distinct from arterial $p\text{CO}_2$ rise (Westlake *et al.*, 1955). The work of Swanson, Stavney, and Plum (1958) bears on this point in support of our findings, as they have shown experimentally that acute changes in arterial $p\text{H}$ affect the cats' E.E.G. only when there is an accompanying rise or fall in blood CO_2 tension.

It has been claimed by Wilson and Sieker (1958) that age is important in determining E.E.G. dysrhythmia in respiratory insufficiency, but our results do not demonstrate this. Failure to observe any change in the E.E.G. from inhalation of oxygen during recording is in agreement with similar lack of effect mentioned by others (Brazier quoted by Austen *et al.*, 1957; Wilson and Sieker, 1958).

The immediate causal mechanism of E.E.G. disorder from impaired respiratory function presumably lies in hypercapnia and hypoxia, and our data do not allow choice of which is the more important factor. Though there may be more remote metabolic changes, such as alteration in intracellular electrolyte concentrations, which have an effect on cerebral neuronal activity, our observations, unlike those of Wilson and Sieker (1958), do not disclose any consistent abnormality of serum electrolytes in relation to E.E.G. appearances. This fact must, however, be treated with reserve, as it is generally appreciated that the electrolyte levels of the circulating blood, and in particular potassium, are not a reliable index of ionic levels within tissue cells.

Lastly, we would suggest that the variability of development of E.E.G. disorder in patients with respiratory insufficiency must predicate a great range in individual cerebral homeostasis in the face of alterations of blood gas levels. In some instances the tolerance of the central nervous system against disturbance by this means must be high and in others relatively low, and the unpredictable behaviour of neural and mental signs and symptoms, *vis à vis* $p\text{O}_2$ and $p\text{CO}_2$ values, can be interpreted in the same light. Our studies satisfy us that there is a close interrelation between neurological semeiology clinically and the presence of E.E.G. dysrhythmia—a closer nexus than exists between either of these phenomena and the arterial oxygen and carbon dioxide tensions.

Summary

Sixteen patients have been studied during 18 hospital admissions for acute or subacute respiratory failure. The E.E.G., clinical signs and symptoms in the nervous system, and blood gas findings have been compared.

The E.E.G. shows symmetrical changes from intermittent theta activity to paroxysmal delta discharge of high amplitude, and the changes resemble those found in other types of confusional state, particularly those of metabolic origin.

There is good correlation between the E.E.G. abnormality and the clinical neurological findings. There is also a correlation between increasing E.E.G. abnormality and a falling $p\text{O}_2$ and rising $p\text{CO}_2$ and plasma CO_2 content when the patients are considered as groups, but not in individual cases. No relationship with arterial $p\text{H}$ was observed. No other relationships were noted.

REFERENCES

- Austen, F. K., Carmichael, M. W., and Adams, R. D. (1957). *New Engl. J. Med.*, **257**, 579.
- Bickford, R. G., and Butt, H. R. (1955). *J. clin. Invest.*, **34**, 790.
- Cameron, A. J. (1933). *Brit. J. Ophthalm.*, **17**, 167.
- Conn, H. O., Dunn, J. P., Newman, H. A., and Belkin, G. A. (1957). *Amer. J. Med.*, **22**, 524.
- Davies, C. E., and Mackinnon, J. (1949). *Lancet*, **2**, 883.
- Donald, K. (1949). *Ibid.*, **2**, 1056.
- Kety, S. S., and Schmidt, C. F. (1948). *J. clin. Invest.*, **27**, 484.
- Leggat, P. O. (1958). *Lancet*, **1**, 672.
- Patterson, J. L., Heyman, A., and Duke, T. W. (1952). *Amer. J. Med.*, **12**, 382.
- Riley, R. L., Campbell, E. J. M., and Shepard, R. H. (1957). *J. appl. Physiol.*, **11**, 245.
- Proemmel, D. D., and Franke, R. E. (1945). *J. biol. Chem.*, **161**, 621.
- Sieker, H. O., and Hickam, J. B. (1956). *Medicine (Baltimore)*, **35**, 389.
- Swanson, A. G., Stavney, L. S., and Plum, F. (1958). *Neurology (Minneapolis)*, **8**, 787.
- Van Slyke, D. D., and Sendroy, J., jun. (1928). *J. biol. Chem.*, **79**, 781.
- Westlake, E. K., Simpson, T., and Kaye, M. (1955). *Quart. J. Med.*, **24**, 155.
- Wilson, W. P., and Sieker, H. O. (1958). *Electroenceph. clin. Neurophysiol.*, **10**, 89.

