FEEBLE-MINDED CHILDREN.

AN INQUIRY INTO MENTAL DEFICIENCY IN SCHOOL CHILDREN WITH SPECIAL REFERENCE TO SYPHILIS AS A CAUSATIVE FACTOR AS DETERMINED BY THE WASSERMANN REACTION.

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I N T R O D U C T I O N.

Any attempt to classify the Mentally Defective is met with considerable difficulty. Hitherto they have been classified according to the degree of defect into Idiots, Imbeciles and Feeble-minded. Well marked types of each degree are easily distinguished, but there can be no hard and fast line drawn between the various grades, the feeble-minded slipping insensibly into the imbecile, and the imbecile into the idiot. Such a division is convenient for purposes of education and treatment but it cannot be regarded as anything save a tentative classification.

My investigation is confined to the group known as feeble-minded.

The term 'Feeble-minded' has been used in America to "include all degrees and types of congenital defect, from "that of the simply backward boy or girl, but little below "the normal standard of intelligence, to the profound idiot, "a helpless, speechless, disgusting burden; with every de-"gree of deficiency between these extremes." There is therefore some confusion as to the exact meaning of the term 'Feeble-minded' and the Americans suggest the use of the word 'Moron' as indicating the lesser degrees of mental defect/

defect. As this term has not come into general use I have adhered to the word Feeble-minded as indicating the lesser degrees of mental defect, defined by the Royal Commission on the care and control of the feeble-minded as "persons who may be capable of earning a living under favour-"able circumstances, but are incapable from mental defect "existing from birth or from an early age (a) of competing "on equal terms with their normal fellows; or (b) of manag-"ing themselves or their affairs with ordinary prudence", and in the case of children are incapable of receiving benefit from instruction in ordinary schools. School Authorities are empowered by the Defective and Epileptic Children (Education) Act, 1899, to provide special schools for such children.

My inquiry is based on the examination of the feeble-minded (using the term in its English sense) children admitted to, and seeking admission into, such special schools. I have not included in the investigation any children who have been refused admission either on account of imbecility, or whom I have considered to be cases of delayed development, the co-called "merely dull and backward." The defect is perhaps only a question of degree, but I am inclined to think that investigations into groups of the different degrees/

degrees may bring out factors in the causation of the defects which will be of importance from a prophylactic point of view. The feeble-minded child is rarely found in an institution, and in many cases is to all external appearance normal, yet is incapable of competing with his fellows, or of managing himself or his affairs with ordinary prudence, and, unless under very favourable circumstances, is likely to become a social wastrel. He is capable, under careful training and guidance, of a certain amount of education and of becoming a more or less useful member of society. Again, there are various degrees and grades among the feeble-minded children and any attempt to classify them presents very great difficulty. A classification according to physical characteristics is impossible. The presence of physical abnormalities or physical defects gives no indication of the degree of mental defect, although it will be seen that physical abnormalities and developmental defects are more frequently present in the feeble-minded than in the normal child.

A classification according to speech has been suggested, but this is not practicable. Although many defective children do present speech defects, an equal number have normal speech, while severe degrees of speech defect are quite/

quite compatible with ordinary intelligence.

A classification on a Psychological basis is a future possibility. The tests devised by Binet and Simon for measuring the intelligence of the child are the first step towards such a classification. It is possible that a classification might be based on a study of the faculty of attention. For this purpose very large numbers of both normal and abnormal children would require to be tested.

A classification on an etiological basis is of importance. In order to aid in determining the causation my chief inquiry has been into the part played by Syphilis in the production of mental deficiency as ascertained by the Wassermann test. I have made no attempt to classify, but have confined myself to an examination of the physical and mental states of the children, and to enquiring into all the factors which may act as possible agents in the production of this condition.

PHYSICAL CHARACTERISTICS OF FEEBLE-MINDED CHILDREN.

Where the germ plasm is so acted upon as to produce mental defect we should also expect to find developmental defects of other organs of the body. Much stress has been laid on this point and various stigmata of degeneracy have been described.

Warner[1] in the examination of 100,000 school children drew attention to the relationship of physical abnormalities of development, low nutrition, nerve signs, and mental dullness. I have followed the lines laid down by him and have investigated the number, kind, and frequency of occurrence of physical stigmata in the feeble-minded, and the frequency of the occurrence of nerve signs and their relation to the physical and mental condition. For this purpose I have examined 124 feeble-minded children attending special schools, and for purposes of comparison 1256 children of normal mental state attending ordinary schools.

TABLE I shows the number of physical defects occurring alone, two together, three or more together, the number of nerve signs alone and associated with physical defect, and the number of cases with defects in the normal and abnormal child respectively.

T A B L E I.

DEFECTS.	1256 Normal Children. No. of Cases.	1256 Normal Children. Per-centage.	124 Defective Children. No. of Cases.	124 Defective Children. Per-centage.
I. Occurring Singly				
Defects of Cranium alone in	38	3·02	0	0
Defects of Palate " "	40	3·2	2	1·6
Defects of Ear " "	3	·24	2	1·6
Epicanthic folds " "	10	·8	0	0
Other defects " "	21	1·69	4	3·2
I. Binary Defects				
Cranium and Palate "	20	1·59	4	3·2
" and Ears "	4	·3	1	·8
" and Epicanthus "	8	·6	0	
" and other defects "	31	2·4	3	2·4
Palate and Ears	8	·6	0	0
" and Epicanthus "	12	·95	1	·8
" and other defects "	66	5·2	10	8·06
Ears and Epicanthus "	1	·08	0	
" and other defects "	7	·55	4	3·2
I. Three or more defects "	46	3·6	84	67·7
Number of cases with defects	448	35·7	121	97·6
Nerve signs without Physical Defect	36	2·8	4	3·2
Nerve signs with Physical Defect	104	8·2	90	72·6

A glance at this table shows,-

1. That physical stigmata occurring alone are as common and in many cases more common in the normal than in the defective child.

2. That Binary defects occur slightly more frequently in the defective than in the normal child -- defects of the palate being more frequent in the defective.

3. That three or more defects are very much more frequent in the defective child, the difference being so great as to be of some diagnostic value. Dr. Jones[2] found two or more defects in 71·2 per cent of his cases.

4. That the number of cases showing developmental defects of any kind is much greater in the defective than in the normal child.

5. That nerve signs by themselves and unassociated with developmental defects are as frequent in the normal as in the defective child. This corresponds with general observation as nerve signs are to be found in the nervous child whose mental capacity is often supernormal.

6. That where nerve signs are associated with physical abnormalities they are again so much more frequent in the defective child as to be of some diagnostic value.

The defects found are detailed in the next table.

T A B L E II.

DEFECTS.		1256 Normal Children. No. of Cases.	1256 Normal Children. Per-centage.	124 Defective Children. No. of Cases.	124 Defective Children. Per-centage.
Cranium small (under 20 inch.) in		105	8·3	34	27·5
Cranium asymetrical	"	23	1·8	27	21·7
External Ear defects	"	27	2·15	42	35·0
Epicanthus	"	66	5·2	11	8·8
Squint	"	18	1·4	13	10·4
Palate defective	"	217	17·2	71	57·2
Oblique orbits	"	7	0·55	9	7·2
Prognathism	"	0		7	5·6
Supernumerary Ears	"	1	0·08	0	
Miscellaneous Congenital defects	"	5	0·32	5	4·0
Defects of General Balance	"	70	5·5	64	51·6
Defects of expression	"	24	1·9	65	52·4
Corrugation	"	6	0·47	6	4·8
Twitches	"	39	3·1	24	19·3
Deafness of all degrees	"	11	0·87	10	8·06
Rickets (marked signs)	"	34	2·7	4	3·2
Low nutrition	"	25	1·9	13	10·4
Defective circulation	"	25	1·9	17	13·7
Broad base of nose	"	35	2·7	15	12·2
Skin conditions	"	7	0·55	5	4·0
Other physical conditions	"	43	3·4	6	4·8
Speech Defects	"	11	0·87	39	31·4
Inability to fix and follow with eyes	"	0		25	20·0

Cranium.

Malformations in the size and shape of the cranium are frequently present in the feeble-minded.

Dr. Lapage,[3] who has made a careful study of this subject, measured heads by moulding metal bands round them and in this way was able to obtain tracings which show accurately any asymmetry present.

For practical purposes I have found that the method advised by Warner[1] is sufficient, i.e. (1) Inspection, (2) Palpation (passing the open hand over the child's head by which means any irregularities can be detected), and (3) mensuration. I have simply measured by means of the tape measure the circumference, the antero posterior and the transverse diameters. I have taken similar measurements in the normal children in order that comparisons might be made. Ashby and Shuttleworth note defective occipital development as being specially common in the feeble-minded and that defective frontal development may also be noticed. Such defects can be detected by the above measurements, together with inspection and palpation.

The circumference at 1 year should measure 19 inches, at 7 years 20 to 21 inches. The normal circumference of the head of the child of school age should be over 20 inches and under 22 inches. A circumference under 20 inches is, therefore/

therefore, taken as a defect.

Small size of head, although not necessarily a sign of mental defect, is very frequent among the feeble-minded.

Lapage[3] found that 34 per cent of his cases had small heads.

27·5 per cent of my cases had small heads, while 8·3 per cent of normal children had a circumference under 20 inches.

Large heads are not so common. In four of my cases the head measured 22 inches but none were over this.

Cranial bosses may be present usually in the parietal or frontal region. These and the large head are frequently the result of rickets, and, therefore, not of significance in feeble-mindedness. Two of my cases showed a very marked protuberance in the occipital region.

Asymmetry is very frequently present in the cranium of the defective child. It is often associated with a more or less marked degree of asymetry of the face.

Lapage[3] found that 26 per cent of defective children had an asymmetrical skull and noticed that the left side was very frequently smaller than the right.

I found asymmetry of the skull present in 21·7 per cent of/

of feeble-minded and in 1·8 per cent of normal children.

Defects of the frontal region may be noted. These are chiefly rounding or bulging, the presence of bosses, narrow from above down or from side to side, receeding, occasionally the presence of a frontal ridge.

Special types of skull are recognised such as the oxycephalic, scaphocephalic, microcephalic, macrocephalic and hydrocephalic.

Only one of my cases was microcephalic, and one showed a tendency towards, although not absolutely typical of, the oxycephalic type of skull.

We conclude that the chief cranial defects to be met with in the feeble-minded are small size of skull, particularly in the occipital and frontal regions, and lateral asymmetry.

The External Ear.

The external ear may be defective

1. In its parts
   a. lobular defects.
   b. defects of the helix.
2. In its size
   a. abnormally small
   b. abnormally large.

3./

3. In its form

a. Increased depth of fossae.

b. Protrusion of the whole ear.

Or it may show a combination of defects. Defects of the external ear, particularly of the lobule, are common both in normal and in abnormal persons. The importance of the ear as a guide to character has been much exaggerated. Various types of ear have been described such as the Criminal Ear and the Insane Ear.

Lapage[3] found defects of the external ear in 34·2 per cent of his cases.

Dr. Jones[2] found defective ears to be present in 20·4 per cent of 191 cases examined.

Ear defects occurred in 34 per cent of my feeble-minded as against 2·15 per cent in normal children.

Tredgold[4] considers lobular defects to be of most frequent occurrence, and next in frequency the large prominent outstanding ear. I have found the commonest defect to be the large thin outstanding ear with convexity posteriorly and concavity in front, this deformity occurring in 9 out of 42 defects. Defective formation of the lobe was also present in 8 cases, while undue elongation of the lobe was present in 1 case. The other defects found were -- differ-

ence/

ence in size or position of the two ears, exceptionally large ears, exceptionally small ears, ears with deficient or nodular helix, general malformation, and in one case a prominent Darwinian tubercle.

Supernumerary Auricles are rare. They were not present in any of my feeble-minded and only in one of the normal children. These and other developmental defects do not occur more frequently in the feeble-minded and are therefore of no diagnostic importance.

Epicanthal Folds.

These folds of skin coming from the upper eyelid and placed across the inner angle of the palpebral opening are frequently associated with a broad spread out base of the nose. This defect may be asymmetrical, occurring only in one eye, but it is commoner in both. The occurrence is almost as frequent in the normal as in the abnormal.

Lapage[3] found it to be present in 8·79 per cent.

Warner[1] noted its presence in 1·37 per cent of all school children.

Obliquity of the Orbits.

Lapage[3] found this in 3·2 per cent.

In/

In my cases it was present in 7·2 per cent as against 0·55 per cent of normals.

Where present it is often associated with asymmetry of the face, one eye being placed on a lower level than the other.

Strabismus

This is a common defect both in normal and abnormal children and can hardly be considered as a stigma of degeneracy, but the fact that it occurs more frequently in the feeble-minded must be noted.

Lapage[3] found strabismus present in 8·7 per cent of feeble-minded children.

Warner[1] found that 1·37 per cent of normal children showed this defect.

I found it present in 10·4 per cent of the feeble-minded and 1·4 per cent of the normal.

Deformities of the palate.

These are of exceedingly common occurrence, but such defects are by no means limited to defective children and in many cases undue importance has been given to them as stigmata of degeneracy. Palatal defects are very common in Rickets and/

and in these days of artificial feeding and dummy teats may not be developmental but induced defects.

Although a great many different varieties have been described they can be put into two main groups.-

1. The highly arched saddle or keel-shaped palate which is the most common type.
2. V-shaped, which is also as a rule higher than normal and sometimes difficult to distinguish from the former type.

Shuttleworth[5] regards a palatal defect as suspicious of mental deficiency.

Petersen[6] found various defects in the palate in 82 per cent of idiots, imbeciles and feeble-minded; in 72 per cent of epileptics and in 80 per cent of the insane.

Clouston[7] has shown that though deformed palates occur in the normal they are much more frequent in neuropaths and the mentally deficient. He found such defects to be present in 19% of the ordinary population.

33% of the insane.

55% of criminals.

61% of Idiots.

Lapage[3] found defects present in 67·1 per cent of defective school children.

Dr Jones[4] investigating feeble-minded school children in/

in London found deformities of the palate to be the most frequent defect present.

In my cases the palate was defective in 57·2 per cent of the feeble-minded and in 17·2 per cent of the normal children.

Taken by itself deformity of the palate is not diagnostic of mental deficiency, but when it occurs in conjunction with other defects, particularly cranial and ear defects, it may be of some diagnostic value.

Cleft Palate is apparently not significant of mental deficiency. It belongs to the same category of defect as the supernumerary auricle.

Langdon Down[8] finds it in ·5 per cent of defectives.

Ireland[9] found it in 1 per cent of idiots.

Talbot[10] found no case present in examining 1977 feeble-minded children.

In none of my cases was it to be found.

Jaws.

Many defectives have receding jaws and this is characteristic of microcephalics.

Lapage[8] found Prognathism in 3·26 per cent of his cases.

It/

It occurred in 5·6 per cent of my feeble-minded and was not present in any of my normal cases.

Growth and Nutrition.

Many observers state that in height and weight the feeble-minded child compares very unfavourably with the ordinary school child of the same age.

MacDonald[11] concludes that children with abnormalities are inferior in height and weight to normal children.

Lapage[3] in the examination of 181 feeble-minded children found that in height and weight they were below the average for school children and that this disparity increased at the older ages; also that the difference in weight was greater at the older ages than the difference in height.

I have compared the heights and weights of my children with the averages of school children attending schools in the same district, therefore with the same environmental conditions in both cases.

In tabulating them I have used the tables published in the annual report on Medical Inspection of Schools in the Govan Parish for 1911-12, and in this way any difference is seen between the heights and weights of children attending the special schools in the Govan Parish, those attending ordinary schools in the Govan Parish, and the standard given by the Anthropometric Committee.

T A B L E III.

AVERAGE HEIGHT.

Boys -- Average Height in Inches.

Age	5	6	7	8	9	10	11	12	13	14	15.
No. Examined	0	0	0	1	2	6	12	6	1	3	0
Govan Parish Mentally Defective Schools	-	-	-	47·2	46·7	51·2	50·3	50·8	54·0	53·5	-
No. Examined.	615	1158	455	271	527	655	463	371	320	556	0
Govan Parish, all Schools Average Height in Inches	40·1	41·0	43·3	45·8	47·9	49·2	51·1	52·5	54·2	57·4	
Anthropometric Committee's Standard	41·0	44·0	46·0	47·1	49·7	51·3	53·5	55·0	56·9	59·3	

Girls -- Average Height in Inches.

Age	5	6	7	8	9	10	11	12	13	14	15.
No. Examined	0	0	3	1	6	3	1	3	1	1	3
Govan Parish Mentally Defective Schools	-	-	42·2	50·2	44·7	46·6	51·7	52·0	54·0	51·5	53·3
No. Examined	600	1274	547	311	467	602	463	399	366	495	0
Govan Parish, All Schools	39·8	41·1	43·1	45·2	47·6	49·1	50·4	51·0	52·5	56·9	
Anthropometic Committee's Standard	40·8	42·6	44·5	46·6	48·7	51·1	53·1	55·7	57·8	59·8	

One girl of 12 weighed 42 lbs. and measured 39·5".

Considering the small number of cases this was not included in the average above.

T A B L E IV.

AVERAGE WEIGHT.

Boys -- Average Weight in Lbs.

Age	5	6	7	8	9	10	11	12	13	14	15.
No. Examined	0	0	0	1	2	6	12	6	1	3	0
Govan Parish Mentally Defective Schools	-	-	-	48·0	54·2	63·2	61·5	65·0	77·0	63·5	-
No. Examined	615	1158	455	271	527	655	463	371	320	556	0
Govan Parish All Schools.	39·1	40·5	44·3	50·2	54·7	58·5	62·6	67·6	71·7	80·4	-
Anthropometric Committee's Standard.	39·9	44·4	49·7	54·9	60·4	67·5	72·0	76·7	82·6	92·0	

GIRLS -- Average weight in Lbs.

Age.	5	6	7	8	9	10	11	12	13	14	15.
No Examined	0	0	3	1	6	3	1	3	1	1	3.
Govan Parish Mentally Defective Schools.	-	-	47·0	60·0	50·6	56·0	63·5	71·9	75·0	68·0	80·0
No. Examined.	600	1274	547	311	467	602	463	399	366	495	0.
Govan Parish All Schools.	38·0	40·2	42·9	47·2	52·9	56·4	60·9	63·7	73·4	81·9	
Anthropometric Committee's Standard.	39·6	42·4	46·7	52·2	55·5	62·0	68·1	76·4	87·0	96·7	

My numbers are too small to form definite conclusions from the results obtained.

It will be noticed by reference to the tables that the height of the feeble-minded boys corresponds fairly closely with the standard in the earlier ages, but is slightly below the standard from 11 years onwards. The weight of the boys shows a still greater falling off at the ages of 12 and 14 (as only one boy of 13 was examined his height and weight can hardly be taken as an average).

The height of the girls corresponds fairly closely with the average for Govan Parish, while the weights in most cases exceed the average and come near to the Anthropometric Committee's standard.

These results coincide fairly accurately with personal observation, it being noticed that the older boys are frequently well grown but thin, and with muscles wanting in tone, while the girls are frequently thick set and fat.

The actual height and weight do not give an accurate idea of the general nutrition of these children. I have judged the nutrition rather by an examination of the muscular condition, and of the skin and mucous membranes.

I found that 10·4 per cent of my feeble-minded cases were/

were of low nutrition, while the same condition was only found in 1·9 per cent of my normal cases.

Defective circulation is frequently met with as evidenced by habitually blue and cold hands, and tendency to the formation of chilblains. 13·7 per cent of my cases showed these signs and only 1·9 per cent of the normal children.

This is of importance in connection with mental action, as with a deficient supply of blood to the Brain its functioning will be lowered.
Ireland[9] states that idiots and imbeciles have small hearts and therefore a poor blood supply to the extremities and the brain. I do not think that cerebral anaemia can of itself produce mental defect but that it is more likely to produce a delayed development. In such cases when the circulation has been improved the development takes place naturally.

Other Physical Conditions,
Such as organic heart disease, enlarged tonsils, bronchial conditions, occur in much the same proportion in the feeble-minded as in the normal child.

The/

The condition of the skin is an exception. In 4 per cent of feeble-minded cases I have found it to be rough and scaly, almost an ichthyosis, while I have only observed this condition in 0·5 per cent of the normals.

Deafness, too, appears to be more common in the feeble-minded, occurring in varying degrees in 8·06 per cent of my cases as against 0·37 per cent of the normals.

Speech Defects.

Speech defects are so frequently found in the feeble-minded as to have a certain diagnostic value. Indeed it has been suggested by Esquirol[12] to base a classification of the defectives on the condition of speech. This, as has been stated, would not be a correct classification as many normal children may show speech defects, and defective speech is not always present in the feeble-minded.

The faculty of speech is composed of,-

(1) a sensory or afferent pathway -- auditory, visual, muscular.

