of any of the 81 persons aged 15 to 60 years. Even in the case of those specially exposed to possible contact with the tetanus bacillus, Lahiri (1939) found no tetanus antitoxin. There is therefore no likelihood of new-born infants having even a slight degree of immunity to tetanus, such as is commonly met with in the case of diphtheria in infants during the first year of their lives.

Can infants be protected against tetanus neonatorum by immunizing their mothers during their pregnancies?—In this connection it is of interest to note that Marvell and Parish in 1940 observed that women responded much better than men to tetanus toxoid injections. There seems good reason to expect that tetanus antitoxins developed in the blood of pregnant women by means of toxoid injections will be transmitted to the child she is carrying, as occurs in the case of diphtheria antitoxins, although I have not found any records of this having been experimentally tested.

The object of this note is to suggest that this possibility should be investigated at the Calcutta School of Tropical Medicine with the help as regards clinical material of the Eden and Dufferin Hospitals by determining the tetanus antitoxin titre of the serum of the immunized mothers and of their newly-born infants. If this test should demonstrate that practically important amounts of tetanus antitoxin are transmitted to the infants, it will clearly be advisable to immunize as many mothers as possible during the prenatal period by means of two doses as early in the pregnancy as proves to be practicable, and to give an additional dose ten days before the expected date of delivery to provide the maximum protection of the babes against the frequent and deadly tetanus neonatorum of tropical countries.

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POLYNEURITIS

WITH SPECIAL REFERENCE TO THE ACUTE VARIETIES

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Introduction

CHRONIC polyneuritis is a clinical entity with which most practitioners are familiar. The diagnosis is easy and an ætiological factor can often, not always, be discovered. What perhaps may not be so familiar is polyneuritis of acute onset occurring in persons apparently completely healthy with no obvious dietetic deficiency or toxic or infective factor. The diagnosis presents difficulties, and the ætiological factor is usually untraceable. That acute polyneuritis is not an unusual occurrence is indicated by the fact that the writer observed six cases in a period of nearly two years at the V. J. Hospital, Amritsar. An account of these cases follows. A brief reference has been made to five cases of chronic polyneuritis which were observed in the same period to give an idea of the relative frequency of the two types of polyneuritis in the experience of the writer.

Case reports

Group I: Polyneuritis with acute onset and negative Wassermann reaction.

Case 1.—A well-nourished healthy looking student, aged 20, was admitted on 1st May, 1943. Soon after excessive physical exertion on 2 successive days involving the carrying of heavy weights in hot weather he began to feel pain in the back and upper limbs and some stiffness of the neck. This sensation lasted 3 to 4 days. One of the legs then felt weak and he began to have some difficulty in micturition and defæcation. The symptoms gradually increased for another 3 to 4 days and the other leg became involved. On admission he was unable to walk, to sit up in bed without the use of arms, to pass urine or to defæcate. He complained of a sense of oppression in the chest and some stiffness in his neck. The lower extremities showed loss of deep reflexes, Babinski sign absent on both sides. Abdominal reflexes absent. The superficial sensations were dull below the knee but not lost, there was no muscular tenderness. No pains. The upper extremities were practically normal. No fever. Physical examination of the rest of the nervous system and other systems negative. No infective foci. Urine negative. Blood W.R. negative, C.S.F. negative. There was no recent history of exanthemata. A previous history of typhoid fever 7 years ago. No history of drug taking. Dietetic history did not reveal anything out of the ordinary. The patient was placed on massive doses of vitamin B_1 by injection. The condition improved steadily. Bladder and intestines started functioning on the 5th day of treatment and were in full function on 10th day. Power in legs started improving on the 5th day and by the 10th patient was able to sit up in bed without support.

On 25th May, 1943, when the patient was discharged he was capable of walking without support but right leg still showed weakness. Sensation of legs normal.

On 4th June, 1943, patient presented himself again. He said he was quite all right but that the right leg was still somewhat heavy.