SYPHILIS AS A CAUSE OF MENTAL DEFICIENCY

BY

J. M. BERG, M.B., B.Ch., M.Sc.

AND

BRIAN H. KIRMAN, M.D., B.Ch., D.P.M.

Fountain Hospital, London

Figures derived from the Ministry of Health (1958) report for 1957 illustrate the fact that syphilis is becoming increasingly rare in England and Wales (Tables I–III).

TABLE I.—Cases of Syphilis Dealt With at Centres in England and Wales

Year	No. of Cases, in all Stages, Dealt with for First Time	No. of Cases of Acquired Syphilis with Infections of Less than One Year
1931	—	8,924
1939	11,878	4,986
1946	23,878	17,675
1952	7,122	1,353
1957	4,977	747

1946 was the post-war peak year. The dramatic decline in the disease is indicated by the fall in attendances to a record low level 11 years later.

Let it be thought that modern methods of treatment obscure the true incidence of syphilis, the figures in Table II demonstrate that general paralysis of the insane, one of the main late clinical forms of the disease, is now much less important than it was.

TABLE II.—Deaths from General Paralysis of the Insane in England and Wales

Years	No. of Deaths
1911–20	2,080*
1936–9	852*
1945–9	359*
1953	117
1957	68

* Average annual deaths.

The decline in acquired syphilis has been paralleled by a similar change in the incidence of the congenital disease, as shown in Table III. In this case the peak of the relatively small wartime rise was reached in 1943, with a steady decline thereafter.

Routine serological tests for syphilis on pregnant women at various regional blood transfusion centres in England during 1957 showed a percentage incidence of

TABLE III.—*Congenital Syphilis in England and Wales*

Year	Cases Dealt with for First Time at Treatment Centres	Death Rates per 1,000 Live Births of Infants under 1 Year, Certified as due to Congenital Syphilis
1931	2,439	0.45
1939	1,614	0.17
1943	1,727	0.23
1950	1,223	0.04
1957	557	Nil

positives of 0.14% for primiparae and 0.29% for multiparae. The equivalent figures for 1953 were 0.21% and 0.43% (Ministry of Health, 1958).

With the fall in the incidence of syphilis in this country, mental deficiency due to it should become correspondingly less common and may eventually disappear from clinical practice.

Syphilis and Mental Defect

For practical purposes, syphilis as a cause of mental defect may be considered under the head of congenital syphilis. Legally, mental deficiency is defined as "arrested or incomplete development of mind existing before the age of 18 years." Occasionally, a 13- or 14-year-old child may acquire syphilis and develop general paralysis before the age of 18, but this must be a rare event. Most cases of syphilis producing mental defect are derived from the mother and are congenital.

In assessing the role of syphilis as a cause of mental defect it is important to exclude cases of acquired syphilis amongst adults previously mentally retarded. One reason for sending feeble-minded women to institutions was, and to some extent still is, the fact of illegitimate pregnancy, prostitution, or promiscuity. In this way there might be a tendency to collect in institutions for mental defectives a group at special risk in regard to acquired syphilis.

A further difficulty arises in regard to diagnostic criteria. Some of the earlier writers were inclined to look upon syphilis as an aspect of "moral degeneracy" rather than as a disease entity, and this may have resulted in increased estimates of the amount of mental deficiency due to the condition. Matters improved with the introduction of the Wassermann reaction into general use prior to the first world war, though technical errors were not infrequent. A refinement has been the development of various parallel tests, though even now the clinician is all too ready to base a diagnosis of syphilis on a single laboratory report. In our experience, it is not uncommon for a single positive report to be followed by several negative ones, and this experience extends over a great number of laboratories.

An additional consideration is that, even though the existence of syphilis in the mother be proved, it does not necessarily follow that any mental defect in the child is of syphilitic origin. A final possibility to be considered is the coexistence in the defective child of congenital syphilis and some other anomaly which might be responsible for the defect.