(2) a central mechanism for the recognition and association of words with things and ideas and their conveyance to the motor speech centre from which

(3)/

(3) an efferent pathway leads to the muscles of respiration, phonation and articulation. Interference with any part of the mechanism will interfere with the production of speech. Defects in the afferent pathway do not commonly cause speech defects. Complete deafness may prevent the development of speech, but where the intelligence is normal the child can be taught to speak.

The central association may be at fault, and this I believe to be very frequently the case. As will be seen later, lack of attention is one of the characteristics of feeble-mindedness, and this lack of attention to the spoken word will produce defective recognition of the object spoken of and also a defective memory for words, just as in the same way lack of attention to the written word will produce defects in its reproduction and in spelling.

Defects are frequently situated in the motor or efferent pathway, which includes both the motor speech centre and the muscles concerned in the production of voice and speech. Complete motor aphasia is said to be rare. In one of my cases absence of speech was apparently due to a defect in the motor speech centre. A bright healthy girl of 8 years who up to the time of her admission to school/

school (2 years previously) had not been known to speak. There was no defect in her auditory apparatus, she answered readily to her name and obeyed simple commands. She was of a very affectionate disposition, but very nervous, showing a tremor of her fingers when held out and with her head inclined slightly to the right side. She was frequently smiling and was interested in all the doings of the other children and of her teacher. She seemed continually on the verge of speech. A few months after her admission her mother reported that when suddenly excited she would say a word or two, but as she never made any attempt to speak in school I was doubtful as to the accuracy of this statement. When, however, I was taking her blood for examination she called out "sore". A few days later she pointed to my stethoscope and said "what's that." There was no defect in articulation but the words were spoken in such a low toneless voice as to be almost inaudible. The chief defects lie, however, in the improper action and want of co-ordination in the muscles concerned in the production of speech. Muscle action is a manifestation of Brain action. Deformities of the palate may lead to a nasal tone but short of actual cleft palate do not lead to/

to articulation defects.

Tredgold[4] and Lepage[3] agree that shortening of the fraenum linguae (tongue-tie) can have very little effect on the movements of the tongue and is therefore negligible as a cause of speech imperfections. I had one girl, however, where the fraenum was so short that protrusion of the tongue was impossible. This did seriously interfere with the movements of the tongue. She was late of beginning to speak, and at eight years had difficulty in pronouncing linguo-dentals, especially the consonant S, for which she substituted Ts.

Thick and clumsy action of the lip and tongue muscles produces the principal defects in speech found in the mentally defective. Stammering is the result of want of co-ordination between the muscles of phonation and the muscles of articulation. It is not specially characteristic of the feeble-minded being only present in two of my cases out of a total of 39, showing some form of speech defect. It is quite as frequently found in children of normal mental state.

Thick, indistinct and slurring speech was very common in my cases. This was largely due to want of attention and muscular control, and improved very greatly under tuition.

The/

The dropping of the terminal consonant, which is a common fault, might also be attributed to want of attention and to carelessness.

The other and chief defect found was a replacing of one consonant by another -- lalling.

Wyllie,[13] Ashby[14] and Lapage[3] have all made extensive investigations into speech defects, and Lapage found the commonest defects to be in the articulation of the linguo-dental and labio-dental consonants. I have found Th to be most frequently mispronounced. This corresponds with Lapage's investigation, who found Th, R, Y, and S to present most difficulty in order of frequency.

Lalling is very often a stage in the development of normal speech and the difficulty here also is presented by the linguo-and labio-dentals. It is due to an imperfectly developed muscular co-ordination. It might, therefore, be supposed that its presence in the older defective child was due to a later and slower and in some cases an imperfect development. An examination into the age at which the defective child first speaks goes to support this. 62 per cent of my cases were over two years before any attempt was made to speak, while 45 per cent were/

were over three years. Lessons in articulation and breathing teach control over the muscles and also fix the attention and thereby many of the defects can be remedied.

We may say then that the majority of speech defects are due to a want of attention and association and to defective control of muscular action, and that by careful and prolonged muscular education the defects can in many instances be remedied, and only in a very few cases is there definite deformity of the speech producing apparatus which is frequently found in the normal child and is not specially characteristic of the feeble-minded child.

A review of the conditions found by an examination of the number and kind of physical abnormalities in feeble-minded and in normal children shows (1) that physical abnormalities by themselves are in no way diagnostic of feeble-mindedness and that two of these defects occur together almost as frequently in the normal as in the abnormal child; (2) that the presence of three or more abnormalities in combination is suggestive of defect, occurring as they do in 67·7 per cent of the feeble-minded as against 3·6 per cent of the normal children; and (3) that deformities and abnormalities of the palate are the defects most frequently found/

found.

The presence or absence of what Warner[1] calls 'Abnormal nerve signs' is of very much greater importance as indicating the mental state of the child. These show themselves in faults of position, movement, action and expression.

All these conditions are dependent on muscular action and as all mental states are expressed by movement they are of great value in diagnosing not only the presence of mental defect, but also to some extent the degree of defect.

The development of defective children is as a rule delayed. They are late of walking, late of talking, late of sitting up without support -- all actions depending on muscular action.

I have tabulated the average ages at which the defective child starts walking or talking. Following Lapage[3] I have arranged the children in groups according as the intelligence was fair, medium or bad. I determined this by means of mental tests, while Lapage had recourse to the teachers report.

The results obtained are very similar although I was dealing with a smaller number of cases.

T A B L E V.

	Average age of Walking.	Average age of Talking.
I. INTELLIGENCE FAIR.		
16 Cases	1.2 years.	1·5 years.
Lapage 61 Cases	1·5 years	1·8 years.
II. INTELLIGENCE MEDIUM.		
29 Cases	1·8 years	2·2 years
Lapage 44 Cases.	1·8 years	2·0 years.
III. INTELLIGENCE BAD.		
31 Cases	2·8 years	3·5 years
Lapage 50 Cases	2·2 years	3·5 years.

It is not easy owing to want of accurate observation on the part of the parents to obtain an accurate history as to when the children sat up without support, but many are reported to be later than the normal children of the family.

Expression of the face.

This has always been recognised as an index of Brain action, expression being produced by the summation of the action of all the small muscles of the face. Any irregularity of nerve action affecting one or more of these muscles will alter the expression of the face. Thus a contraction of the occipito-frontalis muscles will produce a creasing of the forehead, while an over activity of the superior orbicular muscle of the eyelids causes a contraction or drawing together of the inner ends of the eyebrows. This latter is characteristic of intensive thought but where the contraction becomes fixed and habitual a dull puzzled expression is produced which is frequently characteristic of the severer grades of mental defect. The expression may be dull, vacant, heavy, anxious or wanting. In other cases there may be an over mobility of certain muscles causing twitching of various parts of the face.

Lapage[3] observed defects in expression in 58·79 per cent of his cases.

By referring to Table II it will be seen that 65 of my cases showed an abnormal expression, or 52·4 per cent as against 1·9 per cent of normal children; while it will also be seen that corrugation was present in 4·8 per cent of all feeble-minded cases or in 9·2 per cent of those with defective expression.

The General Balance of the Body.

The position in which the child stands, the head balance, the position in which the hands are kept when extended from the shoulders, all give valuable information as to the muscular control of the child. The feeble-minded child very frequently stands with one shoulder higher than the other in a loose and slouching attitude. This position might be due to scoliosis but the cases I have recorded have not shown any fixed curving of the spine and the irregular position is only present when conscious of observation and disappears during play and work.

The head may be held to one or other side or forward with the chin pressed against the chest.

When the hands are held extended in front with the fingers/

fingers spread out it is frequently observed that they are held at a lower level than the shoulders, or that one arm is lower than the other, or that the position is not sustained and the arms gradually fall, or the hands may be in the nervous, fatigued or frightened position (described by Warner[1]) and frequently with a fine tremor of the fingers.

Such alterations of balance may be found in the child of normal intelligence, especially in the nervous child, but they are of much commoner occurrence in the feeble-minded. Lapage[3] gives 10·6 per cent as showing defects of general balance.

I noted defects of balance in 51·6 per cent of cases as against 5·5 per cent in normal children. This very marked difference in results is probably due to the fact that I included all defects of body, head and hand balance.

I found defects of hand balance to be the most common, occurring in 25 per cent in contrast to 5 per cent in the normal children.

Bad position of the head occurred in 20 per cent of defectives and ·63 per cent of normals.

Want of muscular control as evidenced by movements of the eye muscles in fixing and following a bright object held/

held in front of the eyes and moved from side to side and up and down, is of very great importance both in the diagnosis and prognosis of the mentally defective child.

This tests the power of voluntary attention. The power of fixing an object with the eyes even for a short time shows that voluntary attention can be aroused. Many children can fix the object with the eyes, but have apparently no control over the finer movements of the eye muscles, the movements of the object being followed by movements of the whole head. (This is not due to failure to understand what is required of them as in such cases when the head is held steadily by the experimenter the movements of the object are not followed).

Others again showing feeble power of voluntary attention will fix the object for a moment only when the eyes immediately wander off to some other object.

In some cases the child can neither fix nor follow the object with his eyes. The child who is capable of fixing and following an object with his eyes is capable of being educated to a very considerable extent and is the most hopeful case for future improvement. The child who can fix but has difficulty in following the object can be trained to a certain extent. He has the power of voluntary attention/

attention, though in small measure, and where such is present, in however rudimentary a state, it is capable of being utilised and developed by careful care and training.

The child who is incapable, after attempts frequently repeated at varying intervals, of fixing and following an object with his eyes is uneducable. His voluntary attention cannot be aroused, and without the power of voluntary attention education is hopeless.

Defective eye movements were present in 20 per cent of my feeble-minded children.

Disorders and irregularities of movement are frequently present. This may be present as

1. Deficient movement
2. Excessive "
3. Inco-ordinate "

the two latter being frequently combined.

Deficient Movement.

Children showing deficient muscular movement are of the lethargic type. The expression is dull, vacant, or heavy; speech is slow and deliberate, often slurring; movements are slow and sensations diminished; mental capacity often feeble. Such often border on imbecility.

Excessive/

Excessive Movement.

This type is more common among children showing the lesser degree of defect.

There is a motor excitability accompanied by want of control leading to continual restless movements with wandering eyes and attention. Sometimes twitching of muscles or groups of muscles occurs. In many cases speech defects are present. In some of these cases impulsive action is observed, inhibition and power of muscular control being deficient an idea is no sooner formed than acted upon.

Extreme restlessness was present in 25 per cent of my cases, while twitching of varying degrees was noticed in 19·3 per cent.

Inco-ordination of Movement.

This is a marked feature of the feeble-minded child and is specially noticeable at the earlier ages before the child has been to any extent trained. It may be combined with either deficient or excessive movement, more commonly the latter.

In the normal child co-ordinated movements are only produced by practice and training. All my cases showed some difficulty with the finer movements of precision, there being/

being difficulty in lacing boots and buttoning clothes. These finer muscular movements can be trained. Their training and control produce an improvement also in the mental state.

To sum up

1. Developmental defects and abnormal nerve signs are present more frequently in the feeble-minded than in the child of normal intelligence.
2. Although no abnormality or nerve signs by themselves are diagnostic of mental deficiency several in combination are strongly suggestive that such defect exists.
3. A combination of abnormal nerve signs with physical defects is very frequently found in the child of defective intelligence.
4. The presence of abnormal nerve signs is of more significance than the presence of physical defects. No idea of the mental state can be given by the physical defects, while nerve signs indicating want of muscular control, with irregular movements especially of the eyes, are of some diagnostic and prognostic value.

MENTAL CHARACTERISTICS OF FEEBLE-MINDED CHILDREN.

Although much can be learned as to the child's mental state by conversation, by careful and skilled observation of his manner, behaviour, general conduct and natural scholastic ability, especially in cases of marked feeble-mindedness in children the observation must be frequently repeated and much time spent in determining the exact mental condition. This applies especially to cases of the lesser degrees of mental deficiency -- the feeble-minded. These children can rarely be distinguished from the "Dull and Backward" merely by the above methods and as these lesser degrees of defect are the cases which must necessarily be found in our special schools, while the "Dull and Backward" are excluded, it is a matter of importance that the mental peculiarities of the defective child should be known. He must be educated by utilising and cultivating the special powers which he possesses, and therefore a knowledge of these special powers must be obtained.

In determining the mental state of the feeble-minded child and distinguishing him from the "Dull and Backward" I have found the tests elaborated by Binet & Simon to be of great value.

Binet & Simon writing in L'Annee Psychologique in 1905 on/

on "Methodes nouvelles pour faire le diagnostic differentiel des anormaux de l'intelligence" give a series designed to investigate all the important phases of intel lectual capacity. These tests were applied to a large number of normal children between the ages of 3 and 11 years and the insufficiency of the defective estimated by comparison with the results obtained for these children.

In L'Annee Psychologique 1908 the same authors record the results obtained by the application of the tests to a still larger number of normal children. By this means they determined that at certain ages certain powers are developed and that a child can therefore perform certain acts and answer certain questions according to his age.

I have used the tests in all cases where response could be obtained and consider that the feeble-minded child is always more than two years below the intellectual level of the child of normal mental capacity of the same age.

I do not propose to consider the results obtained by the application of the individual tests but to take into consideration the various mental powers and try to determine their condition in the feeble-minded child as shown by personal observation, the Binet Simon, or any other tests available.

ATTENTION./

ATTENTION.

The act of attention is the fixation of consciousness on an object in order to obtain the greatest possible knowledge of the object attended to. It involves interest in the object. It may be voluntary or active and involuntary or passive. It is important to recognise these different forms of attention when considering its presence or absence in the child.

This concentration of the consciousness on an object involves necessarily an inhibition and suppression of interest in other objects. The act of attention also depends on motor control and as we have seen a frequent characteristic of the feebleminded is a lack of motor control. We would therefore expect to find want of attention a characteristic of the feeble-minded child and this actually occurs.

In the dull apathetic type of defective child both forms of attention are dimished or absent.

In the restless excitable child, on the other hand, interest is easily aroused; every new object attracts involuntary attention. But the attention is not sustained, there is no inhibition of other impressions and so the attention flits rapidly from one impression to another.

The want of muscular control interferes gravely with the maintenance of voluntary attention. This is seen in the normal child where the presence of adenoids interferes with the/

the proper respiratory movements and lack of attention results. Hence the very great importance of a training in muscle control.

No special tests for attention are necessary as the response given to every test shows the ability or otherwise to concentrate the consciousness on any object.

PERCEPTION.

The powers of perception depend largely on the powers of attention. Perception is developed slowly and irregularly in the normal child. Perception of objects can be tested by showing the child objects or pictures and asking him to describe them. At three years the child of normal mental capacity will enumerate objects seen, showing a simple perception and recognition of external objects. By seven years the character of the persons and the nature of the things should be described and their connections and relationships noted, this indicating a power of association (apperception).

In testing the defective children I found that the child perceived the objects and enumerated them, but that under nine years of age none were able to describe the pictures, thus showing an inferiority to the normal child in the power of association.

Perception of differences in weight can be tested.

For/

For this purpose I have used four small pill boxes of equal size and shape weighing respectively grs. 45, Drachms 1½, 3 and 4. One is placed in each hand and the child asked which is the heavier. This also involves the muscle sense. The normal child of five years can readily discriminate even the smaller differences of weight. Feeble-minded children of all ages give a very feeble response to this test. In many cases the answers are at random and in some the knowledge that it is necessary to weigh the boxes is wanting, showing not only a deficient perception of difference of weight but also a deficient understanding.

Tactile, painful and thermal sensations although frequently diminished in imbeciles and idiots I have not found to be markedly so in the feeble-minded. These sensations are never absent in the lesser degrees but the response given is often slow and delayed, due more to want of attention and perception than to any defect in the sensory tract itself.

The colour sense is tested both by naming and matching colours. The feeble-minded child is often incapable of associating the colour with its name, yet quite capable of matching colours. I do not think defective colour sense/

sense is to be found any more frequently in the feeble-minded than in the child of normal intelligence.

MEMORY.

Closely associated with the power of association and perception is that of memory. The description of familiar objects from memory brings out this relationship. It also shows the child's power of expression, his judgement, the conception he has formed of an object and the point of view he considers to be the most important. Binet describes three types of answers to this test.

1. Silence or mere repetition of the words. This is found in the apathetic type and in the more marked degrees of the feeble-minded.
2. Definitions according to use (normal child of seven years).
3. Definitions superior to use (normal child of nine years).

Feeble-minded children merely repeat or define by use, the mental images being simple with few associations and such as occur generally relating to the immediate necessities of life.

These points are illustrated by a consideration of some of the answers I have received to the questions:-

What is a fork -- a chair -- a horse -- a mother.

Boy aged 13.

A/

Boy aged 13.

A Fork -- Its to stick into beef.

A Horse -- Draws a cart.

A Chair -- To sit on.

A Mother -- She's a wife.

Boy aged 11.

A Fork -- For taking your food with.

A Horse -- is an animal.

A Table -- for taking your food off.

A Mother -- your own mother.

Girl aged 7.

A Fork -- for beef.

A Chair -- to sit on

A Horse -- to ride on.

A Mother -- to make the dinner.

Girl aged 9.

A Fork -- horns on it to take tatties.

A Horse -- the horse runs and the man on the horse.

A Chair -- legs on a chair.

A Mother -- a head, nose, mouth, neck, ears.

Boy aged 10.

A Fork -- is for taking your meat with.

A Horse -- is an animal.

A Chair -- is for sitting on.

A Mother -- is a lady.

Memory is also closely associated with attention.

Memory/

Memory is the reproduction of past impressions and if the impressions have been feeble owing to lack of attention the powers of reproduction will also be feeble, and as defective attention is characteristic of the feeble-minded so also is defective power of memory.

Memory consists not of one but of several memories in combination -- the visual -- the auditory -- the motor. The degree in which these are combined varies. In some the visual memory predominates, in others the auditory or motor. Perhaps the most common type is the auditory-motor.

The auditory memory may be tested by reading a short passage to the child who is then asked to recall as much of it as possible. This test depends largely on the attention given to the passage read and as the feeble-minded are notably deficient in voluntary attention the responses given are poor and differ very strikingly from those given by the child of normal intelligence. By eight years the normal child can remember two facts in the passage read. I have never found a defective child under nine remember two facts the usual age being 11 or 12 years.

The Immediate memory for unrelated auditory stimuli is tested by repeating numbers. This involves an act of voluntary attention as the isolated figures convey no meaning and/

and have no association.

The immediate memory for intimately associated auditory stimuli is tested by repeating phrases. The normal child of 6 years can repeat a phrase of 16 syllables.

To both these tests I found the defective child gave a very poor response, and conclude that the auditory memory, depending as it does so largely on attention, is poorly developed in the feeble-minded child.

Binet, comparing 12 defective children of nine years with 12 normals of the same age, noted that the memories of the defective children were very poor in this respect. He found one or two exceptions where the children could repeat sentences. On closer examination he considered that their ability to repeat was an'echo' and observed that if an interval was allowed between the hearing of the sentences and its repetition these children also failed in the test.

Visual memory may be tested by exposing to the child's view for a given time a given number of objects or pictures, and determining the number of these the child remembers after they have been withdrawn from his field of vision. I have obtained much better results from this test than from the preceding, mainly, I believe, because the pictures and objects rouse the interest and fix the attention. This fact is/

is of practical importance in the education of the defective child.

Feeble-minded children may show a peculiar retentiveness for certain isolated facts which have impressed them. This was observed in one of my cases-a boy of 11 years who when started would repeat fluently without any intermission and without any understanding, sentence after sentence of disconnected words and phrases dealing with all the social and political problems of the day.

A case showing a remarkable auditory memory was that of W.B. aged 11, a case of word blindness. I was asked to see this boy as his teacher found it impossible to teach him to read or even to recognise his letters. To prove this his book was opened at a page which had been gone over in class some time previously and he was started by having the first word read to him. Immediately, and to the astonishment of his teacher, he read two pages fluently and almost correctly. On careful examination, however, it was found that the boy did not recognise a single letter on the printed page and was repeating the whole story from memory. He could also repeat rhymes which he had learned when he had first come to school, four years previously.

The/

The question arises whether the defects in memory are due to a difficulty to recall the past or whether it is due to a reduced strength of impression. The defective child can recall vivid impressions and striking events almost if not quite as well as the normal child. Therefore his power of recall is not wanting. But with an inability to fix the attention, with continual new impressions passing through his brain, few are vivid or intense enough to be capable of recall.

IMITATION and IMAGINATION.

This involves attention and memory. By attending to the idea of a movement the movement can be reproduced.

As a class feeble-minded children can imitate simple movements and actions. Where the movements are complicated or continued for some time greater difficulty is experienced, as in the latter case the interest flags and the child tires of the exercise.