Case 2.—A college lecturer was admitted on 21st May, 1942, with a history of sudden weakness developing in the legs and spreading to the arms 3 to 4 days previously. On admission the patient was helpless, had flaccid paralysis of all four extremities with wrist-drop and foot-drop and some weakness of the trunk. There were marked sensory symptoms with tingling and pains in the limbs, especially the legs, and marked muscular tenderness. Pains were severe at night. Stocking anæsthesia present in the legs, but no demonstrable sensory loss in the arms. Babinski negative. No troubles with the bladder or rectal sphincters. No fever but patient said he felt out of sorts just before illness. Physical examination of the rest of the body did not reveal anything abnormal. Previous history did not yield anything relevant to the illness. No evidence of dietetic insufficiency or peculiarity. Blood W.R. negative. C.S.F. no abnormality. Urine no abnormality. Patient showed steady and marked improvement on vitamin B_1 therapy. About 3 weeks later when he left, he could walk without support but was

a little shaky, had regained considerable power in arms, and sensory symptoms had greatly improved.

Case 3.—D., 35, M. M., an agricultural worker admitted on 26th May, 1942, was completely incapacitated with flaccid paralysis of all four extremities, wrist-drop and foot-drop, glove and stocking anæsthesia, and negative Babinski. Muscles tender and wasted. The paralysis came on 1½ months previously after a bath, with paræsthesia and muscular weakness spreading upwards from the legs and becoming extreme within a few days. Some disturbance of urination in earlier days. Physical examination of the other systems and previous history did not yield any relevant information. Blood W.R. negative. C.S.F. negative. Patient stayed only 5 days in hospital and subsequent course of the disease is unknown.

Group II : Polyneuritis with acute onset and positive Wassermann reaction.

Case 4.—V. J., 25, O. M., a house servant, was admitted on 14th February, 1942. Two months previously paræsthesia and weakness appeared in the hands and arms. Weakness gradually increased and became marked in a few days involving the muscles of the shoulder girdle and the trunk, the legs also weakened but compared with the arms retained considerable strength and patient on admission could stand and walk slowly but needed a little support. On admission the paralysis was of flaccid type with considerable wasting of the arms, and shoulder girdle muscles Babinski absent. Abdominal reflexes absent. Deep reflexes absent in arms, sluggish in legs. No sensory symptoms whatever. In earlier stages bladder control reported to be defective. Apart from syphilis 5 years previously history and examination of the body yielded no relevant information. Blood W.R.+++. C.S.F. no abnormality. Anti-syphilitic treatment given in the hospital.

On 29th April, 1942, when the patient was discharged, he had regained considerable power in his arms and could walk but was still weak.

Case 5.—B., 35, H. M., was admitted on 22nd September, 1942. Three months previously the patient after his bath felt pain in the loin. Tingling and weakness appeared in the legs and within a few days the legs, trunk and arms were paralysed with retention of urine. No sensory symptoms. Deep reflexes absent. Muscles of arms and legs wasted and paralysed. Recti weak. Babinski absent. Superficial abdominal reflexes sluggish. Bladder functioning normally. Except syphilis 4 years previously history and physical examination negative. Blood W.R.+++. C.S.F. no abnormality. Patient put on anti-syphilitic treatment. On 19th May, 1943, patient could use his arms and walk without help but still weak.

Case 6.—P. B., 40, H. M., was admitted on 6th December, 1941, with history of flaccid paralysis of legs spreading to trunk, arms and face within a few days and temporary bladder disturbance, duration of symptoms about 3 weeks. Marked pain and muscular tenderness and anæsthesia of upper and lower extremities of glove and stocking type. Babinski, tendon and abdominal reflexes absent. As recovery set in, an extensor plantar response (bilateral) appeared without exaggeration of deep reflexes. At this time it was noticed that the extensors of the toe had regained voluntary power, whereas the flexors were still paralysed, and the sole of the foot was hyperæsthetic. The positive Babinski therefore could not be taken as evidence of an upper motor neurone lesion (Monrad-Krohn, 1938). A true pyramidal inversion of the plantar reflex may also occur in acute febrile polyneuritis during the stage of convalescence (Cobb and Coggeshall, 1934). There was a history of syphilis several years previously, history and physical examination yielded no additional information. Blood W.R.++. C.S.F. no abnormality. Anti-syphilitic treatment was given. On 17th April, 1942, when the patient left hospital, he had recovered considerably the use of arms and legs, anæsthesia disappeared but some dullness of sensation over the feet and some tenderness of the soles of the feet on pressure were still present.

