that kernicterus has long been associated with pregnancy and neonatal life. Autopsies have shown that the area of the basal ganglia is most frequently damaged in kernicterus; and the speech disorders being discussed have one thing in common: evidence of the basal ganglia: i. e., disturbance of emotional control of movement

of certain muscle groups. Autopsies have also shown that other areas damaged are areas which could be associated with the etiologies of the speech disorders named. (Chapter III)

Bases for Correlation of Speech Disorders and Blood Incompatibility

In this chapter there will be given a summary of the bases for the correlation of speech disorders and blood incompatibility of pregnancy and in the next chapter the exact correlations will be discussed. A summary of the bases for assuming that there is any possible relation between blood incompatibility of pregnancy and speech development are:

1.Speech development is closely related to birth and the neonatal period, and, as shown in chapter II and III, pathologies not found at other stages of life

2.Many of the established and controversial patholies and symptomatologies of blood incompatibility of the new born as shown in Chapter III IV have been related by speech researchers to speech development.

3.All those reporting on disease of blood incompatibility of the newborn have remarked upon the expected frequency (9%)It is possible, however, that some children who have certain speech disorders of unexplained or unconvincing etiology belong in this missing 8.5%.

4.There are references in the literature in regard to blood incompatibility of pregnancy to cases which meet all the criteria of disease resulting from blood in compatibility of the newborn except that the investigators say the obvious symptoms do not give the classical picture. (Chapter III and the 1st of this Chapter)

5.Doctor, (74)after adding study of his own to a critical review of the literature, made the statement that “sequelae in the central nervous system is very indicative of an atypical erythroblasos is and server

Kernicterus.” Among others, Head, (125) Travis (62B) and West (66B) have placed aphasia, stuttering, spastic speech and delayed speech as sequelae of damage to the central nervous system.

6.Kernicterus, which has been shown to be one of the pathologies resulting from blood incompatibility of the newborn, has symptoms of damage to the central nervous system, (11, 74, 309, 371, 372) and some speech disorders of unknown or unconvincing etiology have kernicteric qualities. (62B, 66B, 14)

7.Patients with no obvious icteric symptoms have later developed symptoms etiologically related to kernicterus. (37, 74, 309, 79) The only ones reported are those who showed apasticity later. Those who had no spasticity were considered “spontaneously recovered”. However, a check later might have shown symptoms not heretofore related to kernicterus but having kernicteric qualities-dystomic speech disorders such as are being discussed in this study.

8.Occasionally the maternal immune antibody produces a cerebral lesion instead of orythroblastosis. (259, 278) A review of the literature of seems to indicate that there is sometimes evidence of cerebral impairment without other evidence of incompatibility. As speech is so definitely a function of the brain, the lesion could be one causing speech impairment alone.

9.Necrosis of ganglion cells in jaundiced parts of the brain has been regarded as the cause of nervous symptoms which frequently occur during the disease (erythroblastosis fatalis) or later. (93) This nervousness may be related to a speech disorder which is evidenced later.

10.Snyder (278) says that, Yannet et al. (363, 364, 365, 366) have shown immune antibodies of the mother may produce injurious effects on brain tissue. The suggestion has been made that the immediate effect of red cell destruction is anoxia and that this lack of oxygen, if it occurs at a time when the brain of the embryo is in a critical stage of development may very well cause damage to that structure rather than produce .5% as the probable number mentally defective from such reaction.

11.As there will be icterus before it shown clinically, the incidence of icterus has variously been placed at from 205 to 80% (11) this variability could possibly

be partly accounted for by the fact that mild symptoms go unrecognized and show up later as speech cases and as the spastic cases of which Phelps (233) speaks. He says a great many children in our public schools who are considered “normal are slightly spastic. In the author’s opinion, the same is true of speech cases spastics coincide in a great many cases.

12.Vaughan (309) says any tissue which has Rh substance as a protoplasmic content may be damaged as well as the blood. Since it has not been definitely established exactly to what extent body fluids and tissue do contain some of the blood incompatible that they do contain others, (60B, 61B, 167, 309) some of the abnormalities of development related to speech of the speech may be due to the fact that the Rh substance is a protoplasmic of certain tissues.

13.It is been generally considered that first born children are not usually (238) affected due to the fact that it takes some time for the antibodies to develop, but Potter (238) thinks it is inconceivable that any condition produces by individual action of a genetic factor, infection or any other etiologic agent should fail to act in a first pregnancies. Snyder (278) has some evidence that those affected first born show gross abnormalities, including spina bifida. Perhaps in other cases where it has been considered that there in no reaction the effect is damage which would not be observable until some time later when speech develops. An extremely mild form of the disease occurs occasionally in the first child. (73, 208) An explanation of the occasional occurrence in the first born is that transfusion of incompatible blood at any time in the past life of the mother might be responsible. Levine (38B) thinks oven intramuscular injection in infancy should he considered and that, he thinks, has been used frequently as therapy in cases of debility. Perhaps the disease occurs more often in the first born and the only noticeable result is dystomic speech. Perhaps those cases in which and those cases in which the develop sever effects and those cases in which the mother has not had an injection are the “sub-clinical” cases. Since speech develops so much later, the mild form” may have been forgotten by the time it is noticed that speech is slow in developing, is absent or asynergic.

14.The condition responsible for isoimmunization ordinarily occurs prior to the pregnancy in which erythroblastosis

is first manifested. (241) A possible explanation might be that “sub-clinical” symptoms are present in the prior pregnancy and night show up later as dystonic speech.

15.Potter (240) observed, but has not proved, that in a majority of cases, a male child is the last one born before the child with evidence of erythroblastosis. As there are speech cases boys than girls, these speech cases may be the ones before the erythroblastotic child.

16.Amberg (3) quotes authorities on sequelae that involve the nervous system as saying they are familial and related to erythroblastosis fetalis, anemia of the newborn, kernicterus and icterus gravis neonatorum. Stuttering, delayed speech and sometimes spasticity are “familial” and are related to the “rheumatic diathesis” which has sequelae that involve the nervous system. Though nothing has been literature in regard to blood incompatibility.

17.Speech case histories involve disorders of the thymus gland and symptoms associated with disorders of the thymus gland. (66B) The author observed frequent mention of the thymus gland in the literature in regard to blood incompatibility. An endocrine imbalance may be the causative factor. (74) Although this is not proof of a relationship in either cause or result of either disorder, a possible relationship is never the less suggested.

18.Dr. Strother, of the University of Iowa, gave a report at the S. S. C. A. convention in Chicago, 1946, of the curve of development of speech sounds during the first thirty months of life. His study showed a definite sponse is retarded in certain cases. Since speech is a function of the central nervous system, retardation in development, even though alight, might be related to a prenatal disease affecting the central nervous system.

19.Travis (62B) says, “Pathological fetal positions: Clinical examinations reveal that there is a positive relationship in individuals between abnormal fetal positions and the incidence of speech defects.” Again, there is no positive proof but prominent in the literature in regard to blood incompatibility. Pearlstein (231)

Says that abnormal fetal positions as found associated with spastic paralysis must be caused by the paralysis speech defects, and other speech defects do not show an amount of spasticity (if any) sufficient to show played the role in causing to positions. Therefore, it would seem more logical to consider that abnormal fetal positions and spasticity have a common cause.

20.Clinical examination has shown speech disorders to be associated with prolonged labor and, as stated in Chapter III, kernicterus develops usually in infants who develop icterus after birth. This is demonstrated by the fact that infants dying of kernicterus have little or no signs of long standing injury due to maternal antibodies. Vaughn (309) thinks, therefore, that factors associated with delivery may account for the kernicterus, at least in some cases.

21.Potter (242) says renal agenesis is associated with an abnormal facial expression and some children with speech disorders such as are being discussed exhibit an abnormal facial expression. Kidney dysfunction is one pathology related to child blood incompatibility. Again, while this is not “proof”, it suggests a possible need for study.

22.It will be recalled that one incentive for this study was that children having certain speech disorders were usually bottle fed. It has been demonstrated that antibodies present in the colostrums and milk of the mother play an important part in the pathogenesis (359, 360, 361)

In this chapter it has been shown that there are reasons for believing an investigation of the blood incompatibility of pregnancy might be a possible factor in the etiology of certain speech cases with unknown or unconvincing etiology. In the next chapter it will be shown more completely how the blood incompatibility of the newborn might be related to stuttering, spastic speech, delayed speech and aphasia.