The defective child is much better at imitating than at initiating. He may be able to copy faithfully a drawing or a model (especially if his interest is aroused) but he can hardly ever originate. This can be well seen in the class where the children will not attempt to do anything till told by their teacher what to do or actually started on a piece of work. In some cases they experience as much difficulty/

ficulty in stopping as in beginning an occupation, which they may continue almost automatically till stopped.

The defective child may be gifted with imagination in the sense of romancing and day-dreaming. This is illustrated by the inventions and imaginings frequently given to take the place of the forgotten fact in the passage read when testing the auditory memory.

The passage read is:-

"Last night in Glasgow a fire destroyed three buildings in the centre of the town. Seventeen families are without shelter. In saving a baby a boy got his hands badly burnt".

Some of the answers given were:-

Boy aged 10.

A Fire. A man saving little children died with the reek going into his mouth.

Boy aged 10.

A boy got his hands burnt, one got burnt and one got saved.

Boy aged 12.

A boy got burnt and a wife got burnt. Glasgow.

But of ~~the~~ imagination in the sense of the formation of ideas, the feeble-minded child is decidedly lacking. If the defective children are watched when at play it will be noticed that they run about in an aimless manner and do not/

not start games in the same way as the normal children do.

The power of imagination may be tested by the ability with which a child can frame a sentence. The child is given three words such as "Girl: Ball River" and asked to form a sentence including these words. It is found that the normal child of 10 years can easily accomplish this task. Of the defectives few attempt to frame a sentence and those who do require considerable help before the 3 words are included in their sentences.

RELATIONSHIP.

This may be estimated by the ability to compare objects or to trace resemblance between objects.

Both acts involve an act of attention. This act of attention must include the two things compared. The consciousness has to be concentrated on each in turn and there must also be an effort to keep both objects within the field of consciousness at the same time. Now we know that lack of attention for one object is a characteristic of the feeble-minded child, and that, therefore, where two objects are involved as in comparing and tracing resemblances there must be still greater difficulty. By testing these capacities we find this to be the case, and it is noticeable that where the objects are not present in the field of vision the difficulty/

difficulty becomes greater as then an effort of memory is also required. The objects compared, as suggested by Binet, are wood and glass, fly and butterfly, paper and cardboard. At 8 years the normal child readily compares objects. I have found no defective child under 9 years good at comparison. The following are examples of some of the answers:-

Boy aged 10.

A butterfly is bigger than a fly.

Glass is bigger than wood.

Cardboard is thicker than paper.

Boy aged 9.

Never seen a butterfly.

Glass is white, wood is a funny colour. round the window.

Cardboard and paper are the same.

Boy aged 9.

Wood is brown and glass white.

Paper andcardboard two of them white.

Butterfly has white wings.

Boy aged 9.

A fly's got wings, black ones. Butterfly white.

Glass is on window, wood's on table.

Paper's the best.

Girl aged 11.

Never seen a Butterfly.

Glass cuts your feet.

Paper and cardboard -- no answer.

Girl aged 12.

A fly's wee and a butterfly sits down on trees.

Wood is heavier than glass.

Pasteboard's thicker than paper.

Girl aged 9.

Wood's the same and glass is different.

The last example is characteristic of the majority of responses given by the feeble-minded child. It is noticed that avvery large number of the answers show that colour is evidently the character which impresses the defective child and which is remembered best by him. All children are attracted by bright and vivid things, but the attraction persists longer and is more intense in the feeble-minded than in the normal child.

Tracing resemblance between two objects is even more difficult than comparing them, and is rarely attempted by the feeble-minded child.

JUDGEMENT, REASONING and IDEATION.

We judge when we form an opinion on something. When any object is judged there must be close observation of that object. There must also have been a careful observation in the past, the facts revealed by these observations must be connected and compared and the results obtained are reproduced as judgement on the present object. This means an effort of/

of voluntary attention, a power slowly developed in the normal child and still more slowly and feebly in the defective child. As we know attention and association are poor in the defective and therefore the power of forming a clear accurate and unprejudiced judgement is wanting.

Reasoning involves higher mental activities than any yet considered. Reasoning presupposes judgement. From the judgement passed on some object we proceed to pass judgement on some other object. This also involves an act of voluntary attention and the power of comparing. From these considerations it is evident that the powers of judgement and reasoning must be poorly developed in the feeble-minded child.

A consideration of some of the answers given to Binet's series of "Problem questions" by my feeble-minded cases illustrates this. Most feeble-minded children can answer the simpler questions which deal with conditions of every-day occurrence, such as:-

What ought you to do when you feel sleepy?

What ought you to do when you feel cold?

The response given to the more abstract questions is not so good and indeed many of the children fail to understand them at all.

The replies bring out in a very striking manner the difference between the mental condition of the normal and that/

that of the abnormal child.

For example the simple question "What ought you to do when you feel cold?" is answered by:-

Warm yourself at the fire.

Put on a coat.

Run about.

While the question "What ought you to do if the boy you are playing with hits you without meaning it?" is answered by:-

Kick the ball.

Hit him back.

Go and tell my mother.

Begin to cry.

The question "What happens to a person who is lazy and does'nt want to work?" is answered by:-

They get tired.

Dont go to work.

Father makes him work.

They should get made to work.

They get into a row.

They go and stand in the corner.

JUDGEMENT OF LENGTH can be tested by means of a card on which are drawn two lines, one 5 cm. and the other 6 cm. long placed 3 cm. apart. The card is shown to the child with the longer line placed to the right, to the left, above and below the shorter and in each case the child

is/

is asked to point to the longest. Suggestibility can be here introduced by immediately afterwards showing two lines of exactly the same length. I compared the results obtained by this test with 20 children in an ordinary school and 20 in a defective school. Of the 20 normal children 18 pointed promptly to the longer line. Only one, a boy, aged 14 years, said that the lines of equal length were the same. The other 19 all pointed to one or other line as being the longer, but in each case after considerable hesitation. It seems probable that they considered the lines to be the same, but felt they were expected to find a difference.

Of the 20 children in the defective School, 13 answered correctly as to the longest line, while every child pointed to one or other of the lines of equal length, and that without any hesitation.

It seems evident from this that the abnormal child is almost as good at judging differences in length as the normal child, but that he is more suggestible.

IDEATION. -- In the formation of ideas, as in the powers of judgement and reasoning the feeble-minded child is notably deficient. They can form general ideas, ideas or images relating to sense-experience and the knowledge of material things.

The ideas formed by the young child as well as by the/

the defective are images of things as a whole. As we have seen in considering memory, the definition of known objects reveals the child's idea of the object. And we saw that the defective child may reach, but never gets beyond, the 7 years standard, i.e. he defines by use. And it will be noticed by referring to some of the examples given that the ideas generally refer to the child's sense perception, especially to the taking of food.

It will be noticed that where a more abstract idea might be expressed as in the definition of 'a Mother' greater difficulty is encountered.

The word Mother calls up an image of the child's own mother as evidenced by the replies--"She's a big stout woman" : "Your own Mother". The general abstract qualities which go to make up the idea of a mother are wanting. In many cases the fact that the previous questions have set the child's mind working along the line of the satisfaction of his sensations produces the reply that "a Mother is to make the dinner".

In some cases a rudimentary reasoning power may be detected as was the case with a boy who said "a horse is made of corn". Yet this same boy when asked what he would do if he missed a train replied "wave my bonnet to the folks in the train".

The powers of ideation, judgement and reasoning (as well/

well as of perception) can be well tested by showing the child pictures and asking him to tell all about them. The normal child of 7 years describes the character of the persons and the nature of the things and notes their connections, while an older normal child can give some interpretation of the scene. As we have seen the older feeble-minded child may describe the picture but I have never found a defective child under 9 years able to do so. Up to 9 years they merely enumerate some of the objects seen in the pictures.

TEMPERAMENT.

As a rule feeble-minded children belong either to the phlegmatic or to the sanguine temperament.

The phlegmatic type are dull and irresponsive, take little interest in their surroundings and are lacking in initiative. Their sensory perceptions are somewhat dimished. The lower degrees may be unaware of cold or hunger.

Those of the sanguine temperament are impulsive, excitable, restless, emotional and easily moved to laughter or tears, but the impressions are quickly removed. They are frequently subject to uncontrollable passion fits which pass off quickly and may leave very little impression behind.

The feeble-minded child is generally affectionate, timid/

timid, dependent and wanting in will power. The simple emotions such as fear, pain, pleasure, anger, surprise are easily stirred but as easily pass away. While the more complex emotions such as indignation, shame, jealousy, contempt and curiosity are often wanting. Many are fond of animals and of children smaller than themselves. Cruelty is said to be a characteristic of some. Except in the case of the morally defective I consider that conscious cruelty rarely occurs as a characteristic of the feeble-minded child. Where cruelty exists it is due to a want of realisation of the suffering produced rather than a conscious pleasure in the spectacle of suffering.

This nature, affectionate, dependent on others, wanting in will power and the complex emotions such as shame indignation and contempt, is a factor of importance in considering the future care and control of the feeble-minded. The amount of education of which these children are capable gives them the ability to follow certain occupations and to do certain things, such as house-cleaning, sewing, tailoring, cobbling, when carefully guided, watched and controlled. At the same time it gives both them and their guardians a sense of independence and freedom, and if not carefully guided and directed these children will easily be led away and fall into evil habits.

From/

From my investigation into the home conditions of these children I am convinced that in the majority of cases the prolonged after-care which is so necessary for these children after leaving school cannot be obtained.

We have seen that all the different faculties are poorly developed in the feeble-minded child. We have seen, too, that all the faculties -- perception, association, memory, judgement, reasoning and ideation are dependent on the power of attention. The defect, therefore, in the lesser degrees of mental deficiency, comes to be one of defective power of attention.

Darwin in the "Descent of Man" tells of how a man who trained monkeys to act in plays offered to pay double price if he could be allowed to keep three or four of them for a few days to select one. When asked how he could know in that time whether a monkey would make a good actor or not explained that it all depended on their power of attention. If when he was talking and trying to explain anything to a monkey its attention was easily distracted the case was hopeless. On the other hand a monkey which carefully attended to him could always be trained.

The training of the child, too, depends on the power of attention. Sully[15] says "Attention is the supreme manifestation of psychical and cerebral energy, and according as it is/

is well supplied or deficient the whole of a child's mental progress will be rapid or slow".

Now the act of voluntary attention requires a power of muscular control. The act of attention is accompanied by a general immobility of the body. Breathing is momentarily arrested and the voluntary muscles are held in a state of tension or contraction. The more intense the concentration of thought the more complete the immobilisation. The child, then, who has little muscular control is not capable of any sustained act of attention.

The training and education of the feeble-minded child must have as a basis a careful training of the power of attention by means of the development of muscular control.

E T I O L O G Y.

The study of any morbid condition is not complete without a consideration of the causes acting in its production. In considering the various causes which may act in the production of feeble-mindedness I have endeavoured to ascertain as completely as possible the family and personal history of each child. I have also by visiting the homes of the children ascertained the environmental conditions and at the same time seen other members of the family. There is great difficulty in obtaining an accurate family history. Few care to admit (particularly to school authorities) the occurrence of Insanity, Alcoholism, Syphilis, or Consumption in their family, and it is often only after repeated and careful inquiry that such facts can be elicited. The same difficulty, though in a lesser degree, presents itself in obtaining the early personal and developmental history of the child. I have been able to obtain a history, more or less complete, in 120 of my cases.

Having obtained these histories I then realised that the frequency of the occurrence of certain factors in the history or environment of the Feeble-minded was of little significance without a knowledge of the frequency of the occurrence of the same factors in the history of children of/

normal mental condition. I therefore made an investigation into the family and early history and environmental conditions of 100 children of normal intelligence attending schools in the same district (Govan Parish). In these cases I did not visit the homes personally but in each case saw several members of the family (in school). The homes were visited by a capable assistant whose observations were made under my directions. As many of the families were personally known to her the difficulties in obtaining an accurate family history (which are even greater than in the case of the defective) were reduced to a minimum.

I have based the analysis of the results obtained on the classification of the causes given by Tredgold (Amentia 1908)

A. Intrinsic -- Heredity.

1. Diseases of the nervous system.
2. Alcoholism.
3. Tuberculosis.
4. Syphilis.
5. Consanguinity.
6. Age of Parents.

B. Extrinsic/

B. Extrinsic -- Environment.

a. Before Birth.
   1. Abnormal conditions of the mother during pregnancy.
   2. Injuries to the Foetus.

b. During Birth.
   1. Abnormalities of Labour.
   2. Primogeniture.
   3. Premature birth.

c. After Birth.
   1. Traumatic.
   2. Toxic.
   3. Epileptic and Infantile Convulsions.
   4. Malnutrition.

Tabulating these various conditions and comparing the figures obtained with those obtained for the normal child brings out the following:--

	120 Feeble-Minded.		100 Normal.	
	Frequency.	Combined with other factors.	Frequency.	Combined with other factors.
Neuropathic Family History occurred in	60.	43.	24.	6.
Alcoholic Family History occurred in	12.	7.	6.	2.
Tubercular Family History occurred in	20.	5.	20.	6.
Consanguinity occurred in	1.	-	-	-
History of illhealth, accident or fright during pregnancy	28.	17.		
Abnormal Labour occurred in	30.	21.		
Primogeniture occurred in	19.			
Premature Birth occurred in	8.			
Trauma after Birth occurred in	20.	14.		
Infectious Fever given as cause in	1.			
Infantile Convulsions occurred in	25.	18.	5	(in other members of family).
Bad Home Conditions were found in	16 (13·3%)		12.	
History of Miscarriages occurred in (more than one)	14		12.	

A. Intrinsic Causes.

1. Disease of the Nervous System.

Tredgold (Amentia) found that over 80 per cent of persons suffering from the severer forms of Amentia had a neuropathic heredity. Of these in 64 per cent the heredity was in the form of insanity or epilepsy.
Beach and Shuttleworth[5] found in 42 per cent a distinct neuropathic heredity.
Lapage[3] 27·9 per cent.
Potts[16] in 28·4 per cent as compared with 10 per cent in families of normal children.

All these writers agree that hereditary mental weakness is the most frequent and potent factor in the production of mental weakness.

I found 50 per cent of my cases to have an antecedent nervous family history and that in 14·2 per cent it was the only cause to be discovered. While in the normal children a nervous family history was obtained in 24 per cent and in 18 per cent no other morbid condition could be discovered. These include all forms of nervous disease and of simple neurosis. On analysing these figures we find that in the case of the feeble-minded a history of Epilepsy occurred alone/

alone in 3 cases and in combination with Tuberculosis in 1; While Insanity was the only factor discovered in 8 cases, but was combined with a Tubercular family history in 1 case and an alcoholic in 2 cases.

A family history of feeble-mindedness occurred alone in 14 cases, combined with Tuberculosis in 2 cases and with Alcoholism in 2 cases.

In the case of the normal child a history of epilepsy was obtained in 2 cases, of insanity in 1, and in no case was feeble-mindedness reported.

As stated before absolute reliability cannot be placed on statistics of this kind, but they are sufficient to show that a neuropathic heredity occurs much more frequently in the defective than in the normal child, and that it must be considered as a potent factor in the production of the feeble-minded.

2. Alcoholism.

It is difficult to estimate the importance of Alcoholism as a factor in the production of the feeble-minded. Tredgold[4] considers that it "is rarely the immediate and sole cause of amentia, although where other factors exist -- particularly/

particularly neuropathic heredity -- it is a most important contributory agent." He found a pronounced history of alcoholism in 46·5 per cent.

Beach and Shuttleworth[5] found a history of alcoholism in 16·38 per cent.

Lapage[3] found a marked history in 7·7 per cent and in 1·9 per cent no other factor was to be noted.

Potts[16] noted 41·6 per cent in contrast to 22 per cent among the normals.

I obtained a definite alcoholic family history in 10 per cent, and in 2·5 per cent it was the only factor noted, while in 7·5 per cent there was also a neuropathic history. Among the normal children an alcoholic history was found in 6 per cent, and in 4 per cent no other morbid condition was noted.

The difficulties in obtaining accurate histories of this condition are very great. In the localities from which my figures were obtained the habit of continually indulging in alcohol is of such common occurrence that probably only cases where there was an occasional severe overindulgence were considered worthy of mention. It appears likely, however, that the continual soaking in alcohol/

alcohol will have a much more deteriorating effect on the germ plasm than an occasional though severe overindulgence.

Alcohol may have a direct effect on the growing embryo, in which case its action is extrinsic or environmental, not hereditary. It is quite evident that alcoholism in the mother may have debilitating effects on the offspring.

Some authorities hold that alcoholism during conception may be the direct cause of imbecility or idiocy, and a case quoted by Andriezen in the Journal of Mental Science, Jan. 1905, is brought forward in support of this theory. The case is that of a man of 22 suffering from dementia praecox, whose father had been in a state of "alcoholic intoxication and exaltation" at the time of his conception. The father was father of several other children who "born of his sober moments, were not afflicted mentally or bodily like the patient referred to". Statistics as to this are almost impossible to obtain. I thought some light might be thrown on the question by a consideration of the month of birth, since over-indulgence in alcohol is specially prevalent at the New Year and July holiday in Glasgow. On investigation I did not find that the birth of my cases had occurred more commonly in April and October than in other months of the year/

year.

Dr. Potts (British Journal of Inebriety, 1909) refers to the investigation of Dr. Macnicholl in New York. He traced the family histories of 3711 ordinary children through three generations with great detail as to the taking of alcohol. It was found that of those free from hereditary alcoholic taint, 96 per cent were proficient, 4 per cent were dullards, and 18 per cent suffered from some neurosis or organic disease. On the other hand, of those with hereditary alcoholic taint, 23 per cent were proficient, 77 per cent were dullards, and 76 per cent suffered from some neurosis or organic disease.

In the presence of other factors alcoholism will be an unfavourable element, but I am inclined to think that alcoholism is more a symptom of defective mental development than a direct cause, and the fact that a nervous history was also present in 7 of the 12 cases would help to bear this out.

3. Tuberculosis.

Tredgold[4] found a family history of phthisis in 34 per cent.

Beach and Shuttleworth[5] in 20 per cent.

Lapage[3] in 11·2 per cent.

Potts/

Potts[16] found 43·2 per cent had a tubercular family history as compared with 17 per cent in normal children.

I obtained a definite and marked Phthisical family history in 16·5 per cent of cases as compared with 20 per cent in normal cases.

This gives a higher percentage for normal than defective cases, and considering the widespread prevalence of Tuberculosis we are hardly justified in considering it to be a direct cause of mental deficiency. Where, however, other factors, particularly a neuropathic heredity, exist it may act as a potent contributory agent.

4. Syphilis -- Discussed later.

5. Consanguinity.

Beach and Shuttleworth[5] found consanguinity in 4.2 per cent and Tredgold[4] in 5 per cent.

In only one of my cases were the parents related. An an etiological factor in mental deficiency this can only be of importance in so far as it intensifies any existing hereditary taint.

6. Age of Parents.

Langdon Down[8] found that in 23 per cent of idiots there/

there was more than 10 years difference in age of the parents.

Tredgold[4] found it in 4 per cent.

It also occurred in 4 per cent of my cases.

It is hardly likely that this is a causative factor. It is more likely that age itself would have an influence than difference in age.

In 23 cases the mother was over 30 years.

In two cases the mother was 18 years, one of these being a microcephalic.

B. Extrinsic Causes.

a. Acting Before Birth.

1. Maternal illhealth, accident or shock.

Lapage[3] found evidence in 7·7 per cent and in 2·7 per cent no other factor was quoted.

23·3 per cent of my cases gave a history of some abnormal condition of the mother during pregnancy, but there is a tendency on the part of the parents, in their anxiety to find an explanation for the condition of the child without revealing any 'family disease' to lay too much stress on such conditions. Illhealth, shocks and accidents occur frequently/

frequently during pregnancies where the child is mentally normal.

Tredgold[4] considers that a general prostration or malnutrition of the mother is more important than actual disease.

Alcoholism, Tuberculosis and Syphilis acting on the growing embryo must have a deleterious effect on the normal development of the child.

It is stated that other poisons such as lead may act in a similar manner.

Illegitimacy is stated as a cause, but this can only act indirectly, in some cases the parents themselves are alcoholics or feeble-minded. Two of my cases were illegitimate, in one the mother was a drunkard and had several other children, in the other no history was obtained.

b. Acting During Birth.

1. Abnormal Labour.

This is frequently given as a cause by the parents.

Shuttleworth and Beach[5] consider protracted labour without instrumental delivery a much more potent factor in the production of both nervous and mental defect than instrumental delivery alone. They found a history of protracted pressure in 14·24 per cent of their cases, and of instrumental/

instrumental delivery in only 3·3 per cent.