Group III : Chronic polyneuritis.

In the same period the writer observed five cases of chronic polyneuritis which could be assigned to the following ætiological groups :—

1. Leprosy. One case. Nervous manifestations in both legs. Nerves thickened. No skin lesion.
2. Nutritional. Two cases. One patient was a Kashmiri, the other was from the Kangra district. Dysentery or diarrhœa, anæmia, œdema of the legs, and peripheral neuritis involving the legs were present in both.
3. Diabetes. One case. Only the legs were affected.
4. Typhoid fever. One case. Patient showed flaccid paralysis of legs, trunk and arms with dysarthria developing soon after an attack of fever lasting about 3 weeks. Initial weakness became steadily worse and paralysis was complete in about 8 weeks. Plantar reflex flexor. No sensory disturbance or bladder involvement. Duration of symptoms about 5 months. Wassermann reaction negative.

Clinical features of group I

1. Onset sudden, spread of paralysis rapid. In case 2 the patient was rendered almost completely helpless overnight. History of fatigue or exposure in cases 1 and 3.
2. Paralysis was of ascending type, beginning from the legs. All the three patients were almost completely helpless on examination. There was involvement of the trunk, but the respiratory and facial muscles showed no paralysis. Wasting was only noticed in case 3.
3. All cases showed paræsthesia. There was little loss of superficial sensation in case 1, and no muscular tenderness. Cases 2 and 3 showed greater sensory loss of peripheral distribution and marked muscular tenderness.
4. Deep reflexes absent; Babinski negative or flexor. Defective control of bladder in early stage in all cases; rectum affected in case 1 only.
5. Cases 1 and 2 showed rapid recovery with an average duration of illness of about 6 weeks. Case 3 was observed for a short period only, but it was apparently a more protracted case and was showing little evidence of improvement. There was no death.
6. No prodromal symptoms were noticed (or they were too slight to be noticed). Patients showed no constitutional disturbance or fever.
7. Cerebro-spinal fluid showed no abnormality; there was no increase in cell content or globulin. Quantitative estimation of protein was not done. The absence of hyper-albuminosis may be attributed to mildness of tissue reaction.
8. Blood Wassermann reaction negative.
9. All cases occurred in April and May.

Clinical features of group II

The signs and symptoms were essentially similar to group I with the following differences :—

1. Both the onset and spread of paralysis were somewhat less acute.
2. Paralysis showed a descending tendency in case 4. The paralysis was not severe, and patient not so helpless, except in case 6 in which the patient was completely paralysed and in addition showed facial paralysis.
3. A preponderance of motor symptoms was noticeable in the group except in case 6.
4. All cases showed a protracted course extending over several months and the recovery was slow.
5. Blood Wassermann reaction was positive in all cases and there was a history of syphilis.
6. No special seasonal incidence was noticeable.

The clinical differences between the two groups are thus quantitative except for the positive Wassermann reaction and history of syphilis.

Clinical features of group III

This group is characterized by a slow onset and chronic course of disease; paralyses mild and limited in extent except in case 5 which showed extensive paralysis without sensory phenomena. In all the cases an ætiological factor could be recognized.

Discussion of clinical features of groups I and II

The clinical picture of all varieties of polyneuritis is essentially the same, the differences being quantitative rather than qualitative.

Clinical features of polyneuritis.—The different ætiological varieties maintain a constant mode of onset and course, and can be divided into acute, sub-acute and chronic polyneuritis. Walshe (1941) describes two varieties of acute polyneuritis occurring in apparently healthy people, in whom no dietetic, toxic or infective factor can be discovered.