CHAPTER VI

POSSIBLE CORRLELATION BETWEEN BLOOD INCOMPATIBILITY OF THE NEWBORN AND STUTTERING, SPASTIC SPEECH APHASIA AND DELAYED SPEECH

In the last chapter, reasons for believing there may be a relationship between blood incompatibility of the newborn and stuttering, spastic speech, aphasia and delayed speech were given. In this chapter the more exact correlations with the different disorders. And as stuttering is the prime interest, it will be correlated first. Next, relationships will be shown with each of the other disorders which will explain why the particular manifestation developed instead of stuttering.

When we think of stuttering we think of certain facts that have already been associated with it. Berry (14) says, “We now have brought together the pertinent information on the five factors which we believe may one day be fitted into the picture of stuttering. High sex ratio, retarded initiation and development of speech left handedness, twinning, certain infections and nervous diseases with an hereditary subsoil: These are the factors which must posses a common denominator.

The common denominator may possibly reside in heredity.”

These five factors and others which have already been related to stuttering, i.e., prematurely, status thymicolymphaticus, similarities to chores tic speech and psychogenic qualities, will be discussed. And, in addition, bottle feeding, the phenomenon of childhood occurrence, and blood factors will be considered.

Heredity

It is well known that stuttering “runs in families.” As stated in Chapter IV, Orton, Travis, Boome and Richardson, Bryant, West, as well as most other workers, attributes at least part of the cause to a heredity factor. As said in the introduction, the number of cases studies by the author is not presented as conclusive evidence, but the case histories of the ones tested were obtained to give a clearer picture of the dysphemic person as considered in this study. In the histories of the 21 cases tested, there were reports of:

8 families having other cases of stuttering
*4 paternal ancestral families having cases of stuttering
*4 maternal ancestral families having cases of stuttering
*1 both ancestral families having cases of stuttering
*(Often more than one case was reported in an ancestral family.)

Nelson (215) found stuttering showed a tendency to be at least partially sexlinked. And though blood substances are inherited as Mendelian dominant not sexlinked, Wiener (340) and others (109) think search must be made for an at least partially sexlinked influencing factor.

Levine (167) says erythroblastosis is grouped in families and stuttering “runs in families”; erythroblastosis is the disease entity through which the Rh blood factor incompatibility was linked with pregnancy and neonatal life. Kernicterus, one of the pathologies of blood incompatibility, has sequelae of

damage to the C. N. S. Since stuttering has been associated with damage to the C. N. S. it seems evident that this hereditary factor may play a role which should be investigated in relation to blood incompatibility and stuttering. In fact, it may prove to be the common denominator as well as the additional factors which will be discussed.

Sex Ratio

They are more males than females among stuttering. The report of the White House Conference (66B) sets the figures at 7,941 males 2,059 females in 10,000 cases and from various reports sets the ratio at from 3:1 to 8:1.

Berry (14) found that males predominate in all factors relating to stuttering. Of the 21 cases chosen at random by the author, 17 are male and 4 females. West (66B) says of stuttering, “Some etiological factor related to the difference between sexes is involved, but this difference does not point directly to any pathological factor.” It will be noted that West says directly. In the sane vein, in regard to blood incompatibility of the newborn, Halparin (109), Cappel (32), and Wiener (345) say perhaps search must be made for some controlling factor of the antigen-antibody mechanism which is at least partially sex linked. There have been no reports of studies of blood incompatibility as related to sex, but the word “male” was conspicuously more prominent in the literature reviewed than was “female”.

Stillbirths have been associated with erythroblastosis and there are more male than female stillbirths with unknown etiology. Halparin (109) says there are more male stillborn from unknown cause than female, more male erythroblastotic babies than female; but more females than males who have erythroblastosis recover. This ratio for incidence of disease has been placed at 3 male to 1 female and ratio for incidence of death due to erythroblastosis at 5 male to 1 female. (11B) Berry (14 says of the stutterer's predisposition to respiratory infection that it is not so much quantitative as qualitative. The stutterers are inclined to have the diseases much more severely than the non stutterers and to have higher fever with them. (It must be remembered the sex ratio of stutterers is approximately 4 males to 1 female.) Oberhelman (220) has associated congenital tricuapid stonosis with a prepondernce of males, prematurity and bottle feeding. As stated before, all congenital diseases, especially those which have any relation to the blood formation and function, should be considered when considering blood incompatibility. And as will be shown later in this chapter, prematurity and bottle feeding should be considered in the same picture.

(Calvin) (14) says males are more susceptible to infant tetany than are females. And infant tetany can be caused by anything which reduces the blood glucose to a critical extent. Tetany, while it may have other causes, should be considered in this picture of blood incompatibility.

Potter (240) thinks she has observed, but not proved, that there are more male than female infants born just prior to the births of the infant with evident erythroblastosis. Since it is the author's hypothesis that some speech disorders may be "subclinical" forms of the disease of blood incompatibility of the newborn (as shown in chapter IV) and sex seems to be intimately related to both speech disorders, related factors and blood incompatibility of pregnancy, it seems sex is a basis for linkage of blood incompatibility of pregnancy and stuttering.

Twins

Twins have always been interesting to those studying possible hereditary factors. Twinning has long been associated with those families having stuttering children. Berry (14) making a study herself and reviewing the literature found that a high percentage of stutterers were twins; the percentage of twins who stuttered was high; the percentage of males among twins was high and among stuttering still higher. She also found that there were more stutterers in families where there were twins than in families containing only single siblings.

There have been reports of cases of twinning related to blood incompatibility of the newborn. (69) As mentioned in the discussion of sex, a review of the literature is all that can be presented as evidence at this time* and in reviewing the literature it seemed to the author twins and male twins were

*Strandskov has in press at this time a study of blood incompatibility and twinning.

prominent, especially among stillbirths and neonatal deaths. Also, the general impression was gained that the female twin or twins seemed less severely affected than the male in cases of disease from blood incompatibility of pregnancy.

As far back as 1932, Diamond, Blackfan, And Baty (69) associated erythroblastosis fetalis and icterus gravis neonatorum “with twins and a feamilial tendency to such an extent that it was decided thay as well as hydrops and anemia of the newborn were varying stages of the same disease.” In 1940 (151) relationship between the blood incompatibility of pregnancy and erythroblastosis was demonstrated which bore out Darrows (52) previous theory that erythroblastosis was caused by an antigen-antibody reaction between the mother and the fetus. The reports bearing out her theory have included symptoms frequently encountered in medical histories of stutterers. This blood incompatibility is cause of kernicterus which the author has shown is related to the etiology of certain speech disorders.

Among others, Aaburg, (1) Aubert,(10, 11) Halparin. (109) Javert, (132, 133) Bornatein, (21) Love. (184) Horder, (124) Beck, (13) Stratton, (290) Gilmour, (93) Kariher, (137) Leonard, (155) Litchfield, (183) Jacobi, (129, 130) Levine (158, 161, 162, 163, 170, 173) and Wiener (330, 334, 338, 348) have associated disease of blood incompatibility of pregnancy and twinning with prematurity, prolonged labor, cyanasis, stillbirths, jaundice, hydrocephalous, spina bifida, polydactyl, anemia, familial tendency, mental retardation with out physical debility, feeding problems, and paleness. Conti

(38) Schwartz, (268) and Trout (299) all associated spina bifida, prematurity, paleness and feeding problems, still births, mental retardation and twinning in families of proved blood incompatibility. Quisenberry (249) and Snyder (279) related spina bifida to blood incompatibility.

Seymour and Koermer, (270) in a study of artificial insemination, found not a single twin in 10,000 births. Could this be due to the fact that the blood was typed? Sanford (266) says twins are often premature and have a tendency toward anemia. Marshall (204) places the single births. He also places the rate of twin births in the United States at 1 pair in 87 births while Schienfeld (57B) gives the proportion in Japan as 1 pair in 160 births. This has been attributed to the fact that Japanese women are small and probably unable to bear twins. However, it might possibly be due to the difference in the ratio of blood incompatibilities in Japan which Graydon (99) and Waller (313) place at 96% Rh+ and 4% Rh-.