Tredgold (1929) reviewed some of the earlier literature on the role of syphilis in mental defect, and expressed his own opinion that "investigation of family histories would lead to the conclusion that syphilis was not a frequent cause of amentia." In his own series, 2.5% presented "undoubted marks" of the disease. He quotes serological investigations, carried out before the first

world war, which gave an incidence varying from 1.5% to 44.9% of positive Wassermann reactions in mental defectives. As recently as 1926, Stewart found 158 positive reactions in 800 mental defectives (19.75%) in Leavesden Hospital. This contrasts with 57 positives amongst 1,631 defectives (3.4%) at the Wrentham State School, Massachusetts (Dayton, 1925). In 1931 Cobb (quoted by Penrose, 1954) found positive serological tests in 129 of 1,275 cases (10.1%).

In the Colchester survey (Penrose, 1938), 51 patients out of 1,280 (4%) were diagnosed as having congenital syphilis, and 41 of them had positive blood Wassermann reactions. Penrose's figure of 4% is identical with that of Benda (1942), who found definite evidence of congenital syphilis in 80 of 2,000 patients of all ages at the Wrentham State School. In contrast to this is Paddle's (1937) finding of 10% for the incidence of congenital syphilis amongst the children at Caterham Hospital.

Syphilis, like rubella, is one of the few diseases in which there is good evidence that maternal infection during pregnancy may damage the infant. The risk is high, and it has been estimated (Moore, 1941) that a woman with latent syphilis has only one chance in six of bearing a normal child if she is untreated. Transmission of infection to the infant probably does not occur till the fifth month of pregnancy, which gives a period of grace during which infections discovered by routine antenatal serological investigation can be treated. Infections acquired later in pregnancy constitute a great danger, however.

Earlier views on the role of syphilis in producing mental deficiency were that it acted on the parental germ plasm; that the disease affected the general health of the mother and hence that of the foetus; that the disease produced mental defect only when there was a "neuropathic taint"; and that simple idiocy, without specific evidence of syphilitic lesions, might be due to the disease. The tendency to regard both mental deficiency and syphilis as punishment for sin is exemplified by Shuttleworth and Potts (1922), who state: "... a solitary infraction of physiological law is not often followed by the penalty of mental abnormality; if we only look far enough we shall generally find that such a culmination is reached by repeated transgressions." These authors also quote work suggesting that syphilis is responsible for 50% of cases of epilepsy as well as a considerable proportion of cases of mental defect showing neither "stigmata" nor any evidence of gross lesions in the central nervous system.

It is now generally agreed that congenital syphilis produces mental deficiency by spirochaetal invasion of the nervous system, and that the clinical syndromes resulting from this invasion can be classified in a similar manner to those which are found in the acquired disease. The main groupings are general paralysis with or without tabes, and meningovascular syphilis. There is no evidence to suggest that any other clinical types of mental defect are due to syphilis.

Incidence and Classification of Syphilis in Present Series

The Fountain Hospital Group provides accommodation for 800 mental defectives, mainly from the London area. Of these, about 80% are children of idiot or imbecile level, the majority of the remaining 20% being feeble-minded adults. A specimen

TABLE IV.—*Findings in Six Mental Defectives with Congenital Syphilis (Two other Cases (Cases 4 and 5) are Reported Fully in the Text)*