I. 'Acute febrile' or 'acute infective' polyneuritis.—Brain refers to an 'acute rheumatic polyneuritis' following exposure to cold, which probably also belongs to this group. The clinical description of this form of polyneuritis varies from author to author. An analysis of symptoms of 122 cases reported in literature by Fox and O'Connor (1942) is therefore instructive. According to these authors—

1. Prodromal symptoms such as coryza, aches and pains, gastro-intestinal disturbance appear in 31 per cent of cases.
2. Motor symptoms are constant and are often first to appear, including paralysis of extremities, disturbance of tendon reflexes and occasionally organic reflexes.
3. Sensory phenomena, pain, paræsthesia or anæsthesia occur in 50 per cent of cases.
4. Intercostal muscles and diaphragm are seldom involved.
5. Facial paralysis occurs in 35 per cent of cases.
6. Blood is normal, perhaps slight leucocytosis.
7. Cerebro-spinal fluid. Protein increased in 50 per cent of cases. Cell count normal.
8. Prognosis—mortality 20 per cent. Recovery takes on average 2 to 3 months.

According to Walshe the patient is intensely toxic and paralysis is sudden and widespread, involving the extremities, trunk, and usually the face; less often paralysis of chest and transient loss of control of the bladder. Sensory phenomena are variable, often slight. The essential feature is a relatively quick recovery in about 4 weeks, if the patient survives.

Brain (1940), although agreeing with the general description of the disease, remarks : 'In the most favourable cases the patient is not likely to be convalescent in less than 3 to 6 months.' According to this author, prodromata may be slight or absent and there may be no fever during the paralytic stage.

Cases in group I, on comparison with symptoms detailed above, show absence of prodromata, fever and toxic phenomena, respiratory and facial paralysis, and of hyper-albuminosis in cerebro-spinal fluid. They however possess the essential features of sudden and widespread paralysis tending to relatively quick recovery in the absence of a fatal issue, and may therefore be taken to represent a mild form of acute infective polyneuritis. The four cases reported by Fox and O'Connor (1942) show prodromata, hyper-albuminosis and quick recovery but little or no toxæmia during the paralytic stage.

II. Acute polyneuritis of unknown ætiology.—Under this heading Walshe (1940) describes a polyneuritis with motor and sensory symptoms, a rapid onset, but on the whole less acute and extensive paralysis as compared with the acute febrile variety; there is no fever or constitutional disturbance, and recovery is long delayed. The two cases reported by him took 9 months to recover. Cerebro-spinal fluid showed slight excess of proteins.

The clinical features of this variety of polyneuritis resemble those of cases included in group II, except that case 6 shows more extensive and severe paralysis rather like group I and cases 4 and 5 show purely motor symptoms. The essential feature—a paralysis of rapid onset with a protracted course of disease—is noticeable in all the reported cases in this group.

There may be some doubt with regard to the classification of case 3 in group I. This case has features which are common to both groups I and II. The acuteness of onset, severity of symptoms, seasonal incidence, and a negative Wassermann reaction have been the chief considerations in the classification of the case, because the clinical course of the case is not definitely known. Such difficulties are unavoidable in a disease with such marked variations in the clinical picture and course as is suggested by descriptions of the disease from different sources.

Ætiology of polyneuritis.—Cobb and Coggeshall (1934) mention over eighty causes divisible into four groups in the ætiology of polyneuritis—