There have been many theories about the cause of twinning. Jordon and Kindred (34) say a chemical factor in the sperm might be responsible causing a developmental arrest at a critical stage of development. “This factor could be inherited.” Wilson and Wolfshan (quoted by Berry) (14) offer as proof of the hereditary factor: “If structural and functional disease of the nervous system occurs, it affects both twins equally, and not one twin alone.” But Schienfeld (57) maintains identical

twins do not have the same inheritance, or the phenomenon of having one twin born alive and other dead would never occur: either the environment is not the some or one gets the best part of the ovum. Schienfeld (57%) says the aptitude for twinning, it is believed can be passed by the mother through the son as well as the daughter. Newman (46%) says both monozygotic and dizygotic twins are probably inherited through the father or both parents by means of a Mendelian mechanism not yet understood.

The question of monozygotic and dizygotic twins enters the picture because it has been on the basis of this difference that genetic studies have made. Newman (46%) says, “fraternal twins are a duplicate set of human genes parading in two bodies” and identical twins are “essentially a physicological isolation of two equivalent growing points due to a partial loss of integration & the bilateral halves of the blastodarm.” Wiener (69B) maintains that the only sure method of determining whether twins are monozygotic or dizygotic is blood typing. Pattern (49%) says a histologic study of the separating membrane is needed to determine the category into which the children fall (Dolff) says “examination for vestiges of the deciduas capsularis” (43) will tell the story but that such an examination is not done because the interest is centered in the welfare of the mother and the child.

No one can be sure his is a single birth; for example, Dr. Aguero (4) reports a a case of delivery of a child and on second examination of the after-birth a papyraceous twin was found.

The existence of this papyraceous twin night easily have been overlooked, and the reported of a single birth would have been inaccurate. Since a dermoid cyst which is really a twin enclosed within the “other twin” sometimes is not discovered until adulthood, there may be some dermoid cysts not ever discovered.

Then twinning, sex and heredity are seen to be linked with stuttering and with blood incompatibility. Schienfeld (57%) says the age of the mother seems to be an important factor in the birth of fractional but not identical twins because the incidence of fractional twinning seems to increase with maternal age. If blood incompatibility enters into the picture of stuttering and all these factors have a common denominator, perhaps, the explanation is that the effect of blood incompatibility seems to be more pronounced with each successive pregnancy. In any event, it can be seen that a more careful study of twinning as related to the genetic history of individuals and speech and blood factors should be made.

The 17 male and 4 female cases studied by the author blend into the picture just given. None of the females was a twin and 3 of the males were, two being reported as monozygotic and one dizygotic.

Handedness

With twinning will be considered handedness. No tests were made to determine the accuracy of the reports but of the author’s 21 cases:

13 families reported other left handed members.
*3 families reported other lefthanded ancestors on maternal side.
*3 families reported other lefthanded ancestors on paternal side.
*2 families reported other lefthanded ancestors on maternal & paternal side.
* (Often more than 1 case was reported in an ancestral family)

Left handedness has long been considered as related to the jigsaw puzzle of the etiology of stuttering. Berry (14) made a critical review and study and concluded that it must be considered in the picture.

Phelps (323) maintains that the story of handedness would at least be our schools. He thinks a surprising number would be found to have sub-clinical symptoms of paralysis. (And it is a hypothesis of the author that these children would also shown symptoms of speech disorders) Bearing out Phelp's contention is the report of Wallace (312) who found 10% of the choreatic patients were lefthanded as compared to 3.3% of the non choreatic. And he found that the lefthandedness was present often before the chorea was demonstrated. In an experiment, Tsai and Maurer (302) produced sinistrality in white rats by producing a deficiency of Vitamin B in the mother.

There have been a number of studies of diet as related to pregnancy. (2B) Administration of Vitamin K is now recognized as a therapeutic measure in cases of blood incompatibility of the newborn. (205,239) Buraham (27) and Dalldor (44) believe that placental villi which allows the initiation of the antigen- antibody

process. Deficiency of Vitamin B (303) has been considered as a possible cause of the nervous symptoms associated with erythroblastosis.

Kernicterus, as explained in Chapter III, has been shown to have sequelae the C. N. S. and kernicterus is a disease entity of blood incompatibility of the newborn. Then kernicterus may be the etiological factor causing the symptoms Phelps has noticed and the lefthandedness in stutterers.

Rheumatic Diathesis

The stutterer has been shown by Berry (14) to have a constitutional predisposition to respiratory disease and to have them more severely than non stutterers. This constitutional predisposition, commonly called the rheumatic diathesis, has recognized sequelae of rheumatic fever is a familial disease which has close relationship to respiratory and tonsillar infections.

Berry (14) has related twins, and the male sex to such respiratory infections and to stuttering. There is still controversy as to the exact relationship of blood incompatibility of the newborn to the rheumatic diathesis. Potter (241) has observed but not proved that there is a high incidence of allergic manifestations related to such cases. Levine and Waller (179) say that it has been known for years that particles of placental villi break off and are transported by the maternal blood to various parts of the body, especially the lungs. Presumably

The villi can only break off when they are ruptured. Could it be that these particles have anything to do with a predisposition to a rheumatic diathesis? As stated above deficiency of Vitamin C has been studied as related to blood incompatibility of the newborn (27, 44) and in relation to malformations. (316, 317, 319) It is known that Vitamin C deficiency allows the capillary walls to rupture. If the deficiency allows the walls of the capillaries the chorion to rupture, particles could be carried to the lungs. This rupture would also allow the passage of the fetal blood into the maternal circulation. And therefore in cases of incompatibility antibodies would be found in the mothers.

All congenital disease which involve the respiratory mechanism or other mechanisms reflexly controlled in the medulla as well as those due to damage to a cranial nerve could, it seems easily be due to the kernicteric manifestation of blood incompatibility of the newborn. If the kernicterus occurs before birth, it is possible that before or during birth the damaged reflex center may cause damage to the respiratory mechanism and related mechanism such as the cardiovascular, and gastro intestinal. Since sometimes at autopsy lungs of infants dead from blood incompatibility have been shown to be damaged, it could follow that infants with milder symptoms who have lived would have damaged lungs. And those children could be the ones with the rheumatic diathesis.

Berry (14) says,” If we recognize the rheumatic diathesis as the common denominator in these disorders, specific in-

factious diseases, diseases of the lymphatic system, diseases of the mouth etc., disease of the trachea and bronchi, and allergy, then we also must take cognizance of the accepted sequelae of this group: nervous disorders." Such a study would be of the relation of stuttering to the C. N. S.

Central Nervous System

As stated in Chapter IV, West, Travis , and others have shown that stuttering has symptoms relates to disturbance of the C. N. S. Doctor (74) has stated that often the kernicteric patient does not live but f he does there is evidence of damage to different areas of the C. N. S. One area almost always affected is the basal ganglia and one sometimes affected is the medulla. Also in Chapter IV, it was shown that the speech disorders being considered have in common an emotional disturbance and according to Arey (5B) emotional control of action is centered in the basal ganglia. Stuttering involves an inability to control under emotional conditions, the respiratory mechanism, the reflex control of which is in the medulla. Rhythmic movement of the vegetative mechanism is related to rhythmic movement of the respiratory mechanism. Then this kernicterus damage to the medulla could be an etiological factor causing such inability.

West (321) has shown stutterers have a sluggish response of the facial and mouth muscles indicative of a slight paresis. The motor innervation of these muscles would be the VII cranial nerve the path of which is one of the areas often found damaged in kernicterus. Twitmeyer, (304) Trumper, (301)

Starr, (285) Travis (300) and Fossler, after studding hemato-respiratory balance, all agree that the disintegration of certain motor speech units involved in breathing is a factor to be considered. Palmer (226) showed that although as he groups older the child's heart normally slows in rate and establishes a rhythm, the heart of the child who stutters slows in rate but remains arythmic. These three, i. e., asynergy of the vegetative mechanism, imbalance of the hemato-respiratory system, and cardio arrhythmic might be accounted for by the fact that the medulla, where the reflex control of the mechanisms is located, is one of the areas often damaged in kernicterus.