	Case 1	Case 2	Case 3	Case 6	Case 7	Case 8
Date of birth	1/2/08	5/10/25	14/5/33	15/12/44	13/11/45	20/1/47
Birth weight	?	?	?	5 lb. 3 oz. (2,354 g.)	5 lb. (2,268 g.)	5 lb. (2,268 g.)
Sex	Female	Female	Female	Female	Male	Male
Status	Legitimate	Illegitimate	Illegitimate	Illegitimate	Legitimate	Legitimate
Mother	Syphilitic history	Syphilis diagnosed 10 years after patient's birth. Incompletely treated	? Syphilitic	Blood W.R. and Kahn positive and rash when 7 months pregnant. Condylomata of anus and vulva at delivery. Treated antenatally	? Syphilitic	G.P.I. diagnosed 4 years before patient's birth. Treated with fever therapy and penicillin. Blood W.R. positive post-natally. Treated with penicillin, bismuth, and arsenicals
	Other pregnancies	8 previous pregnancies (3 miscarriages; 4 stillbirths) and 8 subsequent ones (4 miscarriages)	Previous miscarriage. No stillbirths	3 subsequent normal pregnancies. No miscarriages or stillbirths	Previous illegitimate macerated stillbirth. Previous illegitimate premature child who died at 3 weeks	Previous stillbirth. Previous congenital syphilitic who died in infancy
Father	Nothing known	Nothing known	Nothing known	Nothing known	? Syphilitic. 6 children by former marriage 1 died in infancy; others normal	Treated for syphilis
Patient's progress	Syphilitic history	Syph. periostitis of tibia and positive blood W.R. and Kahn at 7 years. Irregular treatment with bismuth, arsenicals, and mercurials for 6 years	" "	Blood W.R. and Kahn positive at birth. Treated with bismuth and arsenicals. Considered cured at 2 years	Cord blood W.R. positive. At 3½ years blood W.R. and Kahn positive. C.S.F., Lange paretic and excess proteins. Partially blind; optic atrophy; choroiditis; rhagades; snuffles. Treated with penicillin	"Poor specimen" at birth. Syphilitic. Treated with penicillin, bismuth, and arsenicals. No clinical or serological evidence of syphilis at 2 years
	Other features	Walked at 2 years. Went to school for defectives at 7 years	Retarded from infancy	Ophthalmia neonatorum at birth. Retardation and nystagmus at 8 months. Blind at 16 months	Retarded from infancy	Retarded from infancy. Fits for first 2 years of life

Findings on admission to Fountain Hospital

Age (years)	15½	23½	3½	6 2/12	11½	5 2/12
I.Q.	? (Imbecile)	82	29	35	60	24
Height	62 in. (157.5 cm.)	62 in. (157.5 cm.)	32 in. (81 cm.)	39½ in. (100 cm.)	51 in. (129.5 cm.)	36 in. (91.5 cm.)
Weight	62½ lb. (28.3 kg.)	128 lb. (58.1 kg.)	32 lb. (14.5 kg.)	33½ lb. (15.2 kg.)	51 lb. (23.1 kg.)	37 lb. (16.8 kg.)
Serology	?	Blood W.R. and parallel test negative	Blood W.R. and Meinicke positive. C.S.F. normal	Blood W.R., Kolmer, and Meinicke negative	Blood W.R. negative. C.S.F. normal	Blood and C.S.F. normal
Clinical findings	Hemiparesis. Choroiditis. Strabismus	? Frontal bossing	Psychotic features	Blind. Optic atrophy. Nystagmus	Partially blind. Optic atrophy. Choroiditis. Peg-shaped teeth	Strabismus
Subsequent developments	Blood W.R. and Meinicke often positive from 24 to 39 years. C.S.F. W.R. positive and Lange paretic at 26 years. Treated with arsenicals. Employed in community from 36 years	Able to manage own affairs at 26 years	Blood W.R. and Meinicke positive till 14 years. Treated with arsenicals for 2 years. Subluxation of femoral epiphysis at 16½ years. Mentally unchanged at 18 years	C.S.F. W.R. negative at 14 years. I.Q. then 50	Discharged at 12 years with recommendation for admission to school for educationally subnormal	No significant progress in next 6 years

TABLE V.—*Findings in Four Mental Defectives with Maternal History of Syphilis*

	Case 9	Case 10	Case 11	Case 12
Date of birth	25/5/44	9/7/48	7/2/50	2/7/50
Birth weight	10 lb. 7 oz. (4,735 g.)	8 lb. 8 oz. (3,855 g.)	6 lb. 5 oz. (2,865 g.)	9 lb. 7 oz. (4,280 g.)
Sex	Female	Male	Female	Female
Status	Illegitimate	Illegitimate	Illegitimate	Legitimate
Mother	Syphilitic history	Syphilis diagnosed at 38th week of pregnancy. Treated with bismuth and arsenicals	Blood W.R. and Kahn positive twice after labour. Treated with penicillin post-natally	Blood W.R. and Kahn positive 2 years after patient's birth. Treated with penicillin without effect on serology
	Other pregnancies	1 previous child, 2 subsequent children, all healthy. No miscarriages or stillbirths	Nothing	2 subsequent children, normal. 1 subsequent stillbirth
Father	"Healthy"	Nothing abnormal known	Nothing known	No evidence of syphilis
Patient's progress	? Normal development till lens opacities and partial blindness noted at 15 months	? Normal early development	Normal development till head injury at 11 months	Retarded from infancy