Tredgold[4], among the severer grades of defect, obtained a history of protracted labour with asphyxia in 14 per cent, instrumental delivery in 2 per cent, and precipitate labour in two per cent. He considers that unless associated with an inherited neurotic taint abnormalities of labour are not of importance in producing the lesser degrees of mental defect, that where mental defect is produced it is owing to a gross brain lesion, and that it can probably not be given as a cause in more than 1 or 2 per cent of all ments.

Lapage[3] found abnormalities of labour accounted for 4·3 per cent of his cases and that in 3.1 per cent no other factor was present.

I found a history of abnormal labour in 25 per cent of my cases and could discover no other determining factor in 7·5 per cent. In 5·8 per cent there was prolonged labour without instruments, 15·8 per cent instrumental delivery, and 3·3 per cent precipitate labour. Two were cases of Spastic Diplegia (Little's Disease), one of Infantile Hemiplegia, 4 had convulsions in infancy and one was epileptic In the disease described by Little he attributes the condition to meningeal haemorrhage occurring as the result of protracted/

protracted and instrumental delivery (although by some authorities ill health during pregnancy is considered to be the main cause). Yet these cases are not by any means always associated with mental defect. The most severe paralysis of this type is quite compatible with an average or even exceptionally bright intelligence. It has been suggested that this haemorrhage has a syphilitic basis. In the only case of Little's disease associated with mental defect in which I examined the blood serum the Wassermann reaction was positive.

Abnormalities of labour were present in a larger proportion of my cases than in those of other writers. This is probably due to the greater prevalence of rickets in Glasgow. If prolonged pressure and instrumental delivery were causative of mental deficiency we would expect to find an exceptionally large number of the feeble-minded in a district where, like Glasgow, rickets was exceptionally prevalent. In referring to the incidence of amentia in different areas of the United Kingdom, according to the investigations of the Royal Commission 1904, we find the reverse to be actually the case.

Thus/

Thus,-

	Idiots.	Imbeciles.	Feeble-minded Adults.	Feeble-minded Children.	Total per 1000 of Pop.
Glasgow.	0·07.	0·23	0·32	1·00	1·68
Birmingham.	0·09	0·27	1·70	1·60	3·76
Belfast	0·13	0·63	0·70	0·97	2·45.
Stoke-on Trent.	0·21	0·45	2·10	1·10	3·96
Somersetshire.	0·18	1·00	2·10	1·10	4·54

Histories of abnormal labour were given in 10 of the cases in which I examined the blood. 6 of these gave a positive, 3 a negative and 1 a doubtful Wassermann reaction.

I do not consider that precipitate labour can have much influence in the production of mental defect except in so far as it increases the liability to injury of the child. In one of my cases a distinct history of injury was obtained, the child having fallen on the head and a haematoma produced. This case had also a Tubercular family history and a history of convulsions in the mother's family, and gave a positive Wassermann reaction.

I therefore conclude that abnormalities of labour can not/

not of themselves produce mental defect but that where other conditions are present they may act as potent contributory factors.

2. Primogeniture.

I do not consider that this can have much influence in the production of mental deficiency. It occurred in 15·8 per cent of my cases. The children are much more frequently the last born.

3. Premature Birth.

Shuttleworth & Beach[5] noted this as a factor in 3·52 of cases.

Premature birth occurred in 6·6 per cent of my cases.

A child born prematurely has much more liability to physical weakness but apart from other factors this cannot produce mental deficiency.

Prematurity suggests the possibility of a syphilitic origin. 5 of the cases in which I used the Wassermann test had been born prematurely. Of these 2 gave a positive, 2 a negative, and 1 a doubtful reaction.

C. Causes Acting After Birth.

1. Trauma./

1. Trauma.

Shuttleworth & Beach[5] state that of the patients admitted to the Royal Albert Asylum 8·25 per cent had a history of injury after birth.

Lapage[3] obtained a history of trauma in 7·3 per cent and in 3·5 per cent it was the sole factor noted.

16·6 per cent of my cases had a history of trauma and in 5 per cent it was the sole cause given. 4 of those had their serum examined, 3 gave a positive result, one a negative.

A slight injury to the child after birth is hardly likely by itself to produce the lesser degrees of mental defect. Where, however, the injury is so severe as to produce unconsciousness, in some cases followed by paralysis, then it is possible that permanent injury may be done to the brain tissue and mental defect result. In the case where the history of a fall followed by fits is given it is much more likely that the fall has occurred at the first epileptic fit than that the fits have been produced by the fall.

2. Toxic Conditions.

Severe/

Severe febrile illnesses are given as causes in 10 per cent of cases by Shuttleworth & Beach.[5]
Lapage[3] found it in 2·8 per cent.
In none of my cases was the defect found to date from a severe febrile illness, though one case followed meningitis. It may be noted that this case gave a negative Wassermann reaction but that as no member of her family was tested the possibility of a syphilitic infection cannot be excluded.

3. Epileptic and Infantile Convulsions.

These are frequently considered to be the cause of mental deficiency.
Shuttleworth & Beach[5] found that 32·58 per cent of admissions to the Royal Albert Asylum had had convulsions in infancy.
Lapage[3] found a history of infantile convulsions in 16·6 per cent and in 4·6 per cent it was the sole factor.

Savage[31] considers inherited nervous instability to be the most important element in the production of convulsions in infancy.

20·8 per cent of my cases had had convulsions, some epileptic in character, and in 5·8 per cent it was the only condition found. 11 of these had had their serum tested,

6/

6 gave a positive, 4 a negative and 1 a doubtful Wassermann reaction.

None of the 100 normal children had convulsions, though it was reported to have occurred in 5 other members of their families.

I think the convulsions must be taken as evidence of a nervous instability rather than as a causative factor in its production.

4. Malnutrition.

Under this must be included all environmental conditions -- food insufficient in quantity and quality, want of fresh air, want of sleep, dirty and insanitary conditions. These all contribute to the poor bodily development of the child and must affect the developing nerve tissue. But bad environment acts rather in producing the dull and backward child, the child of slow mental development, than the child with actual mental defect.

I have made a careful investigation into the home conditions both of the children of normal and those of defective mental state in the same district. I have considered as showing bad environment cases where the children themselves showed evidence of malnutrition or who were dirty and verminous/

verminous, where the houses were damp, dirty and unventilated, and where the food was poor in quantity or quality. Unfortunately I have not been able to obtain reliable statistics as to the hours of sleep of the children.

It will be seen from the table that bad home conditions were found practically as often in the homes of the normal as of the defective child. But it is found on close inquiry that in the case of the defective child where the home conditions were bad a neuropathic history was obtained in 6 cases, a tubercular in 6 cases, and an alcoholic in 1 case, and no other condition was present in only 3 cases; while in only 3 cases was any morbid condition noted in the normal child.

We must conclude then that bad environmental conditions have not a direct effect in producing mental deficiency, but that in the presence of some other morbid state they may act as exciting cause.

By lowering the whole vitality of the body and rendering it liable to disease bad environment may ultimately have an effect in the production of mental deficiency in future generations.

I have inquired into the number of children born in 100 families/

families where mental defect occurred and in 100 normal families in a poor industrial district, the number of children dead, and the number of stillbirths and miscarriages. The results found are tabulated.

	100 Defective Children	100 Normal Children.
Number Born.	656.	652.
Born Dead	30.	27.
Number died in childhood.	169.	161.
Miscarriages.	45.	42.

This shows (1) that practically an equal number of children are born in the normal as in the defective families; (2) that the number of stillbirths and miscarriages are not appreciably greater in the defective than in the normal families; (3) that the mortality rate is also practically the same in the two groups of cases.

A study of the etiological factors in the production of the feeble-minded, by the means at present at our disposal, would lead to the conclusion that morbid conditions are more frequently present in the family and personal history of the defective than of the normal child, but that no one factor is of itself sufficient to produce mental defect/

defect. A neuropathic heredity appears to be the factor of prime importance. Where it is present any of the other factors may be strong contributory agents.

But a consideration of the results obtained by the Wassermann reaction on the part played by syphilis in the production of mental deficiency will bring out facts of importance and lead to some modification of these views.

SYPHILIS AS A CAUSE OF MENTAL DEFICIENCY AS SHOWN BY THE RESULTS OBTAINED BY THE WASSERMANN TEST.

That syphilis is a factor in the production of mental deficiency has been to some extent recognised for long, but very great differences in opinion have been expressed in regard to its importance. This arose from the fact that with no precise method of estimating its presence, only cases showing definite signs of congenital syphilis, or with a parental history of infection could be included. The statistics as to parental infection are most unreliable, as very few will admit the disease, while the number of those showing signs of congenital syphilis has been noticeably small.

Critchett[17] examined the patients in Redhill Asylum without finding a single case whose teeth were characteristic.

Langdon Down[18] found 2 per cent of idiots with signs of congenital syphilis. While Piper[19] found evidence in 5 per cent.

Hahn[20] found 7·4 per cent of idiots in the Alsterdorfer Asylum to be syphilitics.

Shuttleworth/

Shuttleworth & Fletcher Beach[5] found in idiots a clear evidence of inherited syphilis in 1·17 per cent of cases, but suspected its presence in more cases.

Tredgold[4] found 2·5 per cent.

Kerlin[21] investigated the histories of the parents and the grandparents in 100 idiots, and only obtained a history of syphilis is 2 cases, though there was evidence of its presence in 4 cases.

Heubner[22] in striking contrast to the former observers, showed that of 230 idiots about one-fourth were children of syphilitic parents.

Dr. Sutherland[23] suggests that syphilis is a frequent cause of Mongolian imbecility. Of 25 cases investigated he found definite evidence in 11, and strongly suspected it in 3 others. On the other hand Tredgold[4] investigating 20 such cases did not consider syphilis to be of much importance.

Ashby[14] considers syphilis to be a rare cause of idiocy.

The above figures of the various workers represent the number of cases where syphilis was found in the more marked degrees of mental deficiency, i.e. idiocy and imbecility.

Ziehen[24] investigating the lesser degrees of mental deficiency/

deficiency found definite evidence of congenital syphilis in 10 per cent of cases examined, and considered its presence likely in 17 per cent.

Cases of Juvenile General Paralysis are recognised as being syphilitic in origin, in 80 per cent of cases there being either a syphilitic family history or evidence of congenital syphilis (Shuttleworth).

According to these figures inherited syphilis would appear to play a very small part in the causation of mental deficiency. At the same time many observers considered that it was a more important factor in the production of mental defect than was shown by statistical inquiries.

Dr. Mott concludes that syphilis acting on the nervous system is an active agent in the production of congenital weakness and the degeneracy that accompanies it. He says "that the measure of the effects of syphilis in the production of feeble-mindedness and epilepsy should not be estimated only by the cases in which there are visible and characteristic signs of syphilis on the body for he has observed one member of a family with syphilitic notched teeth, another without any external sign but with severe visceral and brain disease, while a third was an imbecile" and he also states "it may be thought that because syphilis of/

of the parents produces sterility, miscarriage and abortion, its dangers are greatly minimised, but it cannot be doubted that if the poison is sufficiently strong to kill the embryo either before or shortly after birth, it must have a deteriorating effect on the offspring that survive."

(Minute of evidence taken before the Royal Commission on the care and control of the Feeble-minded).

The introduction of the Wassermann test gave a reliable means by which the presence of a syphilitic infection might be determined. In this connection a number of investigations have been carried out on the blood serum of idiots.

Raviart, Breton, Petit Gayet and Cannae[25] obtained a positive result in a little over 30 per cent.

In 1909 Deaul[26] examined 330 cases of idiocy. He obtained a positive result in 51 or 15·4 per cent of the cases. Of these 330 cases 13 or 3·9 per cent presented other evidence of syphilitic infection, while 2 with definite signs gave a negative reaction. Of the 51 cases found to be positive only 7 showed evidence of congenital syphilis.
He also examined the serum from the parents of 10 positive cases. 13 parents were examined, 9 gave a positive reaction.

He/

He found, by a careful analysis of his cases, that the percentage of positive results diminished rapidly after the age of 16 years.

Atwood[27] examining 204 cases got a positive result in 30 cases (14·7 per cent).

Chislett[28] (Journal Mental Science 1911) examining 28 cases obtained positive results in 11 cases, 4 of which showed signs of congenital syphilis and 2 were cases of Juvenile General Paralysis.

Tomsen, Boas, Hjort and Leschly[29] examined 2,061 feeble-minded persons and obtained a positive reaction in only 31 cases or 1·5 per cent.

E. Krober[30] obtained a positive result in 21·4 per cent of 262 idiots examined.

Dr. H.F. Watson examined 105 cases of the more marked degrees of mental deficiency in children and obtained a positive Wassermann reaction in 48·5 per cent.

In inquiring into the presence of syphilis as an etiological factor in the causation of the lesser degrees of mental deficiency I was faced with the difficulty of obtaining reliable statistics from the parents of children attending special schools. I was unable to obtain a history of parental/

parental infection in a single case. An illustration of the difficulty may be given:- A case occurred in the examination of normal school children where an apparently healthy child came up for examination. She was a bright, intelligent girl of 6 years but showed typical Hutchinsonian teeth and had scarring at the angles of her mouth. The father had deserted the family and the mother went out to work. There were three of a family; the eldest a girl of 13, tall and thin but with no signs of congenital syphilis, the second a girl of 8 was markedly feeble-minded, bordering on imbecility, but with no syphilitic stigmata, the third was the girl already mentioned. Considering then the case of the second child, it appeared likely that congenital or inherited syphilis could attack the developing brain producing mental defect without causing any external sign of its presence.

On careful and repeated examination of my 124 feeble-minded cases I found that 12 per cent showed signs of congenital syphilis. With the exception of Ziehen[34] who also investigated the lesser degrees of mental deficiency, and who found 10 probably 17 per cent due to syphilis, this is a higher percentage than that obtained by other observers, whose results were obtained by the examination of idiots and imbeciles/

ciles.

In order then to obtain more satisfactory and reliable knowledge as to the presence of syphilis I examined the blood serum by the Wassermann test in 89 cases of mentally defective children of school age. These investigations I carried out in the Clinical Pathology Laboratory of the Glasgow Western Infirmary by the Browning - Cruickshanks - Mackenzie method. The details of the work done will be seen from the tables. Of the 89 defective children examined 40 gave a positive reaction or 44·9 per cent, 11 gave a doubtful reaction or 12·3 per cent, 38 gave a negative reaction or 42.4 per cent.

I divided the cases into three groups.-

I. Those associated with some physical defect, i.e. spastic diplegia, infantile hemiplegia, microcephaly, defective hearing, defective sight, rickets, tuberculosis, cretinism and motor aphasia.

II. Those showing no physical defect, and III those associated with Epilepsy.

The following results were obtained:-

		No.Examined.	Positive.	Negative.	Doubtful.
I.	With Physical Defect	23	9	11	3
II.	Without " "	42	20	17	5
III.	With Epilepsy	24	11	10	3.
	TOTAL	89.	40.	38.	11.

It/

It will be seen that the cases associated with physical defect give a smaller proportion of positive results than those in which the mental defect is unaccompanied by any defective physical condition.

The detailed physical conditions were:-

Condition.	No.examined.	Positive.	Negative.	Doubtful
Congenital Spastic Diplegia (Little)	1	1	0	0.
Infantile Hemiplegia	2	0	1	1.
Microcephalic.	1	1	0	0.
Muscular Atrophy	1	1	0	0.
Cretinism	2	2	0	0.
Motor Aphasia	1	0	1	0.
Deafness of varying degrees	4	1	3	0.
Semiblindness (specific)	2	2	0	0.
Strabismus	5	0	4	1.
Rickets	3	1	2	0.
Tubercular bone disease	1	0	0	1.
TOTAL	23.	9.	11.	3.

It was thought that the cases where the mental defect was associated with epilepsy of all degrees of severity might form

a/

a group by themselves. Of the 24 cases examined 14 were associated with the lesser degree of mental deficiency, while 10 were imbeciles. The importance was recognised of examining all cases of epilepsy. Therefore in addition to the children showing epilepsy associated with mental defect, the serum was obtained from 10 children, the subjects of epileptic attacks, but whose mental capacity was apparently normal.

Considering the results obtained from the examination of epileptic children we find.-

Condition.	No.examined.	Positive.	Negative.	Doubtful.
Epilepsy with slight mental defect	14.	6.	5.	3.
Epilepsy with Imbecility	10.	5.	5.	0.
Epilepsy without mental defect.	10	4	5.	1.
	34.	15.	15.	4.

In all then 34 Epileptic cases were examined, positive reactions were obtained in 15, negative in 15 and doubtful in 4 cases. These results do not differ materially from those obtained from all mentally defective children, and show an almost equal number of positive reactions, whether the epileptic attacks are associated with slight degrees of mental/

mental deficiency, with imbecility or without apparent mental defect.

It is evident that figures obtained from the investigation of a series of cases is of little value as proof of the causation of mental defect unless compared with the results obtained from a series of normal children. It is of importance too that the serum should be obtained from children of normal mental condition of the same age, taken from the same locality and attending neighbouring schools, i.e. from the same environmental conditions. Unfortunately the number of those examined is small, but the results are significant.

23 children of normal mental capacity were examined. Apart from their mental condition and the fact that they showed no signs of congenital syphilis these children were in no way selected cases; so that the possibility of a syphilitic family history was not excluded. Of the 23 children examined 19 gave negative Wassermann reactions, 2 doubtful (brothers) and 2 positive. The mother of one of the positive children had evidence of syphilis and her serum gave a positive Wassermann reaction. Two fathers and three mothers of the negative children were examined and all gave a negative reaction.

It/

It was recognised that in order to determine the importance of syphilis as a factor in the causation of mental deficiency it was not sufficient to examine only the children themselves, but that as many of the members of their families as possible should also be examined. In many of the cases I was unfortunately unable to obtain any of the relatives.

I examined various members of the families of 34 cases. In all 94 relatives were examined.

Details of these are given in the notes of the cases, but the results may be summarised.

I. Relatives of the Defectives who gave a positive Wassermann reaction.

Case 216. Mother positive, sister negative.

Case 92. Brother negative, Sister & Brother doubtful.

Cases 152, 153 & 161. (members of one family) Father and Mother positive, 3 Sisters positive, 1 brother positive, 3 brothers negative.

Case 180. Mother positive, Father & Brother negative.

Case 3. Father, Mother and 3 Sisters positive.

Case 64. Father, Mother and Sister negative.

Case 218. Mother, 3 brothers and 1 sister positive.

Case 24. Grandmother/

Case 24. Grandmother, Mother, 1 Brother and 2 Sisters positive, Aunt negative.

Case 80. Father, Mother and 2 Sisters positive, Sister negative.

Case 74. Mother positive, Sister (Case 73) doubtful.

Case 90. Father, Mother and Brother positive.

Case 125. Mother negative.

Case 142. Father, Mother and Sister positive, Sister negative, Epileptic Defective Sister negative.

Case 149. Mother negative.

II. Relatives of the defectives who gave a negative Wassermann reaction.

Case 39. Father negative, Mother and twin sister positive.

Cases 172, 173 and 174. Mother positive.

Case 176. Mother positive.

Case 213. Mother and 2 sisters negative.

Case 109. Mother negative.

Case 112. Mother negative.

Case 121. Mother negative.

Case 123. Mother negative.

Case 145. Father/

Case 145. Father, Mother and 2 Sisters positive, Sister negative.

Case 168. Father positive, Mother and 2 Sisters negative.

Case 147. Mother positive.

One defective girl was not examined but her mother and sister both gave positive results.

III. Relatives of the defectives who gave a doubtful Wassermann reaction.

Case 4. Mother negative.

Case 73. Mother and brother (Case 74) positive.

Case 138. Mother positive, sister doubtful.

Case 190. Mother and sister positive, Father, sister and 2 brothers negative.

Relatives of Epileptics showing no mental deficiency.

1. Those who gave a Positive Wassermann reaction.

Cases 134 and 135. Mother and brother positive.

Case 162. Mother positive, two brothers negative.

2. Of those who gave a Negative Wassermann reaction.

Case 193. Mother and sister positive, Father, sister and brother negative, sister (Case 190) negative.

Case 87/

Case 87. Father and Mother negative, 2 sisters positive.

Cases 103 & 104. Mother positive, Father negative.

Cases 128. Mother and brother positive, Father and three brothers negative.

3. Of those who gave a doubtful Wassermann reaction.

Case 107. Mother doubtful (but with a definite Syphilitic history).

The great importance is thus brought out of examining the serum in the other members of the family. It is specially important to investigate the families of children giving a negative or doubtful reaction. By referring to the doubtful group it is seen that of the 4 cases investigated a positive result was obtained in some member of the family in all but one instance, and in the exception only the mother was examined. This increases the number of cases in which there was a syphilitic infection by three. It is unfortunate that more members of the families of those children giving a negative reaction could not be obtained.