Since this kernicterus, which could possibly be the cause of cerebral damage related to speech, is one of the pathologies of blood incompatibility, it is the author's hypothesis that blood incompatibility factor of such disorders.

The stutterer can sing even though he cannot announce the name of the song he is to sing. He can talk while dancing, doing rhythmic exercises or taking part in sports or work requiring physical exercise. Since the reaction of the damaged area is more asynergic than absent, anything which reinforces a rhythmic synergy helps the stutterer control the rhythm of the "fight or flight" mechanism. The fact that he can do the things mentioned above would seem to indicate that dancing or rhythmic exercise and work are "aiding" devices which allow the cortex a sufficient headway over the lower center to enable it to maintain control over the speaking mechanism.

Delayed Initiation of Speech

West (66B) thinks the child with delayed speech have the same inhibition as the stutterer, making it impossible for his muscles to form the sounds because the fascicles of the association pathways are paralyzed. Although feeble minded children as a group begin to talk at a later age than those who are intellectually normal, delay in ability to talk is not necessarily an indication of feeble minded ness. Davis (16B) says twins learn to speak later than single children. Stuttering is found more often among twins than among single siblings, (14) and stutterers are often related in learning to speak. However, stutterers tend to have normal intelligence as do twins. The mental retardation mentioned in relation to kernicterus is a broad term and may include children who have a delay in the initiation of speech. The child with delayed speech is often an emotional problem. Emotional instability is most often found damaged in autopsies performed when the death of an infant is due to kernicterus. Delay in speaking might be caused by a temporary cerebral lesion due to anoxemia and/or jaundice during the time the child is affected with the blood incompatibility process points strongly to a cerebral synthesis which is present at birth or occurs in the first year of life. Karlin & Kennedy (38) reporting on Flechsig’s study of the embryology of the brain say later development of speech in the male may be

caused by a later myelinization of nerve tracts in the speech pattern. The same could be true of a delayed initiation of speech by male of female. These defective tracts might be in any part of the speech synergy: the receptor, or hearing mechanism, the association fibers in the cerebrum, or the efferent motor tracts to the muscles. Doctor (74) says the important lesion in kernicterus is demyelinization and degeneration of the nerve fibers. It may be possible that sometimes the myelinization is only delayed. If the blood incompatibility mechanism was in action at the time the areas for speech were developing the myelinization might have been allowed up in its process of maturing, just as a fruit is critical period of development. Both the speech and fruit will mature and he undamaged if the blighting process is severe. If the blight is more serious the fruit matures but has “frozen areas”. Similarly speech will mature but there will be a slight defect or there may be a slight “general impairment”.

Another theory might be that as long as the jaundice remains, the speaking mechanism is inoperative but as the jaundice wears off, the mechanism is free to operate. Such a process would have a tendency to cause immature emotional, physical and mental development. If amyelinization is permanent, what has been called delayed speech may be aphasia. In some mild cases, practice in speaking makes the disorder unnoticeable after a time but in more pronounced cases, the child is unable to cope successfully with the problem and may be incorrectly classed

as retarded or even feeble minded.

The five factors Berry (14) lists as possible common denominators in the etiology of stuttering have been discussed and it has been shown that they all have a relation to the disease of blood incompatibility of pregnancy. Now other factors prominent in the case histories of stuttering and also in the case histories of disease of blood incompatibility of the newborn will be considered.

Prematurity

Twins are often premature and premature children are often twins. (31B) premature children have a tendency to be anemic. (31B) premature twins and anemic children are frequently seen in speech clinics. Tyson (305) says premature twins have a higher mortality rate than children of single births. The reason for might be that since the necessary quantities of iron, hormones, vitamins, etc. are not supplied to the embryo until the part of the pregnancy, premature children have a low supply and twins must divide even that. (31B) Premature infants are usually anemic and heir to many physiologic and metabolic maladies. All of them are not obvious in the neonatal period and sometimes include gastrointestinal symptoms which do not seem to have the usual etiology. (31B) Calvin (14B) Guild (30B) and Hess (31B) say premature infants are subject to tetany and the symptoms of tetany resemble closely symptoms found in some stutterers. Prematurity in the study of blood incompatibility

has been associated by Doctor, (74) Drummond and Watkins, (79) Tyson, (305) Yepes, (367) Yannet, (366) and others with abnormal labor, spastic paralysis, increased maternal age, placental praevia, dysfunction of the thymus gland, liver and spleen, stillbirths, cirrhosis of the liver, convulsions, mental retardation, anemia, and multiple births.

The Thymus Gland

The thymus gland cannot be kept out this picture. West (66B) on the basis of sex ratio and distribution curve established by the White house Conference in 1930 said, “As stuttering tends to disappear when a major endocrine metamorphosis takes place, it might be suspected that the cause number of stuttering cases especially among boys increases markedly from 6-10 years. Then the number diminishes as puberty approaches. The involution of the thymus gland follows this some curve. (57B) The etiology underlying the dyephemic phenomenon, however, is not removed as evidenced by the occurrences of stuttering under great emotional stress such as battle front conditions. It id well known that one of the problem of rehabilitation of veterans is stuttering which appeared or reappeared during service.

The failure of involution of the thymus gland has been observed by those who have worked with stutterers and in reviewing the literature of blood incompatibility of the newborn,

it was observed that the thymus gland is often reported as being involved. Leonard, (155) Doctor, (74) and others have associated dysfunction of the thymus seen I Rh blood incompatibility of the newborn with stillbirth, kernicterus, spleen, liver, kidney, adrenal cardiac, and respiratory dysfunctions and twins.

The lander (295) says that congenital adrenal cortical insufficiency is associated with macrogenitosomia, respiratory infections, persistent thymus, and familial occurrence. The etiology is obscure he says and evidently tied up with fetal development. He suggests a maternal – fetal interplay. Could this interplay be antigen-antibody reaction of blood incompatibility? Macklin (196) says the real effect of blood incompatibility in pregnancy may be an endocrine imbalance. Berry (14) reviewed the literature and made studies of her own. She found a correlation between glandular disturbance and stuttering both in the literature and her own studies.

In Routree's (55B) discussion of the thymus, he quotes Hemmar as saying it increases in size up to from 11 to 15 years and then normally involutes. This coincides with West's (66B) statement that there seems to be a correlation between stuttering and endocrine metamorphosis. Rountree (55B) reports that Friendlier, Basch, et al showed the thymus has an important bearing upon blood formation, nutrition and growth. All of these have been found deficient in blood incompatibility of pregnancy. However according to Rountree related action of the thymus to calcium metabolism. Kopp (143, 144) showed that the

relative balance between the inorganic substances of the blood of stutterers is not maintained. Could the lack of involution of the thymus of stutterers be the cause of or be related to the cause of this imbalance? Park and McClure (55B) after critically reviewing all the literature and experimenting themselves, decided the extirpation of the thymus may cause retardation in development and closure of the epiphysis. They thought also extirpation might alter the organs of internal secretion. Rountree (55B) reported experiments showing that "the thymus gland of parents is in some way concerned with may be associated with the fact that hyperthyroidism in the mother causes hyperthyroidism in the child.

"Status thymicolymphaticus is a condition often encountered in newborn infants and is supposed by some to be due to disease of the thymus gland." (7B) The thymus is enlarged. West (66B) says of this pathology that "many a case of delayed metamorphosis or development" is caused by it and that" its effects upon speech are to render it infantile and mechanically clumsy." Jacobi (55B) says that since the space between the manubrium sterni and the vertebral column in an infants is very narrow (2.2 cm) an enlargement of the gland may produce:

1. Thymic asthma or thymic strider in which breathing is difficult and the difficulty is aggravated by crying, coughing, exertion, anger, throwing back the head and by acute infections.
2. Status thmicymphaticus, "a condition in infancy and childhood associated with "hypoplasia of the heart

> and blood vessels." Victims of the disease are subject to attacks of cyanosis the patient suggesting a picture of suffocation. "Such children are abnormally sensitive to infection."