Findings on admission to Fountain Hospital				
Age (years)	9½	3 1/12	1 11/12	4 1/12
I.Q.	20	49	26	37
Height	?	37 in. (94 cm.)	34 in. (86 cm.)	36 in. (91.5 cm.)
Weight	78½ lb. (35.6 kg.)	35½ lb. (16.1 kg.)	23½ lb. (10.8 kg.)	36 lb. (16.3 kg.)
Serology	Blood W.R. and Kahn negative	Blood W.R. and Meinicke negative. C.S.F. normal	Blood W.R. and Meinicke negative. C.S.F. normal	Blood W.R. and Kahn negative
Clinical findings	Blind. Emotionally disturbed	Nothing abnormal found	Petit mal. Ptosis. Hemiparesis. Extensor plantars. Ankle clonus	Epilepsy. Strabismus

of blood is taken routinely on admission for Wassermann and parallel tests. Should either be positive, a second specimen is sent for examination. If the serum tests are consistently positive the cerebrospinal fluid is then examined. Data have been accumulated over a 12-year period, 1947-58, during which time some 1,900 patients have been available for examination. A note has been made of cases where serum tests were consistently positive, where there were clinical signs of syphilis, and where there was a history of syphilis in the patient or his mother.

A total of 12 patients fell into one or more of these categories, so that there was a *prima facie* case for consideration of syphilis as the cause of their mental deficiency. In seven of these patients there was convincing evidence of congenital syphilis. In an eighth case the evidence was a little less clear-cut but was acceptable. In the remaining four children there was a history of maternal syphilis but no reliable evidence that the child was suffering from the disease. Details of six of the eight cases with congenital syphilis are summarized in Table IV—the other two being reported in full below—and of the four with maternal syphilis in Table V. It is notable that 4 of our 12 patients were born before the second world war and none after 1950. Also of interest is the fact that 7 of the 12 were illegitimate.

Without the support of neuropathological material it is difficult to make a precise diagnosis in some mental defectives with congenital syphilis. Table VI shows a tentative grouping of our cases.

TABLE VI.—*Clinical Classification of 12 Mental Defectives with Syphilitic History*

Form of Syphilis	No. of Cases	Case No.
Taboparesis	3	4 certain; 6 and 7 probable
Meningovascular	2	1 and 5
Congenital with mental defect	3	2, 3, and 8
Maternal only	4	9, 10, 11, and 12

There was only one classical taboparetic (Case 4), but the association of mental defect with congenital syphilis in Cases 6 and 7 (optic atrophy in Case 6, and positive cerebrospinal fluid findings in Case 7) was thought to justify their inclusion in this group. The two patients with hemiparesis (Cases 1 and 5) were thought to have the meningovascular form of the disease with a localized lesion. The remaining three cases in which congenital syphilis was regarded as established (Cases 2, 3, and 8) may or may not owe their mental deficiency to the disease. There is no good evidence in Cases 2 and 3 to suggest that the nervous system was ever invaded. In Case 8 the history of fits in infancy and treatment including penicillin is compatible with an early neurological lesion which may have been arrested by treatment, leaving the blood and cerebrospinal fluid normal.

Finally, there is the group of four cases with syphilis in the mother but no evidence of infection in the infant. There is no sound basis for considering that these children are mentally defective because of syphilis. On the other hand, the possibility cannot be completely dismissed. For example, in Case 10 treatment was started at the 38th week of pregnancy. It seems just possible that treatment may have arrested disease in the infant's already damaged brain but left it free from clinical signs of infection.

In this series there is not sufficient information about the precise date of the mother's infection to attempt to draw conclusions from this. It is usually thought that neurosyphilis in the infant is more likely to occur in the later stages of the maternal infection. It will be seen that a number of the mothers had had previous miscarriages, stillbirths, or children who died young, including one known to be syphilitic.

Case 4

This patient, a girl, was born on September 28, 1936. No history of parental syphilis was elicited, but the parents were regarded as very unreliable witnesses. The mother had a stillbirth prior to the patient's birth, and, following the latter, four apparently normal children with no further stillbirths or miscarriages.