In one family a boy and two girls were examples of the lesser/

lesser degree of mental deficiency. They attended a class for defective children and were reported to be making considerable progress in school work. Their father had died as the result of an accident and their mother had married again, her second husband being a drunkard. The mother complained of loss of memory and she was dirty and untidy. The three children all gave negative Wassermann reactions but in the case of the mother a positive Wassermann reaction was obtained. It is possible, however, that she may have been infected subsequent to the birth of the three children.

In another case where the serum of the defective girl gave a negative reaction the serum of the mother, who was a big healthy woman and said she had never had a day's illness in her life, gave a positive Wassermann reaction.

Even in the small number of negative cases where the families were investigated, 13 in all, a positive Wassermann reaction was obtained in some member in 8 instances.

We thus see that with the 3 positives in the doubtful cases, and the 1 positive family where the defective girl herself was not examined, the number of mentally defective children where a syphilitic infection was found to be present, either as a result of the examination of the children/

children themselves or of some member of their families, was raised to 52 in 90 cases examined. (Number raised from 89 to 90 by the inclusion of the case where the relatives only were examined). This raises the percentage from 44.9 to 57.7 per cent.

All the negative and doubtful cases of epilepsy without mental defect were investigated and in all but 1 case a positive result was obtained from some member or members of the family. In the one exception the child and mother both gave a doubtful reaction, but the mother had a definite history of syphilitic infection. This would bring the number of those cases with a syphilitic infection to 100 per cent.

If the defective and epileptic children are grouped to-gether we get 100 children showing a syphilitic infection in 62 cases.

	No.Examined.	Positive.		Negative.		Doubtful.	
Mentally Defective without epilepsy.	65	29	40	28	38.	8	11.
Mentally Defective with epilepsy	24	11		10		3	
Epilepsy without mental defect.	10	4		5		1.	
Members of Families of above.	94	49		39.		6.	
Normal Children	23.	2.		19.		2.	
Parents of Normal Children.	6.	1.		5.		0.	
TOTAL..	222.	96.		106.		20.	

44·9 per cent of Defective Children gave a positive Wassermann reaction.

When members of families were included a syphilitic infection was present in 57·7 per cent.

Of 10 Epileptic Children without apparent mental defect 4 gave a positive reaction, but where the families are included syphilitic infection is noted in all cases.

Syphilitic infection was found to be present in 62 per cent of Defective and Epileptic Children, taken together.

My results show a larger proportion of cases giving a positive reaction than any of the previous observers, with the exception of/

of Dr. Watson who found 48.5 per cent of the defective children themselves to give a positive reaction.

Dean[96] has suggested that a difference in results may be due to the fact that the percentage of positive reactions diminishes rapidly after the 16th year, and that in many cases the results are got from the feeble-minded of all ages. This might explain the larger percentage of positive results in my series as no case was over 16 years, the majority being between the ages of 6 and 14. This, too, is borne out by the fact that the results obtained by Dr. Watson in his examination of younger children closely resemble mine.

It must also be remembered that only 50 per cent of cases of latent syphilis give a positive Wassermann reaction, which may also explain the difference in results. It is therefore probable that a greater number than those giving positive results are actually infected and this point may be brought out by a further examination of the families of cases giving a negative result.

With the exception of 10 cases of imbecility associated with epilepsy, and two of congenital imbecility, I have limited my observations to children who are not imbecile but/

but of defective intelligence, yet who are capable of receiving some benefit from instruction in special schools, and therefore an important group from a sociological point of view.

The syphilitic poison may act directly on the germ plasm, impairing its vitality, and in this way produce a hereditary condition. Evidence of this could only be obtained by tracing the infection through several generations. In one instance I was able to examine the blood in members of three generations, the mental defect occurring in the third generation. Case 24 a mentally defective boy with speech defect and petit mal. He was the second of 4 children, all alive. His mother was a strong healthy-looking woman. His father was not seen. The mother and 4 children all gave a positive Wassermann reaction. The mother's father had died of Apoplexy. The mother's mother was alive and well and her serum gave a positive Wassermann reaction. A younger sister of the mother (Aunt of the defective child) had tubercular disease of the spine and a psoas abscess. Her serum gave a negative Wassermann reaction. Although in this case a positive reaction was obtained in the Grandmother, Mother and Children, a reinfection in the mother's case would have to be excluded before it could be taken as definite evidence of/

of hereditary transmission.

Syphilis must then be recognised as a causative factor in a very considerable number of cases of mental deficiency. The question arises, does it occur as sole cause or are other factors necessary before it can so effect the cerebral cells as to produce mental defect?

Tredgold[4] considers that other factors are necessary and particularly a neuropathic heredity. Mott says "Though hereditary neurosis or psychosis greatly increases the liability of the syphilitic poison to affect the nervous system yet in numbers of the cases there was no pre-existing neuropathic family history." I have carefully inquired into the family history in nearly all cases and have obtained a neuropathic history in 9 of the 40 positive cases, and in 4 of the 38 negative cases. This of course represents the minimum owing to the difficulty entailed in such an inquiry. Histories of alcoholism and tuberculosis were also obtained in a small percentage of cases. 12·5 per cent for both. In 2 of the alcoholic cases there was also a neuropathic heredity.

Another noticeable feature is that of the large proportion of cases which have been shown to be syphilitic by the result of the Wassermann reaction with their own blood/

blood, and the Wassermann reaction and history of relatives, only a small number present the stigmata of congenital syphilis. I found it only in 8 of the positive cases or 20 per cent. It is recognised that normal mental development is quite compatible with the existence of congenital syphilis as evidenced by stigmata. It might therefore be that in cases where syphilis exerts its action on the developing nerve tissue the external evidences are not produced.

A consideration of the results obtained lead to the conclusions.-

1. Syphilis is the causative factor in a very considerable percentage of cases of mental deficiency (over 50 per cent) and in many instances it is the sole causative agent, although in a few it is associated with other factors, particularly a neuropathic heredity.

2. Syphilis is also the main causative factor in the production of Epilepsy, both in cases where the Epilepsy is associated with mental deficiency and in cases where no apparent mental defect exists.

3. The investigation by means of the Wassermann test into/

into the families of defective children who have given a negative reaction has shown that syphilis is a causative factor in a still higher percentage of cases; while an examination of the families of those children giving a positive reaction gives further evidence of the presence of a syphilitic infection.

4. A very small percentage of cases giving a positive Wassermann reaction show external evidence of congenital syphilis.

WASSERMANN REACTION POSITIVE.

A. Without Physical Defect.
B. With Physical Defect.
C. With Epilepsy.

- - - - - - - - - - - - - -

A. CASE 5. Girl Aged 14.

Well grown girl with dull expression open mouth and protruding lower jaw. Blepharitis. Palate high and v-shaped with teeth crowded together. Chilblains on ears. Born at 8 months, a quiet baby but slept badly. Had irregular movements of hands and fingers from infancy till 11 years old. Rheumatism at 11 years. Walked and talked at three years. 3rd of a family of 5. Her mother was 30 at the time of her birth and had had pleurisy 3 months previously. 2 mis-carriages between the 1st and 2nd children.

Mother was nervous. Family history good.

Wassermann reaction positive.

A. CASE 8. Boy Aged 10.

Small boy with asymmetrical and deformed face, the result of being run over. His mental condition was said to date from the accident 4 years ago.

Central incisor peg shaped.

Father a sailor

Mother strong. Aged 24 at his birth.

Eldest of 3 children. One died of Pneumonia.

Others strong and well. No mis-carriages.
Family history good. Wassermann reaction positive.

A. CASE 9. Boy Aged 10.

Large well developed boy. His birth was prolonged. He started walking at 9 months, but was late in talking. Was scalded at 3 months.

Was knocked over by a car at 2 years and his head cut. Mental dullness was noticed after this and he was said at times to "whirl round like a top". Has had adenoids and tonsils removed. Youngest of 7.

4 died of Diphtheria, 2 alive and strong.

Father died of consumption.

Paternal grandmother died of consumption.

Mother strong. Neuralgia in the family.

Wassermann reaction positive.

A. CASE 10. Boy Aged 11.

Small boy with rough scaly skin, dry hair and small head. Nervous.

Birth normal, mother aged 29.

He was late of sitting up without support, walked at 1-9/12, and talked over 2 years.

4th of 8 children. One died of convulsions.

Two youngest had convulsions, others healthy.

Maternal grandfather died of apoplexy.

Father's family strong.

Mother well but had cystitis during pregnancy.

No mis-carriages.

Wassermann reaction positive.

A. CASE 12. Boy Aged 11.

Small head. Dull expression. High palate and lisps.

Tendency to eczema oris.

3rd of 6 children, 3 dead, one of Pneumonia, one of Hydrocephalus, one of Gastro-enteritis.

No mis-carriages. Youngest boy mentally backward.

Mother and father healthy.

Wassermann reaction prsitive.

A. CASE 20. Boy Aged 13.

Tall boy with open mouth and dull expression.

Palate high with teeth irregular and slight notching of incisors.

Talked at 3 years, walked at 5 years.

Had one convulsion at 4 years.

Eldest of 2. Other strong and healthy.

Mother dead, cause unknown.

Father alive and well.

Two cousins died of Phthisis.

Wassermann reaction positive.

A. CASE 30. Boy Aged 10.

Healthy Boy. Development normal.

Seventh of 9 children. One died of"blood poisoning".

One sister with defective hearing.

Mother healthy. Has occasional swelling of left knee.

Was 40 years old when boy born.

2 mis-carriages.

Father has "heart disease".

Wassermann reaction positive.

A. CASE 36. Boy Aged 14.

Tall thin boy. Skin rough and scaly. Palate high.

Wassermann reaction positive.

A. CASE 41. Girl Aged 11.

Very feeble-minded. Restless and talkative with open mouth and salivation.

Could not sit up till nearly 5 years.

Walked 5 years, talked 5½ years.

Had one fit at 5 years.

Only child. No mis-carriages.

Mother strong.

Father drinks. His people drink and one stutters.

Wassermann reaction positive.

A. CASE 50. Boy Aged 11.

Healthy boy with small round head. Restless and occasional twitching of mouth. Speech defective. Blepharitis. Had convulsions at $3\frac{1}{2}$ years.

Seven other children. One deaf mute.

Wassermann reaction positive.

A. CASE 216. Girl Aged 9.

Healthy girl. Movement slow but very excitable.

Walked and talked at 1 year.

3rd of 3 children. One died of Pneumonia. Oldest girl alive and strong.

Mother 30 when born and got a fright during pregnancy.

Father strong.

Wassermann reaction positive.

CASE 65. Woman Aged 39.

Mother of above. Feeble-minded. Dirty and untidy.

Wassermann reaction positive.

CASE 66. Girl Aged 17.

Strong healthy girl.

Wassermann reaction negative.

A. CASE 92. Girl Aged 11.

Healthy. Physical condition good.

Did not speak till seven years.

Father alcoholic. Mother dirty and untidy.

House neglected.

Wassermann reaction positive.

CASE 93. Boy Aged 9.

Brother of Case 92. Healthy.

Wassermann reaction negative.

CASE 94. Girl Aged 7.

Sister of Case 92. Notched incisor.

Wassermann reaction doubtful.

CASE 95. Boy Aged 4½.

Brother of Case 92. Fan-shaped scarring at angles of mouth.

Wassermann reaction doubtful.

A. CASE 161. Girl Aged 14.

Mentally deficient. Prostitute.
Notched incisors.
Wassermann reaction positive.

C. CASE 152. Girl Aged 11.

Sister of Case 161. Imbecility with Epilepsy.
Wassermann reaction positive.

A. CASE 153. Girl Aged 9.

Sister of Case 161. Mentally deficient.
Wassermann reaction positive.

CASE 154. Girl Aged 7.

Healthy. Teeth normal. Sister of Case 161.
Wassermann reaction positive.

CASE 155. Boy Aged 5.

Healthy. Brother of Case 161.
Wassermann reaction negative.

CASE 156. Boy Aged 3.

Brother of Case 161. Hydrocephalus. Head Circf. 23".
Wassermann reaction negative.

CASES 157 & 158. Twin Boys Aged 1½ years.

Brothers of Case 161. Healthy.
Wassermann reaction 157 positive, 158 negative.

CASES 159 & 160 Twin Girls 2/12.

Sisters of Case 161. Convulsions in both since birth.
Wassermann reaction in both positive.

CASE 150. M.

Father of Case 161. Drunkard and gambler.
Wassermann reaction positive.

CASE 151. F.

Mother of Case 161. Drunkard and was not sober
for one week before last confinement.
Wassermann reaction positive.

A. CASE 180. Girl Aged 11.

Small thin girl with small head, broad base of nose and epicanthus. Skin dry and scaly. Palate high and arched. Slight notching of left upper incisor.

Father occasionally drinks.

Wassermann reaction positive.

CASE 205.M. Aged 52.

Father of Case 180. Healthy.

Wassermann reaction negative.

CASE 206. F. Aged 44.

Mother of Case 180. Healthy.

Wassermann reaction positive.

CASE 207. M. Aged 22.

Brother of Case 180. Healthy.

Wassermann reaction negative.

A. CASE 184. Girl Aged 12 years.

Bad speaker.

Blepharitis and Eczema oris.

Adopted child.

Wassermann reaction positive.

A. CASE 221. Boy Aged 9.

Small boy. Broad base of nose and epicanthus.

Teeth wide apart discoloured and eroded.

Family history unknown. He is the 3rd illegitimate child. His mother drank, his father unknown.

Wassermann reaction positive.

A. CASE 222. Boy Aged 11.

Big boy. Restless, steals and untruthful.

Suffers from headaches. A reliable history of his development was not obtained as his mother is feebleminded.

Father's brother could not learn to read or write and is now a gambler.

Second of 4 children. All alive and nervous.

4 mis-carriages, first occurred after 3 living children.

Wassermann reaction positive.

A. CASE 203. Girl Aged 14.

Congenital Imbecile. Never been to school.
Salivates. Dirty habits, poorly clad and verminous
Bad tempered. Adopted child. Parents unknown.
Wassermann reaction positive.

A. CASE 204. Boy Aged 13.

Imbecile and only at school 1 year.
Mother unmarried, 3 abortions, 3 mis-carriages,
syphilis 19 years ago.
Wassermann reaction positive.

B. CASE 1. Boy Aged 12.

Small thin boy. Cretin.
Been treated with Thyroid since 6 years with
marked physical and mental improvement.
Very restless.
Birth normal. Noticed to have peculiar movements
of eyes in infancy. Teeth irregular.
Walked 3. Talked 6 years.
4th of 7 children. 5 dead.
2 still-born, one lived one day, one died of
Tubercular Peritonitis, one of Pneumonia.
Father strong. One brother in an Asylum.
Mother strong but nervous, 11 years younger than her
husband.
Wassermann reaction positive.

B. CASE 2. Boy Aged 11.

Small boy with small head. Expression dull with open mouth. High arched palate with irregular teeth. Speech defective. Marked wasting of left Deltoid muscle.

Birth normal. Walked and talked at 1 year.

4th of 5 children. One died of whooping-cough others healthy.

Mother strong 5 years older than father, was 38 when boy born.

Father died of Phthisis.

Wassermann reaction positive.

B. CASE 3. Girl Aged 12.

Small dwarfed child. Anaemic.

Birth was normal. Exceptionally quiet baby.

Walked at 2 and talked at 3 years.

Has grown none since 5 years old.

7th of 11 children. 4 dead. Last were twins who died in 24 hours. One died at 6/12 years of convulsions, one with a "lump at the back of her head".

Wassermann reaction positive.

CASE 115.

Father of Case 3. Chronic Bronchitis.

Syphilitic psoriasis.

Wassermann reaction positive.

CASE 116.

Mother of Case 3. Strong but nervous.

Wassermann reaction positive.

CASE 117.

Girl 17. Sister of Case 3. Slightly deaf.

Wassermann reaction positive.

CASE 118.

Girl 10. Sister of Case 3. Healthy.

Wassermann reaction positive.

CASE 119.

Girl 8. Sister of Case 3. Healthy.

Wassermann reaction positive.

B. CASE 13. Boy Aged 11.

Thin poorly nourished, dirty and venomous. Very restless. Hoarse. Left eye shrunken. Right eye Interstitial keratitis. Almost totally blind. Mother in an Asylum. Father alive and well. Wassermann reaction positive.

B. CASE 19. Girl Aged 15.

Small girl with rickets and marked tibial curves. Precipitate labour. She fell on her head and a swelling formed which had to be lanced.
Walked late and talked over 2.
Has passion fits and loses control.
3rd of 8 children. Onedied of "Consumption" 1-7/12 years. Mother nervous. All her family had convulsions.
Father died of Phthisis.
Wassermann reaction positive,

B. CASE 64. Girl Aged 10.

Cerebral diplegia. Protrusion of lower jaw, open mouth and salivation. Speech defective. High palate with teeth crowded together.

Mother fell two days before birth. Birth was normal but child blue and brought round with difficulty.

Walked $3\frac{1}{2}$ years. Talked 5 years.

One convulsion at onset of Chicken-pox.

Youngest of 2.

Wassermann reaction positive.

CASE 61.

Father of Case 64. Phthisis.

Father died in Asylum result of accident.

Wassermann reaction negative.

CASE 62.

Mother of Case 64. Headaches. Erysipelas 4 years ago. Youngest of 11 and a twin. Bronchitis in family. Oldest child 17. No pregnancy for 6 years when mis-carriage. Next defective child followed by 3 mis-carriages, the last three years ago.

Wassermann reaction negative.

CASE 63. Girl Aged 17.

Sister of Case 64. Strong but stammers.

Wassermann reaction negative.

Small thin delicate boy. Dull expression with open mouth and protrusion of lower jaw. Hearing defective. Oldest of 5 children, all the others well and strong. Father and mother strong. Father drinks and when drunk becomes very passionate. Three of his family have died in an Asylum, all his brothers drink. Wassermann reaction positive.

CASE 67.

Mother of above. Stout healthy woman.
Wassermann reaction positive.

CASE 68. Boy Aged 12.

Healthy boy. Brother of Case 218.
Wassermann reaction positive.

CASE 69. Boy Aged 10.

Healthy. Brother of Case 218.
Wassermann reaction doubtful.

CASE 70. Boy Aged 8.

Healthy. Enlarged Tonsils. Brother of Case 218.
Wassermann reaction doubtful.

CASE 71. Girl Aged 6.

Healthy. Sister of Case 218.
Wassermann reaction positive.

B. CASE 217. Boy Aged 11.

Thin boy with defective sight owing to Interstitial keratitis and internal squint. Has scarring at angles of mouth, and notching of incisors.
No teeth till 1 year. Walked at 2 years. Talked at 1½ years. At one year he had convulsions for a fortnight followed by temporary loss of power in right arm and leg. Had had chorea.
Youngest of 3 children, 5 years after the second.
One died at 14 months of Hydrocephalus.
Mother not strong had Bronchitis and Colitis during pregnancy and was 30 at time of birth.
Her family are consumptive.
Father's family con sumptive and one sister insane.
Wassermann reaction positive.

B. CASE 219. Boy Aged 10.

Small boy with microcephalic skull.
Birth normal, walked and talked before 1 year.
Eldest of 4 all alive and strong.
Family history good. Mother was 18 years when he was born.
Wassermann reaction positive.

Small boy with speech defect.

Labour was precipitate and he was a blue baby.

Has a bad temper.

Second of 4 children, mis-carriage between him and first.

Wassermann reaction positive.

CASE 22. F. Aged 28.

Mother of Case 24. Strong and healthy.

Wassermann reaction positive.

CASE 23. Boy Aged 8.

Brother of Case 24. Not strong. Convulsions in infancy. Wassermann reaction positive.

CASE 25. Girl Aged 3.

Sister of Case 24. Strong.

Wassermann reaction positive.

CASE 26. Girl Aged 1-10/12.

Sister of Case 24. Small and thin. Convulsions in infancy. Diphtheria at 1 year followed by paralysis.

Wassermann reaction positive.

CASE 21. Aged 55.

Grandmother of Case 24. Healthy. Husband died of Apoplexy. Wassermann reaction positive.

CASE 44. Girl Aged 15.

Aunt of Case 24. Tubercular spine and abscess. Wassermann reaction negative.

C. CASE 29. Boy Aged 7.

Small pale boy with small head. Very restless.
Speech defective. Petit mal.
Palate high. Teeth very bad.
Walked 2 years. Talked - not noticed.
convulsions at 2 years. Petit mal since.
4th of 7 children. no mis-carriages.
4 dead - 2 still-born, 2 when teething.
One of others had Cerebro-spinal Meningitis.
Family history good.
Wassermann reaction positive.

C. CASE 74. Boy Aged 10.

Epileptic Imbecile with continual restless movements. Loss of power of right arm with contraction at elbow and wrist. Mouth open and salivation. His birth was precipitate. Was late of sitting up. Walked at 2½ yrs. and talked about 3 years. Convulsions since infancy.
Brother of Case 73.
Wassermann reaction positive.