Stutterers have a predisposition to respiratory infection and seem to have infections more severely than normal. (14) Rountree (55B) quotes Time as saying in regard to status thymicolymphations, "the children mature very slowly. _____.... The developmental abnormalities range from almost complete infantilism to the presence of a maxillary torus in an otherwise structurally perfect individual." Stuttering is associated with infantilism and also with high palatal arch. Time goes on to say that physiologically such children lack a resistance to fatigue and infection. Berry (14) has shown that stutters have this same lack of resistance; chemically they are predisposed to low blood sugar, acidosis, prolonged coagulation time of blood, (administration of vitamin K to decrease the coagulation time of blood is one therapeutic measure indicated in disease of blood incompatibility of the newborn.) and usually lymphocytosis; behaviouristically they remain infantile, follow the path of at least resistance, shirk responsibility and lack the ability to concentrate. "Consequently of stutterers that they tend to follow the path least resistance, shirk responsibility, lack the ability to concentrate, and feel inadequate and inferior.

Rountree (55B) says "enlargement of the thymus is often accompanied by enlargement of the tonsils and the tonsillar

ring.” Berry (14) found a high incidence of tonsillitis among stutterers.

Although recent investigations have thrown doubt upon the existence of status thymicolyphasticus, it is agreed that the thymus is involved but attention has been called to “allergic swelling”. (55B) Berry (14) found a relationship between stuttering and allergic manifestations. Potter (240) thought she observed, but hadn’t proved, that there was a high incidence of allergic manifestations in cases of disease of blood incompatibility of the newborn. In the 21 cases studies by the examiner.

26 cases were reported in 21 families.
4 cases were stutterers themselves.
18 families had cases of allergic manifestation.
6 families had cases among the paternal ancestors.
19 families had cases among the paternal ancestors.
2 families had cases among the paternal & maternal ancestors.
(There were many cases in some of the families)

Attention has also been called to the relation of the adrenals, mechanical pressure, and biological effects. Excess thymus extract has been shown to kill quickly by inducing complete auriculoventricular dissociation or involution of the thymus, the lack of cardio rhythm associated with stuttering, and the mention of “thymus” and damage to the medulla which was observed in histories of Rh in compatibility of pregnancy have a common denominator?

Use of X-ray therapy for thymus gland dyafunction has been associated with later evidence of mental and/or

physical retardation. Rountree (55B) feels that as a result of X-ray treatment in the young, the medical profession is either:

1. Injuring the thymus in a considerable number backward children, or
2. Producing backwardness in children through destruction of the thymus in infancy.

It may be possible that the same factor which caused the thymus to fail to involute, caused the “backwardness”.

As stated before there has been no study but “thymus was observed to be prominent in case histories of blood incompatibility of pregnancy, and stuttering and related factors such as have been discussed: twins, familial incidence, obscure etiology, infections, predisposition to lack of maintenance of proper ratio of inorganic substances of the blood, allergic manifestations, and cardiac arrhythmia.

Psychogenic Manifestation

There has always been controversy as to whether the psychogenic display causes the physical display of dysphemia or vice versa. Stutterers can usually talk to babies and pets when alone or in any situation where the “fight or flight” mechanism is not involved. The individual has very little control over the action of this “fight or flight” mechanism which is initiated in the medulla. The rate of diadochocinesis, especially of the face and mouth muscles, of the dysphemia person is in proportion to the possession of the stuttering phenomenon of the individual. (321)

As stated in the chapter on kernicterus, one of the areas of the C. N. S. most frequently damaged in the occurrence of

the pathology is the basal ganglia through which is transmitted emotional control of movement. This could easily account for the difference in motor reaction of the same set of muscles when the vegetative function is concerned and when speech function is required. When the emotional stimulation is great, the cortex loses control. Those dysphemic persons, who when alone cannot read or talk without stuttering, would be assumed either to have a greater damage to the basal ganglia than the individuals who can read or talk alone, or to have greater stimulation which allows the basal ganglia to gain control.

Bottle Feeding

It must be remembered that the incentive for this study was the observed that children with dystomic speech were usually bottle fed.

In the cases studied by the author the following table gives a summary of the feeding story.

Infant Feeding

Table I

1	2	3	4	5	6	7
Case	Number	Entirely Breast Fed	Entirely Bottle Fed	Partly bottle fed & partly breast fed.	Fed with dropped etc.	No report
Stutterer	21	3	13	5		
Delayed speech	19	1	14	4		
Aphasic	12	3	7	2		
Cleft palate	17		10		7	
Total	69	7	44	11	7	0

Table II

Reasons For Bottle Feeding

1	2	3	4	5	6	7	8
	Mother's milk insuf-ficient	Breast Infec-tion	Mother too nervous	Doctor's orders	Because of palate	Other reason	No reason given
Stutterer	13	1	1	2			2
Delayed speech	10	3	2			2	1
Cleft palate	9	1			7		
Total	39	5	4	1	7	2	4

It is not known how many in column 6 of Table I would have had as insufficient amount of milk or would have had an infection if the child had nursed. And it is not known how many in columns 6. Table II would have fitted into columns 2 – 3 – 4 or 5. However, even without this information it is seen that for some reason children with dystomic speech are unable to breast feed because of maternal incompatibility of some kind.

Davidson (60) says it has known for decades that an erythroblastotic infant cannot thrive on its mother's milk. Darrow (52) in1938 said that in the breast milk of some mothers there was a poison for the infant which was transmitted before birth through the placenta, and after birth by means of breast milk. She and co-workers demonstrated a difference in the Hb of the two bloods. The possibility that breast milk was important in the patholofenesis of erythroblastosis fetalis was suggested by Hlparin. (109) Then it was demonstrated that the Rh antibody was present in the milk and/or colostrums of a pregnant woman who was isoimmunized and might increase the

severity or cause the onset of the disease after birth. (359, 360) The same phenomenon was demonstrated by others, (35, 243, 325) and is now accepted as one of the pieces of the jigsaw puzzle of Rh incompatibility. (109) Undiscovered parts of the jigsaw puzzle are:

1. Why these children normal at birth often develop the pathology a short time afterwards. Perhaps this is due to the fact that sometimes no antigens pass to the mother until delivery, and antibodies then only reach the child through the milk, which has a relatively higher titer for the first few days.

2. Why 25% of all children are jaundiced at birth and recover sometimes spontaneously. Perhaps, children recover the antibodies in the blood at birth are so few that they are soon neutralized. If the child is not breast fed no antibodies are introduced.

3. Why the duration of neonatal jaundice which wears off spontaneously lasts up to about two weeks. The duration of jaundice may wear off spontaneously in about two weeks because as Cappell (32) says the agglutinin content of the mother's milk at that time falls to such a low level that it isn't dangerous. If the child is not breast fed, the agglutinins present in the child's body at birth are neutralized and no new ones are added.

Several suggestions as to undiscovered parts of the jigsaw puzzle and as how the puzzle fits together might be offered.

1. Witebsky (359, 360) says antibodies pass from the mother to the child through the placental circulation and the breast milk.

2. One peculiarity of the intestine is the tendency during the first ten days of life for undigested proteins (and antibodies are protein) to be absorbed and thus gain entrance into the blood stream and furnish primary sensitization if it hasn't occurred <u>in utero</u> by way of the placental circulation. (361)

3. Halparin (109) thought Rh antibodies demonstrated in the mother's milk in instance of incompatibility were probably the cause of the onset of disease after birth or contributed to the severity of the disease.

4. Dr. Elizabeth Berry (15) made a study of breath feeding and bottle feeding and came to the conclusion that babies should be breast fed because, though bottle fed babies weigh more than breast fed ones they are less resistant to infection. She found the morbidity rate twice as high in the artificially fed and the mortality rate ten times as high; but in her study she fed because the mother could not nurse them and ones who were bottle fed because of convenience.

Dr. Mildred Berry (14) has shown that stutterers have a predisposition not only to infection but to severe infections especially those of a respiratory nature, and Dr. Elizabeth Berry (15) says the morbidity and mortality rates among artificially fed infants are higher than those among breast fed infants. Tables I and II on pages 83 & 84 show that some reason stutterers have to be bottle fed because the mother for some reason cannot nurse the child. Could it be that the greater morbidity and mortality of bottle fed infants is the direct result of a condition which necessitated bottle feeding rather than of the bottle feeding itself?