The patient weighed 9 lb. (4,080 g.) at birth. Details about the earlier years of life are not known, but there is a suggestion of normal development till the age of 7. At 9 years she was admitted to hospital because of fits and found to be mentally retarded. Argyll Robertson pupils, tremor of the tongue, and slurred speech were noted. The knee- and ankle-jerks were absent. Plantar responses were flexor and the gait was steady. No other clinical abnormalities were found. The blood Wassermann reaction was positive and the cerebrospinal fluid revealed a positive Wassermann reaction, a colloidal gold (Lange) curve of 5555543200, 36 cells per c.mm., and a protein content of 90 mg. per 100 ml. Cerebrospinal fluid examination six months later, after intrathecal and intramuscular penicillin, yielded a positive Wassermann reaction, a negative Lange curve, 5 cells per c.mm., and a protein content of 100 mg. per 100 ml. Before further investigations and treatment could be undertaken, the mother insisted on taking the patient home.

The child was admitted to the Fountain Hospital when 11½ years old. Her height was 4 ft. 11 in. (150 cm.), her weight 96 lb. (43.5 kg.), and her I.Q. 43. She was co-operative and considerate, her habits were clean, and she could feed and dress herself. She answered simple questions but could not spell her name, did not know her age, and suffered from attacks of screaming. She could count to 20 and knew the alphabet to letter F. There were no physical signs apart from slight Rombergism and absent knee-jerks. The blood Wassermann and Meinicke reactions were doubtfully positive, and the cerebrospinal fluid yielded a negative Wassermann reaction, a Lange curve of 0122100000, and a positive Pandy test. At 14 years the cerebrospinal fluid was normal in all respects apart from a Lange curve of 1121000000. Over the next few years there was slight but not significant improvement in her mental level.

Case 5

This patient, an illegitimate male, was born on July 29, 1944. The mother was found to have secondary stage syphilis seven weeks before the child's birth. She was treated with bismuth and arsenicals till her confinement, her blood Wassermann reaction and Kahn test remaining positive throughout. Treatment continued for two weeks after delivery, when she discharged herself from hospital, taking the child with her. She had had an apparently normal child two years previously. No other pregnancies are known. She said that the father had venereal disease.

The child weighed 8½ lb. (3,855 g.) at birth, following a full-term normal delivery. Wassermann reaction and Kahn test on the cord blood were negative, and several days later were again negative; there was no clinical evidence of syphilis neonatally. The child remained with the mother without further medical attention till 8 months old, when he was investigated in hospital after a convulsion. The fit was associated with otitis media and a respiratory infection, and, in addition, craniotabes and positive blood Wassermann reaction and Kahn test were noted. There were no bony

changes on x-ray examination. The cerebrospinal fluid showed a positive Wassermann reaction and a colloidal gold (Lange) curve of 11100000 with no excess of protein or cells. Antisyphilitic treatment, consisting of penicillin, bismuth, and arsenicals, was begun at this stage and continued for two and a half years. During this period the blood Wassermann reaction and Kahn test were repeatedly positive and the child was ataxic and mentally defective. When 3 years old he developed a left hemiplegia. Thereafter, several blood and cerebrospinal fluid examinations yielded negative results.

On admission to the Fountain Hospital at the age of 3½ years his I.Q. was 48. His height was 39½ in. (100 cm.) and weight 31 lb. (14 kg.). He was a quiet child with a limited vocabulary, who could feed himself with a spoon, help dress himself, and had sphincter control. He took considerable interest in his surroundings, and could point out parts of his body on request. Physically, the only abnormalities were left hemiparesis with a left extensor plantar response, and internal strabismus of the left eye. No evidence of syphilitic infection was found in the blood or cerebrospinal fluid. He progressed in hospital, and by the age of 11 was well within the educationally subnormal range. He then began attendance at a school for educationally subnormal physically handicapped children.

Discussion

Even if all 12 of our cases owe their defect to congenital syphilis, the incidence in this series is only 0.6%. If more stringent criteria are used, however, only Cases 1, 4, 5, 6, and 7 are acceptable, in that they showed evidence of neurosyphilis in the form of changes in the cerebrospinal fluid, or appropriate clinical signs. This gives a figure of 0.2% due to congenital syphilis. Even the larger of these two percentages is very much lower than many previously reported, and the conclusion is that syphilis is not now a major factor in the London area as a cause of mental deficiency. We have no reason to think that the position in London is not typical for the country as a whole. Table VII shows the relative frequency of syphilis compared with other aetiological factors or syndromes found in our series of 1,900 mental defectives.