CASE 72. Aged 40.

Tall nervous woman with extreme anaemia, fullness under eyes and swelling of ankles.
Mother of Cases 73 and 74.
Wassermann reaction positive.

C. CASE 80. Girl Aged 3.

Cogenital Imbecile with Epilepsy. Infantile paralysis at 1½ years involving right arm and leg. Bad tempered. Salivates. No control over bladder or bowels.

Wassermann reaction positive.

CASE 75. M. Aged 38.

Father of Case 80. Grocer. Healthy.

Wassermann reaction positive.

CASE 76. F. Aged 39.

Mother of Case 80. Specific history before marriage. One abortion before marriage. Alcoholic excess. House dirty.

Wassermann reaction positive.

CASE 77. Girl Aged 10.

Sister of Case 80. Congenital Syphilis at 2/12.

Wassermann reaction positive.

CASE 78. Girl Aged 7.

Sister of Case 80. Peeling of skin at 3/12.

Wassermann reaction positive.

CASE 79. Girl Aged 5.

Sister of Case 80. Healthy.

Wassermann reaction negative.

C. CASE 90. Girl Aged 13.

Epileptic with mental defect. Impulsive. 2nd of 3 children.

Wassermann reaction positive.

CASE 88. M. Aged 36.

Father of Case 90. Has specific disease before. marriage. Is weak minded and alcoholic.

Wassermann reaction positive.

CASE 89. F. Aged 36.

Mother of Case 90. Specific disease after marriage.

Wassermann reaction

CASE 91. Boy Aged 5.

Brother of Case 90. No children between the two. Healthy.

Wassermann reaction positive.

C. CASE 125. Girl Aged 11.

Epileptic with mental deficiency.

Father a soldier.

Wassermann reaction positive.

CASE 124. F.

Mother of Case 125. Drinks and lives in very poor lodgings. Three children, 2 oldest died of convulsions in 1st year. Case 125 only living child.

Wassermann reaction negative.

C. CASE 142. Girl 15.

Epileptic, Sister of 145. Feebleminded and a prostitute.

Wassermann reaction positive.

CASE 141. M.

Father of case 142. Drunkard.

Wassermann reaction positive.

CASE 140. F.

Mother of Case 142. Weakminded and immoral. Home conditions bad. Furniture in pawn.

Wassermann reaction positive.

CASE 143. Girl 9.

Sister of Cases 142 and 145. No children between Case 142 and herself. Healthy.

Wassermann reaction positive.

CASE 144. Girl Aged 6.

Sister of 142. Healthy.

Wassermann reaction negative.

CASE 145. Boy Aged 4.

Brother of Case 142. Epileptic with mental deficiency and spasitc diplegia. Youngest of four children.

Wassermann reaction negative.

C. CASE 149. Girl Aged 11.

Imbecile with Epilepsy.

Father a drunkard and feebleminded. Has a brother in an Asylum and two of his children are imbeciles.

Mother weakminded and hysterical. Two abortions before Case 149. Her father was a criminal.

Wassermann reaction positive.

CASE 148.

Mother of Case 149.

Wassermann reaction negative.

C. CASE 115. Girl Aged 10.

Mental deficiency with Petit mal.

Birth was normal 3 weeks before time. Walked at 10 months and talked at 1 year.

4th of 6 children. 2 dead, one of Pneumonia and one still-born. 1 miscarriage.

Mother has had rheumatic fever, was 29 when child born. Home conditions poor.

Wassermann reaction positive.

C. CASE 220. Boy Aged 9.

Pale boy. Very restless. Epileptic, fits increasing very much in frequency.

1st of twins. Instruments required. Had convulsions for two days after birth. At 2 fell down steps and fits date from this accident. He walked at 1 year and 4 months, bottle fed.

Youngest of 4 children, one still-born.

Has an uncle deaf mute.

Wassermann reaction positive.

WASSERMANN REACTION NEGATIVE.

A. CASE 15. Girl Aged 15.

Tall thin girl with arched palate and protrusion of upper teeth, poor circulation and anxious expression.
Her birth and development were normal.
At 7 years had meningitis and convulsions to which her mental state was attributed.
Youngest of 10, 4 dead - girl 20 of gastric ulcer, Boy 14 of Rheumatism, Baby 10/12 of Pneumonia, Boy 2 days - cause unknown. No mis-carriages.
Father strong. Mother stout healthy woman with cataract.
Wassermann reaction negative.

A. CASE 38. Girl Aged 7.

Healthy girl with narrow head and overhanging forehead.
Speech defective. Palate high. Teeth bad. Had operation for "tumours at back of neck" at 8/12 years.
Walked 1½ years. Talked late.
One other child strong and well.
Father and Mother healthy. Family history good.
Wassermann reaction negative.

A. CASE 39. Boy Aged 11 years.

Thin boy with frightened expression and projecting lower jaw.

2nd of twins.

Walked 15 months. Talked nearly 2 years.

Had a fall at 7 years after which unconscious for 10 minutes and seemed to lose power of legs.

Youngest of 8. 2 dead, one of dropsy, one of "Congestion of Brain". Others well and strong.

2 mis-carriages.

Wassermann reaction negative.

CASE 82.

Father of Case 39. Baker. Drinks. Father died of a "cold on his chest".

Wassermann reaction negative.

CASE 83.

Mother of Case 39. Short, fat and talkative.

stralimus of right eye. Has had brain fever.

Her mother died of apoplexy.

Wassermann reaction positive.

CASE 84. Girl Aged 11.

Twin sister of above. Healthy, but nervous.

Wassermann reaction positive.

A. CASE 42. Boy Aged 11.

Healthy boy with defective speech. Palate high.
Walked at 3 years. Started talking over 2 years.
Mischievous. 3rd of 5 children, all the others
dead. 1st at one year from malnutrition, 2nd
from Pneumonia, 4th Measles, 5th
No mis-carriages.
Wassermann reaction negative.

A. CASE 43. Girl Aged 10.

Small restless girl with small head.
High palate and irregular teeth.
Birth was prolonged and blue when born.
Had convulsions in infancy and fits occasionally.
till 7 years old.
13th of 14 children. 6 dead, one of Bronchitis,
1 of fits, 1 of fright, 3 in infancy.
One mis-carriage.
Mother healthy 39 at time of birth.
Father healthy.
Family history good.
Wassermann reaction negative.

A. CASE 45. Boy Aged 11.

Thin boy with dull expression and open mouth.
High palate and irregular teeth.
History not obtained.
Wassermann reaction negative.

A. CASE 47. Boy Aged 12.

Strong healthy boy.

Was run over at 6 years by a car, said to be all right till then.

Father and Mother strong.

Wassermann reaction negative.

A. CASE 171. Girl Aged 9.

Mentally deficient without physical defect.

Slightly anaemic. Her birth was normal.

She walked at 2 years and talked at 1½ years.

Has had no convulsions.

First noticed to be dull and quiet at six years of age.

The 3rd of 6 children all alive and well.

Family history good.

Wassermann reaction negative.

A. CASE 172 Girl Aged 10.

Mentally defective.

Wassermann reaction negative.

A. CASE 173 Girl Aged 8.

Sister of Case 172. Also mentally defective.

Wassermann reaction negative.

Father was killed in an accident. Drank.

Step-father also drinks and home conditions very poor.

CASE 209. F. Aged 44.

Mother of cases 172 and 173.

Anaemic and with internal squint. Dirty and untidy and complains of loss of memory.

Has had 9 living children, 3 dead of diphtheria, measles, and whooping-cough.

2 mis-carriages.

Wassermann reaction positive.

A. CASE 175 Boy Aged 11.

Mentally defective, associated with word blindness.

Wassermann reaction negative.

A. CASE 176.Girl Aged 16.

Mentally defective girl with no physical defect. Healthy but speaks badly. Was born three weeks before full time was very blue and was brought round with difficulty. She was 5 years before walked and over 5 before attempted to talk and still talks badly.

3rd of 4 children. 1st still-born, 2nd died of Measles at 1 year, 3rd well and strong.

Family history good.

Wassermann reaction negative.

CASE 208.F. Aged 53.

Mother of 176. Big stout healthy looking woman who says she has never been ill.

Wassermann reaction positive.

A. CASE 179. Girl Aged 10.

Mentally defective with speech defect.

Palate high and arched. Teeth irregular.

Mother healthy, great talker and house very untidy.

Wassermann reaction negative.

A. CASE 182. Girl Aged 14.

Big healthy girl with large head. Palate arched.

Has attacks of Bronchitis.

Mother threatened with Phthisis.

Wassermann reaction negative.

A. CASE 214. Boy Aged 10.

Small and pale with fullness under eyes.
His mental deficiency is very slight but he had also a brother in the mentally defective school.
Mother very small woman. Talks incessantly.
Youngest of 10, others all dead and several died suddenly.
Wassermann reaction negative.

B. CASE 7. Boy Aged 11.

Stout boy with internal squint. Continued restless movements. Palate high and narrow with irregular eroded teeth.
His labour was induced 3 weeks early, he was small and blue and there was some difficulty in bringing him round. Had convulsions at 3 months which lasted for about 1 week. Was late of noticing surroundingsand late of sitting up without support.
Walked at 18 months. Started talking but very badly at 18 months. 2nd of 4 children, one other living, one died of whooping-cough, i still-born.
Mother aged 30 at birth. Family history good.
Wassermann reaction negative.

A. CASE 183. Boy Aged 11.

Healthy boy. Extreme restlessness.

Palate high and teeth irregular.

Mother strong and home conditions good.

Wassermann reaction negative.

A. CASE 213. Boy Aged 8.

Small restless nervous boy. Will not speak.

Youngest of 6. 2 dead. 1 mis-carriage.

Both grandfathers died of apoplexy.

Wassermann reaction negative.

CASE 210.F.Aged 45.

Mother of 213. Small and very anaemic.

Wassermann reaction negative.

CASE 211. Girl Aged 13.

Sister of Case 213. Healthy.

Wassermann reaction negative.

CASE 212. Girl Aged 10.

Sister of case 213. Healthy.

Wassermann reaction negative.

B. CASE 16. Girl Aged 7.

Small stout girl with rickets. Mongul type.

Mental condition poor.

Walked 2 years. Late of talking.

4th of 7 children. 2 died of convulsions in infancy.

Father and Mother both have rickets.

Some of Mother's family said to be"thick at speaking".

Wassermann reaction negative.

B. CASE 17. Girl Aged 7.

Small stout girl with open and small mouth.

Defective speech. Deaf.

Mental condition poor.

Mother strong. Family history not obtained.

Wassermann reaction negative.

B. CASE 31. Girl Aged 9.

Small round head. Restless. Slightly deaf with enlarged tonsil and otorrhoea.

Instruments at birth. Walked 1-3/12 years. Talked 2 years. 4th of 5 children. 3 dead. 2 of phthisis one measles. Father died of Phthisis.

Consumptive family.

Mother strong.

Wassermann reaction negative.

B. CASE 32. Girl Aged 8.

Well nourished girl unable to speak but hears.

Restless and nervous.

Her birth was long and instruments required.

Walked 1-7/12 years.

First of 3 children others well.

Mother nervous during pregnancy aged 26.

First pregnancy ended in mis-carriage.

Wassermann reaction negative.

B. CASE 37. Girl Aged 9.

Small thin girl with small head and internal squint.

Palate high and teeth crowded together.

Was 7 months child small and blue and difficult to rear. Walked and talked at 1 year.

Mental condition good.

11th of 12 children. 4 dead, 2 of c onsumption and 2 of measles. Girl of 17 very backward and could not learn to read. 2 mis-carriages before birth of girl.

Mother suffers from chronic Bronchitis. Was 37 when girl born.

Father strong.

Wassermann reaction negative.

B. CASE 40. Girl Aged 10.

Thin girl with small head. Infantile hemiplegia affecting right side.

Wassermann reaction negative.

B. CASE 46. Girl Aged 12.

Stout healthy girl very deaf and with defective speech. Palate high and v. shaped.

Mother dead cause unknown. Sister insane.

Wassermann reaction negative.

B. CASE 178. Girl Aged 10.

Mentally defective. Rickets with marked tibial curves.

Mother a stewardess.

Wassermann reaction negative.

B, CASE 181. Girl Aged 14.

Thin girl with prominence of occipital region and protrusion of lower jaw.

Internal squint and wears glasses. Headaches.

Father and mother strong and well.

Wassermann reaction negative.

B. CASE 174. Boy Aged 12.

Internal squint. Mentally defective and a bully.
Two sisters M.D. without physical defect.
Both gave negative wassermann reaction. Cases 172 and 173. Mother positive Case 209.
Wassermann reaction negative.

C. CASE 6. Boy Aged 12.

Tall well developed boy with occasional epileptic fits. Speech defective. Palate high and narrow. Teeth eroded and discoloured. No family history obtained.
Wassermann reaction negative.

C. CASE 27. Boy Aged 12.

Big strong boy with nocturnal epilepsy
Fits since 2 or 3 years old.
Mother didd as the result of a burn.
Father alive.
Youngest of 12. 8 dead cause unknown.
Wassermann reaction negative.

C. CASE 109. Girl Aged 14.

Epilepsy with mental deficiency.

Her father and two brothers are in Canada.

She was not allowed to land and so returned with her mother to this country.

Wassermann reaction negative.

CASE 110. F. Aged 34.

Mother of Case 109. Healthy.

Wassermann reaction negative.

C. CASE 112. Girl Aged 11.

Epilepsy with imbecility.

Two maternal uncles were in Ayr Asylum.

Her mother drinks to excess.

Her matermal grandfather drunkard.

" " great grandmother was epileptic.

Wassermann reaction negative.

CASE 111 Femald Aged 39.

Mother of Case 112.

Wassermann reaction negative.

C. CASE 121. Boy Aged 14.

Epileptic with mental deficiency. Had to leave school 2 years ago owing to impulsive attacks.
Father a drunkard.
Wassermann reaction negative.

CASE 120. F.

Mother of Case 121. Has had 4 pregnancies, 3 of which were abortions between 3 and 4 months.
Case 121 is the only living child.
Epileptic, but fits occur at long intervals.
Wassermann reaction negative.

C. CASE 123. Boy Aged 9.

Epileptic with mental deficiency and infantile paralysis of both legs and right arm.
Mental defect is said to have followed paralysis.
Wassermann reaction negative.

CASE 122. F.

Mother of Case 123. Alcoholic. Syphilis before marriage. She has had 6 pregnancies, 2 children died of meningitis aged 2 and 4 years, following that 3 abortions at 3rd month. Case 123 only living child.
Wassermann reaction negative.

C. CASE 147. Boy Aged 5.

Epileptic with mental deficiency and infantile paralysis. 2 sisters alive and well.

Wassermann reaction negative.

CASE 146. F.

Mother of Case 147. Drinks and does not live with her husband.

Wassermann reaction positive.

C. CASE 168. Girl Aged 5.

Epilepsy with mental deficiency and spartic diplegia.
Cannot walk nor sit up.
Labour lasted two days and instruments were required. Fits began at 3 months.
Wassermann reaction negative.

CASE 166. M.

Father of Case 168.
Wassermann reaction positive.

CASE 167. F.

Mother of Case 168.
Wassermann reaction negative.

CASE 169. Girl Aged 3.

Sister of Case 168. Had convulsions at 3 months, none since.
Wassermann reaction negative.

CASE 170. Girl 6/12.

Sister of Case 168. Healthy.
Wassermann reaction negative.

C. CASE 177. Girl Aged 14.

Thin girl with small head. Infantile paralysis of right arm and leg. Epileptic and mentally defective. Talkative and restless.
4 of family. 2 dead both still-born.
Father drunkard and not living with the family.
Girl healthy till 8 months when took infantile paralysis. Had a bad fall at 7 years from which the fits were said to date.
Wassermann reaction negative.

CASE 48. F. Aged 38.

Mother of M.D. girl aged 13 whose wassermann was not done. Subject to Bronchitis. Nervous and hysterical.
She has had six children and no mis-carriages.
Defective girl is 3rd. 3 dead, one in infancy, one of Diphtheria and one convulsions.
Husband a sailor and his family strong.
Wassermann reaction positive.

CASE 49. Girl Aged 15.

Daughter of above and older sister of defective girl.
Very hysterical.
Wassermann reaction positive.

WASSERMANN REACTION DOUBTFUL.

A. CASE 18. Girl Aged 12.

Pale anaemic girl with slight convergent strabismus.

High palate but teeth regular and good.

No history obtained.

Wassermann reaction doubtful.

A. CASE 4. Boy Aged 15.

A large well grown boy with a tendency to oxycephalic type of head. Had been four years in school and had made very considerable progress. Nervous, talkative, untruthful, easily influenced. His birth was normal walked and talked at about one year. Had one convulsion when teething.

Eighth child in a family of 11, 4 of whom were dead, 2 of convulsions and 2 in infancy.

Father 10 years older than mother who was 30 when boy born

Family history good.

Wassermann reaction doubtful.

CASE 34.

Mother of above Case 4 Aged 45.

Tall thin and pale. Suffered for years from varicose ulcers and had had neurasthenia several years previously.

Wassermann Reaction negative.

A. CASE 28. Boy Aged 10.

Tall thin boy.

Wassermann Reaction doubtful.

A. CASE 35. Boy Aged 11.

Big healthy boy. Very imaginative.

Born at 8 months. Walked 2 years. Talked 3 years.

Suffered from headaches.

Mental condition medium. Imaginative.

Youngest of 5 children. One died at 2 years of consumption. Others well and strong.

Mother strong.

Father drinks and has deserted family.

Wassermann Reaction doubtful.

A. CASE 73. Girl Aged 15.

Tall thin girl with receding chin and open mouth.

Instruments at birth. At three years of age got a fright was very excited during the night and next morning had lost power of right side. Had two convulsions in infancy. Walked and talked at one year. Bottle fed.

Oldest of 4 children. One died at 1½ of convulsions.

Youngest child Epileptic Imbecile.

Second child healthy.

Mother 4 years older than father, had swelling of legs during pregnancy and aged 25 at birth of girl.

Father healthy, suffered from varicose ulcers of legs which were very slow in healing.

Wassermann reaction doubtful.

B. CASE 11. Boy Aged 12.

Small thin boy with blepharitis and internal squint.
Nervous head. Palate high, teeth irregular and eroded.
Troubled with Bronchitis.
Birth normal, mother aged 33.
Walked at 5 years. Late of talking.
Did not get first teeth till after 2 years and lost them at once.
Sixth of 8 children. One died of pneumonia, others alive and well.
Father and mother alive and strong. No mis-carriages.
Family history good.
Wassermann reaction doubtful.

B. CASE 33. Boy Aged 8.

Small boy with hemiplegia affecting the right side.
Extreme restlessness.
Wassermann reaction doubtful.

B. CASE 51. Boy Aged 14.

Tall thin boy with amputation of right leg above the knee for tubercular disease. Tubercular lesion of left arm. Instruments at birth. Late of sitting up.
Walked nearly 3 years. Talked 5 years.
Discharging ears in infancy.
Youngest of 4. Others strong. No mis-carriages.
Father died of apoplexy.
Mother Epileptic, aged 33 at birth.
Wassermann reaction doubtful.

C. CASE 14. Girl Aged 15.

Big healthy girl. Slight alternating strabismus.

Epileptic, passionate, restless and talkative.

Had convulsions in infancy and fits have occurred at intervals ever since. She is the 3rd of 4 children, 2 first dead, one at 2 years of pneumonia, one at 7/12 of Gastro-enteritis.

Mother weak-minded, one of 11, 4 dead of pneumonia and one brother insane.

Father drinks. His mother died of Phthisis, his father of pneumonia, one brother died of Phthisis and Alcoholism.

Wassermann reaction doubtful.

C. CASE 138. Girl aged 14.

Epilepsy with mental deficiency.

Father killed on the railway. Home conditions poor.

Wassermann reaction doubtful.

CASE 137. F.

Mother of case 138. Hair thin and marked eczema oris

Wassermann reaction positive.

CASE 139. Girl Aged 12.

Sister of Case 138. Has notched upper incisor..

Wassermann reaction doubtful.

C. CASE 190. Girl Aged 15.

Epileptic and Mentally defective. Impulsive

Wassermann reaction doubtful.

CASE 191. Girl Aged 13.

Sister of Case 190. Healthy.

Wassermann reaction negative.

CASE 192. Girl Aged 11.

Healthy. Sister of Case 190.

Wassermann reaction positive.

CASE 193. Boy Aged 3.

Epileptic. Brother of Case 190.

Wassermann reaction negative.

CASE 194. Boy Aged 1.

Convulsions. Brother of Case 190.

Wassermann reaction negative.

CASE 189. M.