The author would be interested in knowing how many of the children Dr. Elizabeth Berry studies had to be bottle fed because for some reason the mother could not nurse them.

Dr. Elizabeth Berry (15) says infants who have pyloric stenosis must be breast fed because such feeding aids in the success of any medical and surgical treatment. Ballantyne (11B) says that pyloric stenosis is "a functional disorder of

the nerves of the stomach and pylorus leading to an ill-co-ordination and therefore an antagonistic action of the muscular arrangement resulting in hypertrophy". Lehman (11B) says it is a congenital condition which produces a reflex irritation and stimulation to the nervous mechanism causing spasms of the musculature which in turn give rise to the hypertrophy. The observation may be significant that pyloric stenosis develops during the same period as neonatal jaundice and anemia occur (2 week) after birth. (11B) It is congenital and inheritable, and occurs four times as frequently in males as in females. It has been reported in twins and occurs more often in bottle fed babies. (11B) as the reflex control of the pylorus and stomach (Vagus nerve) is centered in the medulla, pyloric stenosis may be involved in the same damage which could cause stuttering. If they are caused by Rh incompatibility, breast feeding would be contra-indicated. If the damage is from some other source such would not be the case.

Oberhelman (220) related bottle feeding to congenital tricuspid stenosis, feeding to tetany, rickets, profound disturbance of the blood; Davidsohn, (56) Darrow, (53) Witebsky, (359, 360) and others showed that in disease of blood incompatibility of pregnancy there was an antibody transmitted to the infant by the mother's milk and authoer has shown that children with the speech disorders investigated usually have to be bottle fed because of some maternal-infant incompatibility. It would seem then that "feeding" holds a prominent place in the

jig saw puzzle which, when pieced together , may help explain the etiology of stuttering.

Chorea

The dysphemia person often has accompanying spasms which resemble the choreatic spasms of the spastic. Often he throws or pulls his head up spasmodically, throws or pulls it to one side or throws or pulls it down. By some this has been considered the stutterer's avoidance of facing the person to whom he is speaking. Berry (14) says 9.3% of the stutterers she studied had face, head, and shoulder tics while only 3.3% of the normals had them. (It must be remembered that when a control group for stutterers is taken, there may be potential stutterers in the group). Since a stutterer always has the same pattern of spasms and they and centered in the neck and face muscles more often than in may other group of muscles, a possible explanation is that, as the damage to nuclear centers in and near the medulla. Best and Taylor (7B) say convulsions may be caused by hyperactivity of the respiratory center. The superior cervical ganglion which partially controls the neck muscles (15aB has a twig connecting to the carotid sinus reflex and that reflex is controlled is controlled in the medulla. (7B) the medulla it must be remembered is one of the areas found damaged in autopsies of kernicteric patients.

West (66B) has called attention to the fact that those

children who have speech symptoms associated with choreaform movements, when they become conscious that their speech is different from those around them, exhibit a tremor which resembles a stutter; in fact, he thinks it may have the same paychogenic origin. Since the thalamus is an area with poor blood supply it would be expected to be one of the first areas of the brain to be damaged in disease involving blood supply. This has been found to be the case in kernicterus. (Chapt. III) if the child with choreaform movements and the stutterer both have brain damage common to both.

Injury Incident to Birth

A cerebral damage obviously may have other causes than kernicterus and it is known that cerebral palsy has other causes. Ford (15B) says 6% of the cases of cerebral palsy are due to instrumental birth injury. Instrumental births are associated with stuttering. Since instrumental birth injury has been shown to cause brain damage in cerebral palsy, it would seem logical that birth injury due to instrumental birth may cause stuttering.

Perlstein (231) says the most common cause of congenital cerebral palsy is anoxia. And he names prolonged labor and abnormal presentation in delivery as causes of anoxia. The prolonged labor may cause the child to breathe before it is born and in so doing aspirate mucous or the contents of the

womb. Uterine atony during the first two stages of delivery causes prolonged labor. Then when the fetus is expelled, the contractions and retractions become normal. (6B) This would seem to indicate incompatibility between the mother and the fetus. Could it be blood incompatibility?

If the child is not born head first, his oxygen supply may be cut off before he has access to the outside air. The umbilical cord clamps shut, cutting off the maternal supply, as contact with the outside sir cools it; and as the head is enclosed, the child suffocates or drowns according to whether or not breathing has been initiated.

More children born feet, or breech, presentation have cerebral palsy than those born head presentation. (231) Perlstein thinks that probably the paralysis is not due to the manner of presentation, but that the manner of presentation is due to the paralysis. Could it not be that they have a common etiological factor?

Travis (69B) says there a definite relation between the manner of presentation in delivery, and speech disorders and this has been the author’s clinical observation. Also it has been the author’s clinical observation that prolonged labor is frequent in case histories of stillbirths with prolonged labor, Doctor (74) has associated stillbirths, prolonged labor and kernicterus. Yannet (363) has associated cerebral palsy, prematurity and increased maternal age with prolonged labor.

It was observed in the literature in regard to blood incompatibility

that in cases in which delivery was mentioned, abnormal presentation and prolonged labor predominated. Yannet (363) quotes Murphy as saying possibly some maternal factor might be responsible for reproductive inefficiently in cases of prolonged labor. Could this be an antigen- antibody reaction? Unger and Wiener (306) say there is some indirect evidence to suggest that the maximum amount of fetal blood escapes into the maternal circulation at the time of labor and delivery perhaps due to a hormonal and nutritional factor. Could the nutritional deficiency which has been considered by some as being the causative factor in the initiation of the antigen-antibody reaction be related to prolonged labor and abnormal presentation in delivery?

Common delay after birth in the development of signs of erythroblastosis fetalis suggests that a factor associated with delivery may be of great importance in the study of the disease. The fact that kernicterus is very common in infants who develop icterus only after birth and the fact that certain infants dying of kernicterus have little oi no signs of long standing injury due to maternal antibodies suggests further that these factors associated with delivery may be of such significance as to account for kernicterus in some babies. (20, 197, 221, 309, 222, 214, 94) Of course, such meager knowledge does not mean that there are more abnormal presentations in such deliveries because very few cases such information. However, since it has been shown that spastic speech and stuttering

have common symptoms which could be caused by anoxia, and cerebral palsy has been associated with these two abnormal phenomena of unknown etiology which cause anoxia, the possibility that stuttering is either caused by them or has a common etiological factor with them should be investigated.

The histories of pregnancies of the mothers of the 21 cases studied by the author recorded 6 instrument deliveries 3 were considered difficult but the manner of presentation and length of labor were not reported; 8 involved anemia, 3 jaundice, 2 dropsy; 8 were miscarriages (4 to one mother); 2 were cases of erythroblastosis fetalis. Two families reported erythroblastosis on the maternal side and 1 on the paternal side. One family reported a case of polydactylism on the maternal side, 1 a mongol in the immediate family, and one reported mongol and hemophiliac cousins. His information cannot be considered accurate because people tend to conceal information about which they are sensitive or ashamed, and because the attending physician probably did not record anything considered nonessential to the welfare of the patient. If the hypothesis of this study is correct, subclinical symptoms which may not be recorded at birth may be related to the cause of dystonic speech. In a study of infant mortality during the first year De Porte and Parkhurst (64) found that in some states, though the birth certificate had record, the death certificate recorded some anomaly of birth as the cause of death. They therefore concluded that the absence of any record of anomalies on the birth

certificate cannot always be accepted as proof that none were present.

In the literature many cases are reported of infants with no seeming pathology who died and at autopsy showed kernicteric signs. (306, 74) Others thought to have jaundice or anemia, when death occurred unexpectedly, were found at autopsy to have had kernicterus. (74, 93) Others having no noticeable symptoms later developed evidences of C. N. S. damage. (74) Such symptoms have been recognized as spasticity or mental retardation; (94, 371, 372, 142) but Crothers (15B) says caution must be exercised in disgnosis, since C. N. S. damage in the infant may not manifest recognizable symptoms until the time at which the infant should walk and talk. Perhaps stuttering is another of the symptoms which if carefully studied neurologically could be identified infancy.