TABLE VII.—Cases Diagnosed in Certain Categories in Fountain Hospital Group, 1947 to 1958.

Category	No. of Cases
Mongolism	249
Meningitis (tuberculous and purulent)	40*
Phenylketonuria	28
Kernicterus	21
Tuberous sclerosis (epiloia)	15
History of syphilis	12
Rubellar embryopathy	10
Cerebral lipoidosis	8
Sturge-Weber syndrome (naevoid amentia)	4

* Includes six cases where meningitis was of doubtful aetiological significance.

An obvious explanation for a decline in the proportion of mental deficiency due to syphilis is the general decline in the incidence of the disease as shown in the figures given above. The results arrived at in earlier surveys were, however, so variable that we are inclined to believe that many of the higher estimates were unreliable. It is well known that the technical level of the results in different laboratories has varied (Aselmeyer, 1939). An additional factor which may account for some of the reported differences has been mentioned—namely, failure to insist on a repetition of tests before regarding a patient as syphilitic on serological evidence. Despite the conflicting evidence from earlier figures, there is no doubt that there has been a striking and gratifying

decline in the number of cases of mental defect due to syphilis over the past 20 years. Penrose's Colchester estimate may be taken as a conservative basis for comparison.

A striking feature of the figures quoted earlier for new cases of acquired syphilis was the big increase during and after the second world war. It might have been anticipated that this would bring about a similar rise in congenital syphilis. It is presumably a tribute to the efficacy of treatment that the rise was relatively small. It may also be that effective treatment of affected infants will have prevented some from developing neurological lesions and hence from becoming mentally defective.

Conclusion and Summary

Congenital syphilis is now rare as a cause of mental deficiency in England and Wales. The rise in the incidence of acquired syphilis associated with the second world war does not seem to have been paralleled with a great increase in congenital syphilis or mental defect due to this cause. This is presumably a result of good antenatal supervision of pregnant women and prompt treatment of acute cases amongst expectant mothers and infants.

There is no good evidence that syphilis produces any forms of mental deficiency other than well-recognized syndromes such as general paralysis and meningo-vascular syphilis. Syphilitic mental deficiency is eminently preventable and should disappear. So long, however, as the number of adult cases remains at the present still considerable level, it would be unwise to relax precautions like a routine antenatal Wassermann test or routine investigation of admissions to mental deficiency hospitals, with appropriate attention to the families if positive cases are found. Increasing contact with countries which have been less successful in resolving this problem is an additional argument in favour of retaining such precautions.

Among some 1,900 mental defectives, mainly idiot and imbecile children, studied in the Fountain Hospital Group from 1947 to 1958 there were 12 with a history of syphilis. In eight cases there was congenital syphilis—three of these were classified as taboparetic and two as meningovascular, leaving three whose mental defect was not certainly attributable to their congenital syphilis. In the remaining four cases the mother only was affected and there was no proof of infection in the child.

Our thanks are due to Dr. Mary Woodward and other present and past members of the Psychology Department, Fountain Hospital, for their reports; and to our medical and other colleagues for help and advice.

REFERENCES

- Aselmeyer, A. J. (1939). *Urol. cutan. Rev.*, **43**, 1.
- Benda, C. E. (1942). *Amer. J. ment. Defic.*, **47**, 40.
- Dayton, N. A. (1925). *Ment. Hyg. (N.Y.)*, **9**, 760.
- Ministry of Health (1958). *Report for the Year 1957*. Part II. H.M.S.O., London.
- Moore, J. E. (1941). *The Modern Treatment of Syphilis*, 2nd ed., p. 475. Thomas, Springfield.
- Paddle, K. C. L. (1937). *J. ment. Sci.*, **83**, 372.
- Penrose, L. S. (1938). *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, No. 229.
- (1954). *The Biology of Mental Defect*, revised ed., p. 192. Sidgwick and Jackson, London.
- Shuttleworth, G. E., and Potts, W. A. (1922). *Mentally Deficient Children*, 5th ed., p. 70. Lewis, London.
- Stewart, R. M. (1926). In *Annual Report for 1925-1926 of Metropolitan Asylums Board*, p. 213. London.
- Tredgold, A. F. (1929). *Mental Deficiency (Amentia)*, 5th ed., p. 43. Baillière, Tindall and Cox, London.