(1) Virus infections. (2) Bacterial infections. (3) Deficiency or disorder of metabolism. (4) Chemical poisons. Such a multiplicity of causes indicates that the causes are imperfectly understood. It is not surprising that Walshe opines that not more than ten factors can be rightly incriminated. Walshe is of opinion that in spite of the diversity of causes in the main clinical varieties of polyneuritis—acute, sub-acute and chronic—there is an essential clinical and pathological uniformity, the difference being quantitative rather than qualitative. It is, therefore, possible that a single metabolic poison is produced in the body as a result of the different causes, and acts as a neural and myocardial poison. Such a poison is produced in beri-beri as a result of disturbed carbohydrate metabolism due to vitamin B_1 deficiency, but the nature of the poison is not known; it does not appear to be pyruvic acid. Whether a similar mechanism operates in other forms of polyneuritis due to vitamin B_1 deficiency it is impossible to say at this stage, but Walshe believes that the results of vitamin B_1 therapy at least do not support this hypothesis. In particular he finds it difficult to explain the acute forms of polyneuritis on the hypothesis of avitaminosis. Current opinion appears to be that vitamin B_1 deficiency plays an important rôle in the causation of chronic forms of polyneuritis such as beri-beri, chronic alcoholism, pregnancy, chronic disorders of the alimentary canal and possibly restricted diet.

In group III an ætiological factor is recognizable in all cases.

Acute febrile polyneuritis is believed to be due to a specific but unknown virus infection. The rapid onset of symptoms in apparently healthy people, without obvious dietetic or constitutional defects, the factors of fatigue and exposure which precede some cases, seasonal incidence, febrile and toxic manifestations, possible leucocytosis, favour the hypothesis of infection. In group I cases some of these features are present, and the absence of toxic manifestations can be attributed to infection with a virus of low toxicity, giving rise to a benign type of disease.

Acute polyneuritis of unknown ætiology.—Walshe does not attempt to assign a cause to this form of polyneuritis, but he does not favour the avitaminosis hypothesis. Cases in group II, which have been classified under this heading, have the common characteristic of a positive Wassermann reaction and a history of syphilis. In a population with a positive W.R. rate of 10 to 15 per cent this association may be a pure coincidence, but it seems to the writer to be a suspicious coincidence. Polyneuritis due to leprosy may be associated with a positive W.R., but it was ruled out on account of the absence of other clinical evidence of leprosy and the fact that leprosy is not known to cause an acute form of polyneuritis. Is the *Treponema pallidum* the dominant factor in the causation of this group of cases? Textbooks are silent or lukewarm on the subject of syphilitic polyneuritis. Some of the books refer to a polyneuritis complicating the secondary stage of syphilis as a rare phenomenon. Cobb and Coggeshall (1934) deny the existence of syphilitic polyneuritis. Drought (1940) describes an acute polyneuritis with slight sensory loss, which may complicate the secondary stage of syphilis. The cerebro-spinal fluid is normal and pupillary changes absent. He also records a case, reported by Macnamara, of 'Landry's paralysis' with purely motor symptoms 6 years after syphilitic infection, from which the patient recovered but subsequently succumbed to tabes. According to present conceptions, a purely motor ascending paralysis which tends to recover completely is a form of acute polyneuritis with purely motor symptoms, and cases 4 and 5 in group II show purely motor symptoms. The *Treponema pallidum* has a special affinity for the nervous system, and in tabes dorsalis it admittedly attacks the root portions of the peripheral nerves. Might it not, occasionally, attack the rest of the peripheral neurone? It appears to the writer, therefore, that the relationship of syphilis to group II cases cannot completely be denied. Unfortunately there is no reference to the venereal history of the two cases described by Walshe.

Vitamin B_1 deficiency in acute polyneuritis.—An obvious objection is that syphilitic infection is common, whereas syphilitic polyneuritis is rare (if the experience of the writer is taken as unusual). The same objection applies to acute febrile polyneuritis which, although a virus disease, is said to be rare. Other virus diseases are far more common. The writer believes that in the causation of both varieties of acute polyneuritis some cause other than organismal also operates, and in spite of the authoritative opinion of Walshe which deserves the greatest respect, the most likely factor is vitamin B_1 deficiency, or some poison due to it. Although no dietetic peculiarities were noticed in the reported cases, and the average Punjabi diet is said to be adequate in vitamin B_1, and gross vitamin B_1 deficiency is a rare phenomenon in the Punjab (the writer is only stating what one is asked to believe), the existence of sub-clinical deficiency cannot be entirely ruled out. Williams and Spies (1938) remark: 'The American diet is one of the best in the world and yet many Americans lack an optimum diet for growth and full vigour. Careful study has convinced most students of the disease that sub-clinical forms of vitamin B_1 deficiency occur frequently.' If this is true of America, what about the Indian masses whose economic and educational standards, taken with the appalling frequency of malaria and bowel disease, tuberculosis and other infectious diseases, provide the most favourable conditions for vitamin deficiency? Such factors as unbalanced, mainly carbohydrate diet, inadequate intake, deficient