Father of Case 190.

Wassermann reaction negative.

CASE 188 F.

Mother of Case 190. 3 abortions between Cases 192 and 193.

House dirty and children venomous.

Wassermann reaction positive.

EPILEPTICS WITHOUT MENTAL DEFECT. POSITIVE CASES.

CASE 102. Girl Aged 13.

Epileptic without mental deficiency.

Her sister aged 15 is a prostitute and gave a positive Wassermann reaction when examined by Dr. Watson.

Father and Mother dead cause unknown.

Wassermann reaction positive.

CASE 193. Boy Aged 3.

Epileptic without mental deficiency.

Brother of mentally defective girl 190.

Wassermann reaction negative.

Family investigated. Sister gives + result.

CASE 134. CASE 135. Twin Boys Aged 4½.

Epilepsy without mental deficiency.

Wassermann reaction positive in both.

CASE 133. F.

Mother of Cases 134 and 135. Healthy.

Wassermann reaction positive.

CASE 136. Boy Aged 9.

Brother of Cases 134 and 135. No children between.

Healthy. Teeth normal.

Had convulsions at 1½ years.

Wassermann reaction positive.

CASE 162. Boy Aged 5.

Epileptic without mental deficiency. Fits began 2 years ago and there are several months between each. Specially severe on right side and difficulty in moving that side after fits pass off.

Father a sailor. Oldest of 3 children.

Wassermann reaction positive.

CASE 165 F. Aged 31.

Mother of Case 162. Syphilis before marriage.

Wassermann reaction positive.

CASE 163. Boy Aged 3.

Brother of Case 162. Healthy.

Wassermann reaction negative.

CASE 164. Boy Aged 2.

Brother of Case 162. Healthy.

Wassermann reaction negative.

NEGATIVE CASES.

CASE 87. Girl Aged 12.

Epileptic without mental defect.

Wassermann reaction negative.

CASE 85. M. Aged 53.

Father of Case 87. Had specific disease before marriage.

Wassermann reaction negative.

CASE 86 F. Aged 49.

Mother of Case 87. Dirty and untidy.

Two abortions after 1st child. Next pregnancy child died of convulsions.

Two other children had their serum tested on a previous occasion by Dr. Watson. Girl aged 3 typical C.S. Wassermann reaction positive. Girl 5 days with spinal meningitis. Wassermann reaction positive.

Mother's serum gave a negative Wassermann reaction.

CASE 103. Boy Aged 6½.

Epileptic without mental deficiency.

Oldest of two. Brother also epileptic. Case 104.

Wassermann reaction negative.

CASE 104. Boy 3½.

Epileptic without apparent mental deficiency.

Brother of Case 103.

Wassermann reaction negative.

CASE 106. Aged 35.

Father of Cases 103 and 104. Healthy.

Wassermann reaction negative.

CASE 105. Aged 38.

Mother of Cases 103 and 104. House small, dirty and badly ventilated.

Wassermann reaction positive.

CASE 128. Boy Aged 3½.

Epilepsy without mental deficiency.

Fits began one year ago.

Youngest of 7 the first being an abortion, the others alive and well.

Wassermann reaction negative.

CASE 127 M.

Father of Case 128. Healthy.

Wassermann reaction negative.

CASE 126. F.

Mother of Case 128. Healthy. Hair fell out after marriage.

Wassermann reaction positive.

CASE 129. Boy Aged 12.

Brother of Case 128. Healthy.

Wassermann reaction negative.

CASE 130. Boy Aged 10.

Brother of Case 128. Healthy.

Wassermann reaction negative.

CASE 131. Boy Aged 7.

Brother of Case 128. Healthy.

Wassermann reaction negative.

CASE 132. Boy Aged 5.

Brother of Case 128. Anaemic.
Wassermann reaction positive.

CASE 107. Boy Aged 14.

Epileptic without mental deficiency.

His Father was epileptic but is dead.

Wassermann reaction doubtful.

CASE 108. Aged 37.

Mother of Case 107. History of rash, sore throat, loss of hair and frequent hoarseness.

Has had 3 abortions.

House clean and tidy.

Wassermann reaction doubtful.

F. CASE 52.

Very anaemic. Mother of children of normal mental capacity.

Wassermann reaction negative.

CASE 53. Boy Aged 20.

Had lupus of nose.

Wassermann reaction negative.

CASE 54. Girl Aged 16.

Tubercular disease of wrist.

Wassermann reaction negative.

CASE 55. Boy Aged 12.

Healthy.

Wassermann reaction negative.

CASE 56. Boy Aged 10.

Healthy.

Wassermann reaction negative.

Baby sister of Case 56.

Wassermann reaction negative.

CASE 57. F.

Mother of children of normal mental capacity.

Wassermann reaction negative.

CASE 58. Girl Aged 12.

Anaemic.

Wassermann reaction negative.

CASE 59. Girl Aged 10.

Healthy.

Wassermann reaction negative.

CASE 60. Boy Aged 8.

Healthy.

Wassermann reaction negative.

CASE 81. Boy Aged 9.

Healthy.

Wassermann reaction negative.

CASE. 96. Girl Aged 11.

Healthy. Normal mental condition.

Wassermann reaction negative.

(Her sister was examined previously by Dr. Watson when a negative reaction was obtained.)

CASE 99. Boy Aged 14.

Healthy. Normal mental condition.

Wassermann reaction negative.

CASE 100. Boy Aged 3½ years.

Normal mental state. Brother of 99.

Wassermann reaction negative,

(Father and mother both gave a negative reaction examined by Dr. Watson).

CASE 101. Girl 5½.

Healthy. Normal mental condition.

Wassermann reaction negative.

(Mother and sister both gave a negative reaction examined by Dr. Watson.)

CASE 97. Girl Aged 10.

Healthy. Mental condition normal.

Wassermann reaction doubtful.

(Sister gave a negative reaction examined by Dr.Watson).

CASE 98. Girl 12 years.

Healthy. Normal mental condition.

Wassermann reaction doubtful.

(Two sisters both gave negative reactions examined by Dr. Watson.)

CASE 186. Boy Aged 4½.

Healthy. Mental condition normal.

Wassermann reaction negative.

CASE 187. Boy.

Brother of 186. Normal.

Wassermann reaction negative.

CASE 185. M.

Father of 186 and 187.

Wassermann reaction negative.

CASE 198. Boy Aged 15.

Healthy. Mental condition normal.

Wassermann reaction negative.

CASE 199. Girl Aged 13.

Healthy. Sister of 198.

Wassermann reaction negative.

CASE 200. Girl 10.

Healthy. Sister of 198.

Wassermann reaction negative.

CASE 201. Boy Aged 7.

Healthy. Brother of 198.

Wassermann reaction negative.

CASE 202. Girl Aged 6.

Healthy. Sister of 198.

Wassermann reaction negative.

CASE 197. Boy Aged 4.

Normal mental condition Anaemic.

Wassermann reaction negative.

CASE 196. M.

Father of 198.

Wassermann reaction negative.

CASE 195. F.

Mother of 198.

Wassermann reaction negative.

CASE 114. Girl Aged 9.

Normal mental condition.

Pale and anaemic with notching of incisor.

Lencorrhoea.

4th of 10 children. 5 dead. Oldest 16, healthy. 2nd Girl 12 healthy. 3rd boy died of "Prematurity". 4th Case 114, 5th died of Whooping-cough at 1-9/12 years. 6th and 7th twins born at 8 months lived 2 weeks.

8th and 9th twins, one died of Meningitis 11/12, other healthy. 10th Girl Aged 2, healthy.

Father apparently healthy.

Wassermann reaction positive.

CASE 113. F. Aged 36.

Mother of Case 114. Has had specific disease.

Wassermann reaction positive.

T A B L E I.

		1.	2.	3.	4.	5.	6.	Serum Controls 7.	8.	Results.
Complement Dose.		·012.	·024	·035.	·05	·07	·1	·01.		
Case 1	(L	0	ft. tr.	tr.	tr.					
	(L.C.	–	0.	0.	0.	ft. tr.	V.M.	C.		+
Case 2	(L	0	0	ft. tr.	tr.					
	(L.C.	–	0	0	0	0	J.C.	J.C.		+
Case 3	(L.	0	ft. tr.	tr.	Dist.					
	(L.C.	–	0	0	ft. tr.	tr.	Dist.	C.		+
Case 4	(L.	tr.	a.c.	C.						
	(L.C.	–	m.	J.C.				J.C.		?
Case 5	(L.	0	o	0	ft. tr.					
	(L.C.	–	o	0	0	0	tr.	J.C.		+
Case 6	(L.	J.C.	J.C.	C.						
	(L.C.	–	A.C.	J.C.				J.C.		—
Case 7	(L.	M.	V.M.	C.						
	(L.C.	–	J.C.	C.				J.C.		—
Case 8	(L.	0	0.	0.	0					
	(L.C.	–	0	0.	0	0	0.	Dist.	V.M.	+
Case 9	(L.	0	0	0	ft. tr.					
	(L.C.	–	0	0	0	ft. tr.	M.	A.C.	C.	+
Control	(L.	0	ft. tr.	Dist.	V.M.					
	(L.C.	–	tr.	Dist.	Dist.	C.		C.		
Control	(L.	C.								
	(L.C.	–	C.							
Emul-	(L.	O.M.	J.C.					C.	1.	4.
sions	(L.C.	J.C.	C.					·012	·02.	·03 ·04
								tr.	V.M.	A.C. A.C.

---

ft. tr. = faint trace
V.M = very marked
Dist = Distinct
a.c. = almost complete
J.C. = Just complete
C = Complete

TABLE II.

	1.	2.	3.	4.	5.	6.	Serum Controls 7.	8.	Results.
Complement dose.	·012	·023	·035.	·05	·07	·1	·01	·02.	
Case 10 (L.	V.M.	C.							
(L.C.	–	tr.	v.m.	c.			J.C.		+
Case 11 (L.	V.M.	J.C.							
(L.C.	–	Dist.	C.				J.C.		?
Case 12 (L.	M.	J.C.	J.C.	C					
(L.C.	–	tr.	A.C.	C			V.M.	C.	+ (weak).
Case 13 (L.	tr.	a.c.	C.						
(L.C.	–	0	M.	J.C.	C.		V.M.	C.	+
Case 14 (L.	tr.	a.c.	C						
(L.C.	–	tr.	J.C.	J.C.	C.		tr.	A.C.	?
Case 15 (L.	V.M.	J.C.	C.						
(L.C.	–	V.M.	C.				V.M.	J.C.	–
Case 16 (L.	V.M.	J.C.							
(L.C.	–	M.	C.				M.C.	C.	—
Case 17 (L.	a.c.	C.							
(L.C.	–	J.C.					J.C.		—
Case 18 (L.	Dist.	J.C.		J.C.					
(L.C.	–	tr.	J.C.		C.		V.M.	C.	?
Case 19 (L.	V.M.	J.C.							
(L.C.	–	0	M.	C.			V.M.	J.C.	+
+ Control (L.	a.c.	C.							
(L.C.	–	Dist.	V.M.	C.			J.C.		
– Control (L.	a.c.	C.					C. 1, 4.		
(L.C.	–	C.							
							·02	·03	·04 ·05
Emul- (L.	a.c.	C.							
sions (L.C.	J.C.	J.C.					A.C.	A.C.	J.C. J.C.

TABLE III.

		1.	2.	3.	4.	5.	6.	Serum Controls 7.	8.	Results
Complement Dose.		·012	·024	·035	·05	·07.	·1	·01	·02.	
+ Control.		v.m.	j.c.	m.	v.m.	c.		a.c.	c.	
		–	tr.							
Case 20.	(L.	m.	a.c.	c.						
	(L.C.	–	v.m.	j.c.	c.			v.m.	c.	+(weak)
Case 21	(L.	v.m.	c.							
	(L.C.	–	tr.	v.m.	j.c.	c.		a.c.	c.	+
Case 22	(L.	0	m.	o.m.	c.					
	(L.C.	–	0	0	0	tr.	a.c.	v.m.	c.	+
Case 23	(L.	0	tr.	m.	c.					
	(L.C.	–	0	dist.	v.m.	j.c.	c.	v.m.	c.	+
Case 24	(L.	0	0	0	tr.					
	(L.C.	–	0	0	0	0	0	v.m.	a.c.	+
Case 25	(L.	0	ft. tr.	tr.	v.m.					
	(L.C.	–	0	0	0	tr.	dist.	m.	j.c.	+
Case 26	(L.	0	0	0	ft. tr.					
	(L.C.	–	0	0	0	0	0.	v.m.	c.	+
Case 27	(L.	v.m.	j.c.							
	(L.C.	–	v.m.	c.				v.m.	c.	—
Case 28	(L.	Dist.	j.c.							
	(L.C.	–	dist.	j.c.	c.			v.m.	c.	?
Case 29	(L.	v.m.	c.							
	(L.C.	–	m.	v.m.	j.c.	c.		a.c.	c.	+
Case 30	(L.	v.m.	j.c.							
	(L.C.	–	tr.	a.c.	j.c.	c.		v.m.	c.	+
Case 31	(L.	v.m.	c.							
	(L.C.	–	m.	c.				v.m.	c.	—
– Control	(L.	v.m.	c.			c.1:4				
	(L.C.	m.	c.							
						·02	·032	·04	·05	
Emul-	(L.	j.c.	c.							
sions	(L.C.	j.c.	c.			a.c.	j.c.			

T A B L E IV.

		1.	2.	3.	4.	5.	6.	Serum Controls 7.	8.	Results.
Complement Dose.		·01	·02	·03	·045	·06	·085	·01	·02	
Case 32	(L.	v.m.	j.c.					a.c.	c.	—
	(L.C.	-	m.	j.c.						
Case 33	(L.	dist.	v.m.	c.				v.m.	c.	?
	(L.C.	-	tr.	a.c.	j.c.					
Case 34	(L.	tr.	a.c.	c.				tr.	c.	—
	(L.C.	-	v.m.+	j.c.						
Case 35	(L.	dist.	a.c.					j.c.		?
	(L.C.	-	m.	j.c.	j.c.					
Case 36	(L.	tr.	j.c.					v.m.	c.	+
	(L.C.	-	ft. tr.	v.m.	j.c.					
Case 37	(L.	m.	j.c.					v.m.	c.	—
	(L.C.	-	dist.	a.c.	c.					
Case 38	(L.	v.m.	c.					v.m.+	c.	—
	(L.C.	-	dist.	j.c.						
Case 39	(L.	dist.	v.m.	c.						
	(L.C.	-	a.c.	j.c.				dist.	c.	—
Case 40	(L.	v.m.	j.c.							
	(L.C.	-	m.	j.c.				v.m.	c.	—
Case 41	(L.	tr.	a.c.	c.	a.c.	c.				
	(L.C.	-	tr.	m.				v.m.	c.	+
Case 42	(L.	tr.	c.							
	(L.C.	-	j.c.					dist.	c.	—
Case 43.	(L.	dist.	a.c.	c.						
	(L.C.	-	v.m.	j.c.				dist.	c.	—
+ Control	(L.	ft. tr.	dist.	a.c.	c.					
	(L.C.	-	0	tr.	m.	c.		a.c.	c.	
− Control	(L.	m.	j.c.							
	(L.C.	-	v.m.	c.				j.c.		
Emul-	(L.	v.m.	a.c.	c.						
sions	(L.C.	a.c.	a.c.	j.c.						

C.1: 4.

·02	·03	·04
a.c.	j.c.	c

T A B L E V.

		1.	2.	3.	4.	5.	6.	Serum Controls 7.	8.	Results.
Complement Dose.		·012	·024	·035	·05	·07	·1	·01	·02	
Case 44	(L.	a.c.	c.							
	(L.C.	-	j.c.					j.c.	c.	—
Positive Control	(L.	tr.	v.m.	a.c.	c.					
	(L.C.	-	0	0	tr.	v.m.	a.c.	j.c.		+
— Control	(L.	w.m.+	c.							
	(L.C.	-	j.c.					j.c.		
Emulsions	(L.	v.m.	a.c.	j.c.						
	(L.C.	ft. tr.	a.c.	j.c.			C l :4			
						·01	·02	·03		·04
						tr.	m.	v.m.		v.m.+

T A B L E VI.

		1.	2.	3.	4.	5.	6.	Serum controls 7.	8.	Results.
Complement Dose.		·012	·024	·034	·046	·065	·09	·01	·02	
Case 45	(L.	a.c.	j.c.							
	(L.C.	-	j.c.	c.				j.c.		—
Case 46	(L.	a.c.	j.c.							
	(L.C.	-	j.c.					c.		—
Case 47	(L.	j.c.								
	(L.C.	-	j.c.					c.		—
Case 48	(L.	v.m.	j.c.							
	(L.C.	-	tr.	v.m.	a.c.	j.c.		a.c.	c	+
Case 49	(L.	a.c.	j.c.							
	(L.C.	-	dist.	v.m.	j.c.	c.		a.c.	c.	+
Case 50	(L.	0	0	0	0					
	(L.C.	-	0	0	0	0	0	c.		+
Case 51	(L.	a.c.	c.							
	(L.C.	-	v.m.	j.c.				a.c.	c.	?
+ Control	(L.	tr.	dist.	o.m.	j.c.					
	(L,C.	-	0	0.	0	tr.	j.c.	a.c.	c.	
— Control	(L.	a.c.	c.							
	(L.C.	a.c.	c.					j.c.	c.	
Emul-	(L.	a.c.	c.							
sions	(L.C.	tr.	c.					c. 1 : 4.		

	·02	·03	·04	·05
	a.c.			

T A B L E VII.

		1.	2.	3.	4.	5.	6.	Serum Control 7.	Serum Control 8.	
Complement Dose.		·011.	·023	·04	·058	·075	·11	·01	·02.	Results.
Case 52	(L.	v.m.	c							
	(L.C.	-	j.c.					c.		—
Case 53	(L.	a.c.	j.c.							
	(L.C.	-	c.					c.		—
Case 54	(L.	v.m.	c							
	(L.C.	-	j.c.					c.		—
Case 55	(L.	j.c.								
	(L.C.	-	j.c.					c.		—
Case 56	(L.	a.c.	c.							
	(L.C.	-	j.c.					c.		—
Case 57	(L.	v.m.	j.c.							
	(L.C.	-	a.c.					c.		—
Case 58	(L.	j.c.								
	(L.C.	-	c.					c.		—
Case 59	(L.	a.c.								
	(L.C.	-	c.					c.		—
Case 60	(L.	c								—
	(L.C.	-	c.					c.		
Control	(L.	o	tr.	m.	a.c.					
	(L.C.	-	0	0	tr.	m.		c.		

T A B L E VIII.

		1.	2.	3.	4.	5.	6.	Serum Controls. 7.	8	
Complement Dose.		·012	·024	·035	·05	·065	·09	·01	·02.	Results.
Case 61	(L.	j.c.								
	(L.C.	–	v.m.	j.c.				c.		—
Case 62	(L.	j.c.								
	(L.C.	–	v.m.	c.				c.		—
Case 63	(L.	c.	~~j.c.~~							
	(L.C.	–	j.c.	c.				c.		—
Case 64	(L.	v.m.	j.c.							
	(L.C.	–	0	ft. tr.		dist.	j.c.	c.		+
Case 65	(L.	tr.	v.m.	dist.		c.				
	(L.C.	–	0	0		tr.	dist.a.c.	c.		+
Case 66	(L.	c.								
	(L.C.	–	c.							—
Case 67	(L.	v.m.	c.							
	(L.C.	–	ft. tr.	v.m.		c.		c.		+
Case 68	(L.	a.c.	c.							
	(L.C.	–	ft. tr.	v.m.		c.		c.		+
Case 69	(L.	j.c.	c.							
	(L.C.	–	dist.	c.				c.		?
Case 70	(L.	j.c.								
	(L.C.	–	m.	c.				c.		?
Case 71	(L.	0.	dist.	v.m.		c.				
	(L.C.	–	0	0	ft. tr.		tr. a.c.	c.		+
Case 72	(L.	tr.	tr.	dist.		c.				
	(L.C.	–	0	0	0	ft. tr.	c.	c.		+

T A B L E VIII (CONTINUED)

		1.	2.	3.	4.	5.	6.	7.	8.	Results
Case 73.	(L.	j.c.	v.m.	c.						
	(L.C.	-	tr.	c.				j.c.		?
Case 74	(L.	m.	a.c.	j.c.						
	(L.C.	-	o.	0	0	ft. tr.	tr.	c.		+
Case 75.	(L.	0	a.c.	c.						
	(L.C.	-	m.	v.m.	c.			c.		+
Case 76	(L.	0	0	0						
	(L.C.	-	0		ft. tr.	0.	ft. tr.	c.		+
Case 77	(L.	0	ft. tr.	0 tr.	tr.					
	(L.C.	-	0	0	0	ft. tr.	ft. tr.	c.		+
Case 78	(L.	0	0	ft. tr.	tr.					
	(L.C.	-	0	0	0	0	ft. tr.	c.		+
Case 79	(L.	j.c.	c.							
	(L.C.	-	c.					c.		-
Case 80	(L.	tr.	dist.	m	c.					
	(L.C.	-	0.	0	tr.	tr.	tr.	c.		+
+ Control	(L.	0	tr.	a.c.	j.c.					
	(L.C.	-	0.	0	ft. tr.	m.	j.c.	c.		
- Control	(L	c.								
	(L.C.	-	c.					c.		
Emul-	(L.	j.c.	c.							
sions	(L.C.	a.c.	j.c.							