Phenomenon of Childhood Occurrence

Craig (11B) says kernicterus is associated with erthroblastosis fetalis, lesions in the gastrointestinal tract, congenital heart, tetany, urinary infection, prematurely, asphyxia, liver and blood dysfunction, and familial incidence, and does not occur in adults. Blood incompatibility of pregnancy is obviously a phenomenon of antenatal, natal, and neonatal life. Development of speech is manifested in childhood but is presumably influenced by antenatal, natal and neonatal conditions. Myelinization of cerebral nerve fibers which it is assumed is related to speech development occurs in and is affected by conditions

of antenatal, natal and neonatal periods. All of the factors related to kernicterus have been related in clinical observation to stuttering.

Children who have dystonic speech have peculiarly set facial expressions not observed in individuals who show speech disorders later in life unless the adult-acquired disorder is accompanied by or is a result of a paresis. Potter (242) says renal dysfunction. Clark (33a) says, “most erythroblastotic babies have mongoloid faces”.

Infant tetany; congenital heart; pyloric stenosis; Susceptibility to respiratory, urinary, hepatic, hematogenous and gastrointestinal infections; bottle feeding; convulsions and predominance of male incidence of all have been associated with each other and with the pictures presented by blood incompatibility of the newborn. (3B, 8B, 11B, 14B, 40B,50b, 58b, 70B) Calvin (14B) maintains there are cases of infant tetany due to brain lesion not dependent upon brain trauma. Though it can be caused by cerebral injury from forceps, he maintains there is evidence it is also caused by eclampsia, placenta praevia and prematurity all of which he says are associated with congenital heart disease, asphyxia, bottle feeding, convulsions, rickets and male incidence.

Craig (11B) calls attention to the frequent relationship of intracranial hemorrhage and intrapulmonary and urinary infections.

A factor in the etiology of cardiospam is a defective vegetative nervous system on an hereditary basis. (1, 14B) West (66B) says speech is an overlaid function of the vegetative mechanism.

Calvin (14B) gives a good description of stuttering when he says that among other symptoms infant tetany shows:

1.Severe or slight laryngospasm.

2.Spasm of facial muscles imparting stiffness to the face.

3.Hyperirritability of the motor nerves.

Involvements of eye and ear, delayed acquisition of motor skills, disease peculiar to infancy (14) prematurity have been related to stuttering. Dr. T. H. Best in an extensive study of the ear found that otosclerosis has its beginning at about the mid period of fetal --- at least that is the time at which the anatomical change is first seen. The ossification defect occurs in a large number of fetuses but in many fetuses corrects itself during the last months of fetal life and remains as a slightly defective area in the capsule as in such cases it apparently never progresses further, it causes no difficulty. Nature makes each time. If some vital part becomes involved, “hardness of hearing” sets in due to hardness of the vital part.

The cause is not known. The chemical changes have not been analyzed; it maybe some nutritional problem or it may be a

hormonal one. Could it be that deficient nutrition enters the picture by allowing a blood incompatibility to become effective? And that otosclerosis is another pathology which should be investigated in the light of blood incompatibility?

All of these pathologies with unknown or unconvincing etiologies are associated with the neonatal period. The dystonic speech disorders discussed in this study have unknown or unconvincing etiologies, are associated with the life and clinical observation has associated with kernicterus.

As kernicterus is a pathology resulting from blood incompatibility of the newborn, a common etiological factor would be indicated which might possibly reside in blood incompatibility of the newborn.

Diseases Later Than Birth

Berry (14) concluded that the diseases most immediately and most frequently associated with the inception of stuttering are specific infectious diseases of the respiratory tract and diseases of the nervous system. If the hypothesis of the author is correct, stuttering can follow any disease which damages the medulla and basal ganglia just as cerebral palsy is a sequela of any disease which damages certain areas of the brain. The historic explanation for this theory is that the stutter is able to manifest itself. An alternative theory is that the stutterer is predisposed to the rheumatic diathesis because

the respiratory center and the basal ganglia are inefficient and easily damaged. Perhaps the disease really starts the stuttering instead of allowing the latent stutter to manifest itself.

Nelson (215) found, “There is a constant ratio favour of the greater incidence of diseases and other precipitating factors occurring at the incidence of stuttering among stuttering propositi who belong to pedigrees showing no stuttering in their ancestry, than among stuttering propositi who belong to pedigrees with stuttering in their ancestry.” This would seem to indicate that it might not be the stuttering which is hereditary but a predisposition to something which can cause the stuttering.

It can only be concluded from this that all stuttering may not be hereditary. Possibly the predisposition to the rheumatic diathesis “is inherited and when the individual has a severe infection, the respiratory center is damaged. What we call a “predisposition” may actually be an inefficient but not damaged center.

Accidents

In the same manner it would be logical to conclude that stuttering could be caused by any brain injury from accident but it would be supposed such an impairment does not often occur to be damaged and because of the fact that one of those areas is such vital spot that the result of the accident would usually be fatal.

Nationality and Blood Tests

Haldane (107) and Wiener (324) say subgroups and sub-factors are probably due to mixture of races. In the 21 stuttering cases studied by the author the only significant finding was that the percentage of German blood was only 25% although in the state of Wisconsin the normal expectancy would be 40%. This result may not be significant since the factor causing the low percentage was that three cases were of 100% Russian Jewish percentage and one of 100% English. In a small group four such cases would have a significant effect upon percentages. Blood tests of sixteen of these stutterers and their parents (48 tests) were 100% Rh positive. Since the number examined was very small, no positive conclusions can be drawn. Since, however, the cases of cleft palate tested did not show such uniformity, there is an indication that the results are significant.

Aphasia

(The term aphasia as here used means all forms of language inability in all degrees.) According to Travis, Henry Head define aphasia as (69B) disturbed physiological activities in certain parts of the cerebral cortex resulting in impaired capacity to use language. The aphasia demonstrates a lack of ability to shift mental attitudes which is analogous to the scanning speech and the ataxic gait of the spastic. In fact, Huber (125) has shown that the aphasic demonstrates a form of muscular spasticity in his inability to shift from surds to

sonants; this inability may be mental or physical but in any event would seem to indicate that aphasia is related to spasticity.

West (66B) considers that aphasia is etiologically related to spastic paralysis: “In fact, it may be associated with spastic dysathrias, or either may be present without the other.” West also says aphasia is a specialized amentia. In aphasia, the lesion is in the association areas of the cerebrum in motor skills. Like the stutterer and the spastic, the aphasia demonstrates a disturbance of emotional control which it is assumed has resulted from damage to the basal to the basal ganglia.

Although there are two peaks of incidence of aphasia, baby-hood and old age, this study is concerned only with the former. Klingman and Carlson (142) associated mental retardation with kernicterus before blood incompatibility was discovered. After the discovery of the incompatibility, Doctor, (74) Gimson, (93) and others (371, 372) maintained that one result of disease of blood incompatibility of the newborn was mental retardation. The “backwardness” spoken of by Rountree in discussing thymus dysfunction may be the same “mental retardation”. As aphasia is a specialized amentia perhaps some of this “backwardness” associated with status thymicolymphaticus and also with blood incompatibility of the newborn is really aphasia. Delayed speech has been associated with aphasia. Possibly the difference between delayed speech and aphasia is the difference in the severity of the “blight”. When the aphasic begins to speak

the jaundice has faded as jaundice always fades but a traumatized area has been left. When the child with delayed speech begins to talk not only has the jaundice faded but only slight, traumatized areas have been left. The difference might be explained on the basis of difference in development at the time of the onset of the disease, an early onset preventing mylinization and a late one delaying the myelinization or inhibiting this ability of the nerve to function as long as the jaundice is present. Whatever the process stuttering, delayed speech, spastic speech and aphasia would frequently seem to have an etiological factor in common kernicterus. Kernicterus could be responsible for the symptoms which the disorder have in common; i. e., sluggishness of response and lack of emotional control of movement. In each of the four types of disordered, the area controlling emotional stability, the basal ganglia, would be response in each of the four different manifestations would differentiate the four disorders.

In this chapter, it has been shown that the four speech disorders studied have common symptoms which may have common etiological factors and that those factor may frequently reside in blood incompatibility of pregnancy.

Cleft Palate

As the original study included cleft palate, a summary of the findings in regard to that pathology will be given at this time. In the etiology of cleft palate, the possible inheritance of at least two heredity recessive genes, one partially sex linked, is mentioned more than any other one cause.