absorption, excessive requirements, and deficient utilization will frequently meet the eye of a careful observer. Given a sub-clinical B_1 deficiency, possibly associated with increased susceptibility to toxic and infective influences, a virus, *Treponema pallidum*, or an unknown toxin may attack a devitalized peripheral neurone giving rise to rapid and even sudden loss of function. This would also mean that acute polyneuritis should be more common in the tropics which, indeed, the experience of the writer seems to suggest. In the absence of B_1 deficiency, the virus or the treponema may not gain a foothold. On the other hand in beriberi, in which bacterial causes are not believed to operate, deficiency of vitamin B_1 may exist for months, even years, before chronic neuritic manifestations set in.

Diagnosis of acute polyneuritis.—From the clinical point of view, acute polyneuritis presents a diagnostic problem of some complexity because the symptoms are widespread involving apparently the cord and sometimes the cerebrum. The difficulty is partly due to a narrow conception of the pathology of the disease. If it is realized that the peripheral neurone extends from the grey matter of the central nervous system to the peripheral end-organs, and that the whole of it or only part of it may show the maximum incidence of pathological changes, the variation in symptoms and signs in different cases and the different permutations of sensory, motor and reflex phenomena will be easily understood. It should also be remembered that transitory peripheral symptoms may be met with in acute virus infections of the central nervous system such as poliomyelitis and encephalitis lethargica.

1. *Acute myelitis.*—It is characterized by sudden motor and sensory paralysis below a segmental level. The paralysis is flaccid in type but exaggerated reflexes and extensor plantar response soon make their appearance indicating a lesion of the upper motor neurone. Involvement of the bladder is of a more serious nature than in acute polyneuritis.

2. *Poliomyelitis.*—The paralysis is asymmetrical and inconstant, often maximal at onset and then receding. In other cases it may spread from below upwards. There is no sensory loss. Recovery is also irregular. Some of the muscles recover rapidly, others show permanent paralysis with great wasting.

In cases of acute polyneuritis with preponderant or purely motor symptoms the resemblance is more marked.

It may be remarked here that the so-called Landry's paralysis is not now considered a separate clinical entity. An acute paralysis of spreading type without sensory phenomena may be due to acute anterior poliomyelitis or acute infective polyneuritis with preponderant motor symptoms.

Treatment.—According to Walshe, vitamin B_1 therapy does not influence the course of acute or chronic polyneuritis. He concludes that therapeutic results at least do not lend support to the vitamin B_1 deficiency theory of causation of polyneuritis. This is an observation from a great authority, and it cannot be treated lightly. Furthermore, in the acute form of polyneuritis the course of disease varies considerably from case to case, and it would be difficult to assess the value of a therapeutic measure unless a large number of cases were treated under properly controlled conditions. In the reported series of group I, cases 1 and 2 received intensive vitamin B_1 therapy at an early stage of the disease and both recovered rapidly. Case 3 did not receive this treatment and was in a protracted state of helplessness. It would be rash to draw conclusions from such a short series, but the results suggest the therapeutic utility of vitamin B_1 administered at an 'early' stage of the disease before structural changes have set in. On the other hand, the cases reported by Fox and O'Connor, which the writer considers comparable to this group, recovered without vitamin B_1 therapy in an even shorter period. In group II cases, anti-syphilitic treatment alone was administered. Vitamin B_1 could not be given and the course of improvement was slow but certain. In the protracted course, treponema infection may have been the principal factor. Whether the administration of vitamin B_1 to this group of cases would have hastened recovery it is impossible to say. What would have been the course of the disease without anti-syphilitic treatment it would be difficult to say without controlled observations on similar cases.