Complement 1 : 4

·02 ·03 ·04 ·05

C

TABLE IX.

	1.	2.	3.	4.	5.	6.	Emulsion Control. 7.	8.	Results
Complement Dose.	·012	·024	·034	·05	·065	·095	·01	·02	
Case 81 (L.	v.m.	a.c.	j.c.						
(L.C.	-	a.c.	j.c.				c.		—
Case 82 (L.	tr.	v.m.	a.c.	j.c.					
(L.C.	-	tr.	v.m.	v.m.	c.		c.		+
Case 83 (L.	0	ft. tr.	ft. tr.	m.					
(L.C.	-	0	0	dist.	v.m.	c.	c.		+
Case 84 (L.	a.c.	c.							
(L.C.	-	c.					c.		—
Case 85 (L.	m.	v.m.	a.c.	a.c.					
(L.C.	-	a.c.	c.				c.		—
Case 86 (L.	tr.	a.c.	a.c.	j.c.					
(L.C.	-	a.c.	a.c.	j.c.			j.c.		—
Case 87 (L.	a.c.	c.							
(L.C.	-	j.c.					c.		—
Case 88 (L.	m.	a.c.	c.						
(L.C.	-	j.c.	c.				c.		—
Case 89 (L.	ft. tr.	ft. tr.	dist.	v.m.					
(L.C.	-	ft. tr.	ft.tr.	m.	c.		c.		+
Case 90 (L.	Dist.	0	tr.	m.					
(L.C.	-	ft. tr.	tr.	m.	v.m.	j.c.	c.		+
Case 91 (L.	tr.	tr.	tr.	m.					
(L.C.	-	ft. tr.	ft.tr.	dist.	a.c.	j.c.	c.		+
Case 92 (L.	0	tr.	tr.	dist.					
(L.C.	-	0	0	tr.	tr.	tr.	v.m.		+
Case 93 (L.	v.m.	v.m.	a.c.	j.c.					
(L.C.	-	a.c.	a.c.	c.			a.c.	c.	—
Case 94 (L.	Dist.	m.	a.c.	a.c.					
(L.C.	-	m.	v.m.	j.c.			a.c.	c.	?

T A B L E IX (CONTINUED)

		1.	2.	3.	4.	5.	6.	7.	8.	Results
Case 95	(L.	m.	v.m.	a.c.	j.c.					
	(L.C.	-	a.c.	a.c.	c.			c.		?
Case 96	(L.	a.c.	j.c.							
	(L.C.	-	j.c.					c.		—
Case 97	(L.	tr.	dist.	m.	a.c.					
	(L.C.	-	dist.	m.	a.c.	j.c.		v.m.	j.c.	?
Case 98	(L.	tr.	m.	v.m.	a.c.					
	(L.C.	-	dist.	m.	v.m.	c.		a.c.	j.c.	?
Case 99	(L.	0	v.m.	j.c.						
	(L.C.	-	a.c.	j.c.				j.c.		—
Case 100	(L.	m.	v.m.	j.c.						
	(L.C.	-	j.c.	j.c.				a.c.	j.c.	—
Case 101	(L.	dist.	m.	v.m.	a.c.					
	(L.C.	-	a.c.	j.c.				a.c.	j.c.	—
Case 102	(L.	v.m.	a.c.							
	(L.C.	-	v.m.	v.m.	a.c.	c.		j.c.		+ (weak).
Case 103	(L.	v.m.	a.c.	j.c.						
	(L.C.	-	v.m.+	j.c.				j.c.		—
Case 104	(L.	v.m.	j.c.							
	(L.C.	-	c.					c.		—
Case 105	(L.	tr.	v.m.	a.c.	j.c.					
	(L.C.	-	0	ft.tr.	m.	c.		c.		+
Case 106	(L.	a.c.	a.c.	c.						
	(L.C.	-	c.					c.		—
Case 107	(L.	dist.	m.	v.m.	a.c.					
	(L.C.	-	v.m.	j.c.				c.		?
Case 108	(L.	v.m.	a.c.	c.						
	(L.C.	-	v.m.	a.c.	j.c.			c.		?

T A B L E IX (CONTINUED).

		1.	2.	3.	4.	5.	6.	7.	8.	Results
Case 109	(L.	j.c.								
	(L.C.	–	c.					c.		—
Case 110	(L.	a.c.	c.							
	(L.C.	–	c.					c.		—
Case 111	(L.	j.c.								
	(L.C.	–	j.c.					c.		—
Case 112	(L.	j.c.								
	(L.C.	–	c.					c.		—
+ Control	(L.	0	ft.	tr.	tr.	dist.				
	(L.C.	–	0	0	ft.tr.	m.	a.c.	c.		
— Control	(L.	v.m.	a.c.	j.c.						
	(L.C.	–	a.c.	j.c.				c.		
Emul-	(L.	j.c.								
sions	(L.C.	j.c.				C. 1 : 4				
						·016	·024	·032	·04	
								j.c.		

T A B L E X.

Complement Dose		1. ·01	2. ·02	3. ·032	4. ·046	5. ·066	6. ·09	Serum Controls 7. ·01	8. ·02	Results.
Case 116	(L.	0	m.	j.c.						
	(L.C.	-	o.	tr.	m.	v.m.	j.c.	c.		+
Case 117	(L.	0	0	a.c.	c.					
	(L.C.	-	0	ft.tr.	dist.	c.		c.		+
Case 118	(L.	0	tr.	dist.	j.c.					
	(L.C.	-	0.	0	0	0	tr.	c,		+
Case 115	(L.	0	tr.	dist.	c.					
	(L.C.	-	0.	0	dist.	m.	a.c.	a.c.	c.	+
Case 119	(L.	0	0	m.	c.					
	(L.C.	-	0	0	0	tr.	dist.	c.		+
Case 113	(L.	tr.	dist.	c.						
	(L.C.	-	0	0	tr.	v.m.	c.	c.		+
Case 114	(L.	tr.	a.c.	c.						
	(L.C.	-	0	0	m.	v.m.	c.	c.		+
Case 120	(L.	v.m.	c.							
	(L.C.	-	j.c.	c.				c.		—
Case 121	(L.	v.m.	j.c.							
	(L.C.	-	a.c.	c.				c.		—
Case 122	(L.	a.c.	c.							
	(L.C.	-	j.c.					c.		—
Case 123	(L.	a.c.	j.c.							
	(L.C.	-	j.c.					c.		—
Case 124	(L.	j.c.								
	(L.C.	-	j.c.					c.		—
Case 125	(L.	dist.	m.	j.c.						
	(L.C.	-	dist.	m.	v.m.	a.c.	a.c.	v.m.	j.c.	+

T A B L E X. (CONTINUED)

		1.	2.	3.	4.	5.	6.	7.	8.	Results
Case 126	(L.	m.	j.c.							
	(L.C.	-	broken	v.m.	a.c.	j.c.		c.		+
Case 127	(L.	a.c.	c.							
	(L.C.	-	a.c.	c.				a.c.	c.	—
Case 128	(L.	c.								
	(L.C.	-	j.c.					c.		—
Case 129	(L.	j.c.								
	(L.C.	-	c.					c.		—
Case 130	(L.	c.								
	(L.C.	-	c.					c.		—
Case 131	(L.	a.c.	c.							
	(L.C.	-	c.					c.		—
Case 132	(L.	tr.	m.	a.c.	c.					
	(L.C.	-	0	tr.	dist.	v.m.	j.c.	a.c.	c.	+
Case 133	(L.	tr.	c.							
	(L.C.	-	tr.	dist.	m.	j.c.	c.	c.		+
Case 134	(L.	0	v.m.	j.c.						
	(L.c.	-	0	dist.	m.	a.c.	c	c.		+
Case 135	(L.	tr.	a.c.	c.						
	(L.C.	-	ft.tr.	dist.	m.	a.c.	j.c.	c.		+
Case 136	(L.	0	dist.	a.c.	c.					
	(L.C.	-	0	dist.	v.m.	v.m.	a.c.	c.		+
Case 137	(L.	dist.	v.m.	c.						
	(L.C.	-	m.	v.m.	a.c.	a.c.	c.	c.		+
Case 138	(L.	m.	v.m.	a.c.	j.c.					
	(L.C.	-	v.m.	a.c.	j.c.			c.		?
Case 139	(L.	m.	a.c.	c.						
	(L.C.	-	v.m.	c.c.	j.c.			c.		?

T A B L E X. (CONTINUED).

		1.	2.	3.	4.	5.	6.	7.	8.	Results
Case 140	(L.	0	ft.tr.	dist.	a.c.					
	(L.C.	-	0	0	0	m.	j.c.	c.		+
Case 141	(L.	0	tr.	dist.	m.					
	(L.C.	-	0	0	0	dist.	m.	v.m.	j.c.	+
Case 142	(L.	0	tr.	dist.	m.					
	(L.C.	-	0	0	tr.	m.	v.m.	c.		+
Case 143	(L.	0	tr.	m.	a.c.					
	(L.C.	-	0.	0	dist.	m.	v.m.	j.c.		+
Case 144	(L.	c.								
	(L.C.	-	c.					c.		—
Case 145	(L.	a.c.	c.							
	(L.C.	-	c.					c.		—
Case 146	(L.	m.	v.m.	j.c.						
	(L.C.	-	dist.	v.m.	a.c.	j.c.		a.c.	c.	+
Case 147	(L.	c.								
	(L.C.	-	c.					c.		—
Case 148	(L.	v.m.+	j.c.							
	(L.C.	-	a.c.	c.				c.		—
Case 149	(L.	0	0	tr.	dist.					
	(L.C.	-	0	0	dist.	m.	a.c.	v.m.	a.c.	+
+ Control	(L.	ft.tr.	tr.	v.m.	a.c.					
	(L.C.	-	ft.tr.	tr.	dist.	a.c.	c.	c.		
— Control	(L.	c.								
	(L.C.	-	c.					c.		
Emul-	(L.	v.m.	j.c.	c.						
sions	(L.C.	m.	v.m.	a.c.						

C. 1 : 4.

·01 ·022 ·032 ·04

a.c. c+

T A B L E XI.

Complement Dose.		·012	·024	·034	·05	·07	·1	Serum Controls ·01	·02	Results.
Case 150	(L.	0	m.	c.						
	(L.C.	–	0	tr.	dist.	v.m.	c.	v.m.	c.	+
Case 151	(L.	0	tr.	j.c.						
	(L.C.	–	0	dist.	m.	a.c.	c.	a.c.	c	+
Case 152	(L.	0	tr.	m.	c.					
	(L.C.	–	0	ft. tr.	tr.	v.m.	c.	tr.	j.c.	+
Case 153	(L.	0	ft.tr.	a.c.	c.					
	(L.C.	–	0	dist.	m.	a.c.	a.c.	c.		+
Case 154	(L.	0	tr.	a.c.	c.					
	(L.C.	–	0	tr.	dist.	m.	a.c.	a.c.	c.	+
Case 155	(L.	a.c.	c.							
	(L.C.	–	j.c.					c.		–
Case 156	(L.	m.	c.							
	(L.C.	–	m.	j.c.				c.		–
Case 157	(L.	0	dist.	j.c.						
	(L.C.	–	tr.	dist.	m.	a.c.	c.	c.		+
Case 158	(L.	v.ft.tr.	c.							
	(L.C.	–	c.					c.		–
Case 159	(L.	0	ft.tr.	j.c.						
	(L.C.	–	0	tr.	m.	v.m.	c.	j.c.		+
Case 160	(L.	0	0	v.ft.tr.	j.c.					
	(L.C.	–	0	0	0	v.ft.tr.	tr.	a.c.	c.	+
Case 161	(L.	0	0	v.ft.tr.	c.					
	(L.C.	–	0	0	0	v.ft.tr.	dist.	c.		+
Case 162	(L.	0	0	tr.	j.c.					
	(L.C.	–	0	0	v.ft.tr.	dist.	v.m.	c		+
Case 163	(L.	m.	j.c.	a.c.						
	(L.C.	–	c.					c		–

T A B L E XI (CONTINUED).

		1.	2.	3.	4.	5.	6.	7.	8.	Results
Case 164	(L.	m.	a.c.	j.c.						
	(L.C.	-	a.c.	j.c.				c.		—
Case 165	(L.	0	dist.	j.c.						
	(L.C.	-	0	0	dist.	m.	c.	c.		+
Case 166	(L.	0	0	a.c.	c.					
	(L.C.	-	0	0	tr.	m.	v.m.	c.		+
Case 167	(L.	dist.	j.c.							
	(L.C.	-	m.	c.				c.		—
Case 168	(L.	a.c.	c.							
	(L.C.	-	j.c.					a.c.	c.	—
Case 169	(L.	v.m.	j.c.							
	(L.C.	-	j.c.					a.c.	c.	—
Case 170	(L.	v.m.	c.							
	(L.C.	-	j.c.					c.		—
Case 171	(L.	c.								
	(L.C.	-	c.					c.		—
Case 172	(L.	j.c.								
	(L.C.	-	j.c.					c.		—
Case 173	(L.	m.	j.c.							
	(L.C.	-	c.					c.		—
Case 174	(L.	j.c.								
	(L.C.	-	c.					c.		—
Case 175	(L.	a.c.	c							
	(L.C.	-	c.					c.		—
Case 176	(L.	j.c.								
	(L.C.	-	j.c.					j.c.		—
Case 177	(L.	c.								
	(L.C.	-	c.					c.		—

T A B L E XI (CONTINUED).

		1.	2.	3.	4.	5.	6.	7.	8.	Results.
Case 178	(L.	tr.	v.m.	j.c.						
	(L.C.	-	a.c.	c.				c.		—
Case 179	(L.	tr.	a.c.	j.c.						
	(L.C.	-	a.c.	c.				c.		—
Case 180	(L.	v.ft.tr.	m.	j.c.						
	(L.C.	-	m.	v.m.	a.c.	c.		c.		+
Case 181	(L.	j.c.								
	(L.C.	-	c.					c.		—
Case 182	(L.	a.c.	c.							
	(L.C.	-	c.					c.		—
Case 183	(L.	v.m.	c.							
	(L.C.	-	c.					c.		—
Case 184	(L.	0	0	ft.tr.	v.m.					
	(L.C.	-	0	0	tr.	m.	v.m.		c.	+
Negative Control	(L	c.								
	(L.C.	-	c.					c.		
Emul-sions	(L.	c.								
	(L.C.	c.				C 1 : 4.				
					·02	·03	·04	·05.		
					a.c.	c.				

T A B L E XII.

		1.	2.	3.	4.	5.	6.	Serum Controls 7.	8.	
Complement Dose.		·012	·024	·036.	·05.	·068	·098	·01	·02	Results.
Case 185	(L.	c.								
	(L.C.	–	j.c.					c.		–
Case 186	(L.	–	c.							
	(L.C.	–	c.					Broken	c.	–
Case 187	(L.	m.	c.							
	(L.C.	–	tr.	tr.	m.	a.c.	c.	c.		+
Case 188	(L.	0	0	0	tr.					
	(L.C.	–	0	0	0	ft.tr.	ft.tr.	c.		+
Case 189	(L.	a.c.	c.							
	(L.C.	–	a.c.	j.c.				c.		–
Case 190	(L.	m.	c.							
	(L.C.	–	m.	a.c.	j.c.			j.c.		?
Case 191	(L.	dist.	m.	c.						
	(L.C.	–	m.	c.				c.		–
Case 192	(L.	0	0	c.						
	(L.C.	–	v.ft.tr.	tr.	dist.	j.c.		c.		+
Case 193	(L.	m.	c.							
	(L.C.	–	j.c.					c.		–
Case 194	(L.	c.								
	(L.C.	–	j.c.					c.		–
Case 195	(L.	c.								
	(L.C.	–	c.					j.c.		–
Case 196	(L.	m.	c.							
	(L.C.	–	j.c.					c.		–
Case 197	(L.	a.c.	c.							
	(L.C.	–	j.c.					c.		–

T A B L E XII (CONTINUED)

		1.	2.	3.	4.	5.	6.	7.	8.	Results.
Case 198	(L.	m.	c.							
	(L.C.	-	j.c.					c		—
Case 199	(L.	a.c.	c.							
	(L.C.	-	j.c.					c		—
Case 200	(L.	a.c.	j.c.							
	(L.C.	-	v.m.	c.				c		—
Case 201	(L.	c.								
	(L.C.	-	j.c.					c		—
Case 202	(L.	a.c.	j.c.							
	(L.C.	-	j.c.	c.				c.		—
Case 203	(L.	0	0	0	tr.					
	(L.C.	-	0	0	0	ft.tr.	tr.	m.	j.c.	+
Case 204	(L.	0	0	0	c.					
	(L.C.	-	0	0	0	0	tr.	v.m.	c.	+
Positive Control	(L.	0	0	tr.	a.c.					
	(L.C.	-	0	0	0	tr.	a.c.	c.		
Negative Control	(L.	c.								
	(L.C.	-	c.					c.		
Emul-sions	(L.	c.								
	(L.C.	j.c.								

C 1 : 4.

·02	·03	·04	·05
a.c.	c.		

T A B L E XIII.

Complement Dose.		1. ·012	2. ·024	3. ·036	4. ·05	5. ·07	6. ·1	Serum Control 7. ·01	Serum Control 8. ·02	Results.
Case 205	(L.	j.c.	c.							
	(L.C.	–	c.					c.		–
Case 206	(L.	0	0	ft.tr.	ft.tr.					
	(L.C.	–	0	0	0	m.	c.	j.c.		+
Case 207	(L.	j.c.	c.							
	(L.C.	–	c.					c.		–
Case 208	(L.	0	0	dist.	v.m.					
	(L.C.	–	ft.tr.	tr.	a.c.	c.		c.		+
Case 209	(L.	0	0	tr.	a.c.					
	(L.C.	–	0	tr.	v.m.	c.		j.c.		+
Case 210	(L.	v.m.	j.c.							
	(L.C.	–	j.c.					c.		–
Case 211	(L.	v.m.	c.							
	(L.C.	–	j.c.					c.		–
Case 212	(L.	m.	c.							
	(L.C.	–	c.					c.		–
Case 213	(L.	j.c.								
	(L.C.	–	c.					c.		–
Case 214	(L.	a.c.	c.							
	(L.C.	–	j.c.					c.		–
Positive Control	(L.	0	tr.	m.	m.					
	(L.C.	–	0	0	tr.			j.c.		+
Negative Control	(L.	m.	c.							
	(L.C.	–	c.			c.				
Emulsions	(L.	dist.	c.							
	(L.C.	dist.	c.							

C 1 : 4.

·01	·02	·03	·04
0	tr.	m.	c.

T A B L E XIV.

								Serum Control.		
		1.	2.	3.	4.	5.	6.	7.	8.	Results.
Complement Dose.		·012	·024	·038	·055	·075	·11.	·01	·02	+
Case 215	(L.	ft.tr.	tr.	v.m.	j.c.					
	(L.C.	-	0	ft.tr.	tr.	a.c.	c.	a.c.	c.	+
Case 216	(L.	tr.	dist.	j.c.	c.					
	(L.C.	-	0	a.c.	a.c.	j.c.	c.	a.c.	c.	+
Case 217	(L.	0	0	0	m.					
	(L.C.	-	0	0	0	tr.	c.	c.		+
Case 218	(L.	ft.tr.	m	c.	c.					
	(L.C.	-	0	dist.	a.c.	a.c.	c.	c		+
Case 219	(L.	0	tr.	dist.	c.					
	(L.C.	-	0	tr.	a.c.	c		c.		+
Case 220	(L.	0	tr.	v.m.	c.					
	(L.C.	-	0	0	dist.	c.		j.c.	c.	+
Case 221	(L.	v.m.	c	c.	c.					
	(L.C.	-	ft.tr.	v.m.	c.			c.		+
Case 222	(L.	tr.	tr.	c.	c.					
	(L.C.	-	0	0	m.	c.		a.c.	c.	+
Negative Control	(L.	c.								
	(L.C.	-	j.c.					a.c.	c.	+
Emul-sions	(L.	tr.	c							
	(L.C.	c.					C 1 : 4. ·013 a.c.	·02 c.		

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