Among other causes of cleft palate are mentioned (1) poison in the mother's system; (2) pituitary dycfunction; (3) endocrine imbalance; (4) changes in the amniotic fluid; (5) malnutrition; (6) hypothyroidism; (7) position in the uterus; (8) intra uterine pressure.

Abnormalities associated with cleft palate are: monstrosities, spina bifida, congenital blindness, underdeveloped head, congenital heart, club hands and feet, anencephalus, hydrocephalus, myoceles, supernumerary fingers and toes, oversized fontanelles, absence of extremities, twinning.

The incidence of occurrence of cleft palate seems to rise in later birth ranks and in offering of older mothers.

It has usually been considered that there are twice as many males as females among cleft palate cases, more twins than single births and more premature than full term births. (See figure of the Wisconsin state Department of Public Instruction). The supervisor of the Cleft Palate Clinic of the State of Wisconsin has observed, but hasn't proved, that more cleft palate children prefer the left hand than the right and that they tend to be retarded in growth.

This brief summary based on a study on file at the Wisconsin Bureau for Handicapped Children seems to indicate that a description of the study o cleft palate gives conclusions coincident with the study of blood incompatibility of pregnancy. Taking the statements reported in the first part of the above discussion the following explanation is offered:

Heredity

Blood Tests of Cleft Palate Children

	Results of Test			Nationality				Type of Cleft
				Paternal		Maternal		
Cases	Father	Mother	Child	Father	Mother	Father	mother	
1	Rh-	Rh+	Rh+	Pol.	Pol.	Pol.	Pol.	C. P.
2	Rh+	Rh-	Rh+	Oer.	Swiss	Penn. Dutch		Rt. Hl & c.p.
3	Rh+	Rh+	Rh+	Nor.	Nor.	Pol.	Pol.	C. P.
4	Rh-	Rh-	Rh-	Ger.	Ger.	Ger.	Swiss	Left hl & C. P.
5	Rh-	Rh+	Rh+	Ger.	Ger.	Wis.	Wis.	Rt. Hl & C.P.
6	Rh+	Rh+	Rh+	Ger.	In.	Ger.	Eng.& Dutch	Bi. hl & C.P.

It is seen that the presence of a recessive gene cannot be ruled out although there is no conclusive evidence of its presence. Wiener (355) says of blood incompatibility that search must be made for an influencing factor which may be a recessive gene as stated above one theory of the cause of cleft palate is that it is inherited by a recessive gene. It might prove significant that the two cases having cleft palate without harelip do not have German in the percentage.

The other causes mentioned above have been related at least

Indirectly to blood incompatibility of pregnancy. (1) Poison in the mother's system, (2) pituitary dysfunction, (3) endocrine imbalance, (4) changes in the amniotic fluid, (5) Malnutrition- experiments have been made with different deficiencies in diet and the experimenters have concluded that deficient diet is an etiological factor in the cause of cleft palate. Burnhan (27) says since a deficiency of Vitamin C. causes capillary walls to rupture, a deficiency of Vitamin C in pregnancy would cause chorionic villi to rupture. This would permit the passage of blood from the fetus to the mother. If the blood is incompatible, antibodies will be formed which pass back to the fetus as it is normal for protein to pass and antibodies are protein. (6) hypothyroidism, and (7) position in the uterus could be ascribed to the same cause as the abnormal presentations in delivery which Perlstein (231) says are due to spasticity (in cases of same etiological factor as the spasticity since stuttering, delayed speech, and aphasia manifest the same abnormal presentations but not the same spasticity. (8) Intrauterine pressure are factors which seem to be "in the picture".

Abnormalities

The abnormalities mentioned have been related either directly or indirectly to blood incompatibility. As was shown in the first part of this study.

Incidence of Occurrence

The incidence of occurrence in later birth ranks and in offspring of older mothers may be explained on the theory that it takes some time for the antibodies to be formed in a sufficient amount to cause damage. It has been considered that first children are seldom affected for this reason. (240)

Sex Twins Prematurity

It was the author's observation in reviewing the literature in regard to blood incompatibility of pregnancy that the male sex predominated and was affected more severely than the female sex.

It was also observed that twins were often mentioned. If the twins were of opposite sex, the male twin frequently seemed to be more severely affected than the female.

Many of the cases reported were premature and the symptoms associated with prematurity were prominent in the literature – such as related growth as mentioned in the first part of the discussion cleft palate.

Left handedness

It has been observed cleft palate children often prefer the left hand. It would be interesting to investigate whether left- handedness is characteristic of those having a cleft on the left side, and whether there is less dexterity in the use of both hands among those having a bilateral cleft.

Bottle Feeding

The original incentive for this study was the observance that children with certain speech disorders were bottle fed and that Rh antibodies were discovered in breast milk. Of 17 cases studied by the author 9 were bottle fed because the mother did not have sufficient milk, 1 because the mother had a breast infection. Of the 7 who were fed with dropper, syringe, bottle etc, because of the cleft palate, it is not known how many would have had to be bottle fed because of insufficient milk, breast infection etc if the same thing that causes the palatal malformations also prevents successful breast feeding by the mother. Perhaps it is nature’s method of preventing further damage.

There seems to be less conclusive evidence of the relationship between blood incompatibility and the etiology of cleft palate than is true in the cases of the other four disorders studied. However, it seems that a study of vitamin deficiency and blood incompatibility might be profitable. Also a racial study related to the extent of the cleft might throw new light on the subject.

CHAPTER VII

As the study of the Rh factor is in fluid stage, nothing can be definitely stated but the following conclusions in regard to blood incompatibility of pregnancy and stuttering, spastic speech, delayed speech and aphasia are implied in the literature studied:

1. Stuttering, spastic speech, delayed speech and aphasia may have a common etiological factor in disease of blood incompatibility of pregnancy.
2. If there is a relationship between blood incompatibility and stuttering, spastic speech, delayed speech and aphasia the form of the disease causing the damage may be kernicterus.
3. Stuttering, spastic speech, aphasia and delayed speech may be due to brain damage of certain areas: the area damaged determining the form the disorder manifests.
4. The same symptoms which seem to be prominent in case histories of the four speech disorders discussed seem to be prominent in disease of blood incompatibility of the newborn.
5. There may be an inherited predisposition determining the area of the brain which is damaged.
6. Stuttering is the manifestation of damage to the medulla and basal ganglia mainly and the extra pyramidal system to a less extent. This damage is exhibited both organically and functionally.
7. choreathetotic speech is the manifestation of damage chiefly to the extra-pyramidal system and the basal ganglia, and the medulla to a extent. (Of course the spastic demonstrate paralysis of any muscle group.)
8. The extent of damage to the medulla and the extra pyramida system determines whether the individual is a stutterer with choreathetotic symptoms of a choreathetotic with stuttering symptoms.

9. The location of cerebral damage which manifests aphasia has already been determined by Head and others. Any individual who has had kernicterus or any other transumption of the brain could have a dystonic speech with the symptoms of one or all of the types of speech discussed unless a predisposition determines the area of the brain damaged.

10. Delayed speech it would seem is due to:
    1. kernicterus which off and allows the speech to develop, or
    2. The inhibition of maturation at a critical period.

11. Stuttering may be caused by any accident which could cause damage to the basal ganglia and the medulla; and in addition to kernicterus, such a disease may be any infection that is accompanied by a high fever.

12. Stuttering could be caused by any accident which could damage the basal ganglia and the medulla. But it would be supposed such an impairment seldom occurs because the medulla is such a vital spot that such an accident would usually cause death.

13. Stuttering, aphasia and delayed speech are all specialized forms of cerebral palsy.

14. Only some cases of stuttering, spastic speech aphasia and delayed speech are hereditary.

15. Deficient diet is probably an important etiological factor in the cases of such hereditary speech disorders as are discussed in this study.

16. Probably a study of all congenital diseases in relation to blood incompatibility and speech disorders would help complete the missing links both in the study of blood incompatibility and the study of speech disorders.

17. Either incompatibility between the mother and the fetus or the same factor which causes the incompatibility, seems to cause the mother to tend to be unable to nurse the infant.

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