Summary and conclusions

1. Acute polyneuritis occurring in apparently healthy people in whom no toxic, infective or dietetic factors can be discovered is not an uncommon clinical experience. In the same period the writer observed as many as six cases of acute and only five cases of chronic polyneuritis.

2. The essential feature of acute infective polyneuritis is extensive paralysis of sudden onset from which the patient recovers rapidly if he does not succumb to the disease. Three cases of this type (group I) are described in the text, but the disease appears to be relatively benign, possibly due to infection with a virus of low virulence.

3. A less acute and milder form of polyneuritis with a protracted course and of unknown ætiology is described by Walshe. Three cases (group II), described in the text, belong to this variety. In the cases observed by the writer, however, syphilis appears to be one of the ætiological factors.

4. Vitamin B_1 deficiency is not believed to play a part in the direct causation of acute polyneuritis, but it has been suggested that sub-clinical deficiency may be a predisposing factor and a specific virus and *Treponema pallidum* precipitating factors. There may be

other unknown precipitating factors to explain such cases as those reported by Walshe.

5. The efficacy of vitamin B_1 therapy in polyneuritis, particularly in the acute forms, is open to considerable doubt, but the writer's experience suggests that it is worthy of further trial under controlled conditions.

6. In chronic polyneuritis an ætiological factor can usually be found. A reference to five cases observed in the same period (group III) has been made. Details of one case of polyneuritis, with no sensory loss, following typhoid fever are included.

7. Case 6 illustrates the presence in exceptional circumstances of extensor plantar response in peripheral paralysis.

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[*Note.*—The editor is interested in this subject partly because he himself in 1939 suffered from a very severe attack of acute polyneuritis following directly on a respiratory infection of 'influenzal' character. The paralyses were extensive, including the limbs, the face, the respiratory muscles and the glottis, and the treatment consisted in artificial respiration (Bragg-Paul), artificial feeding, and heroic doses of vitamin B_1. Whether the latter had any effect it is impossible to say. Recovery took several months.—EDITOR, *I. M. G.*]

DETERMINATION OF BLOOD GROUPS FROM MEALS OF BLOOD-SUCKING INSECTS

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The material.—Mosquitoes and bed bugs were fed on subjects of known blood groups and squashed on chemically pure filter-paper. The stains so obtained were dried.

The insects were bred from eggs in the Entomological Department of the School of Tropical Medicine. The mosquitoes were fed once only and then squashed, some after 3 hours and others after 24 hours. The bed bugs were fed for several days until a suitable size was reached. Then, like the first batch of mosquitoes, they were squashed 3 hours after a feed. The difference between the squashing in the two cases was accidental, not intentional or significant in any way.

The method and results.—The technique described by the writers for the determination of blood groups from stains was followed (Greval, Bhattacharji and Das, 1943). Briefly, (*i*) the MDESA (minimal dose of equal and simultaneous agglutination) of a serum ab (from a subject O) was determined—and found to be 1 in 16*; (*ii*) 25 milligrams of the stained filter-paper were left in contact for half an hour at blood heat and overnight at ice-box temperature with 0.1 c.c. of the serum dilution containing 3 MDESA in a unit volume—3 in 16 dilution, and (*iii*) the serum dilution after contact was removed by centrifuging, and was tested with 2 per cent suspensions of known rbc A and rbc B for the loss of isonins a and b.

The serum dilution absorbed with the stain obtained by squashing a mosquito fed on subject A had lost a; the dilution absorbed with the stain obtained by squashing a mosquito fed on subject B had lost b; the dilution absorbed with the stain obtained by squashing a mosquito fed on subject AB had lost both a and b; and the dilution absorbed with the stain obtained by squashing a mosquito fed on subject O lost neither a nor b.

The stains obtained by squashing bed bugs were tested in the same way with the same results.

Medico-legal implications.—Mosquitoes and bed bugs may suck blood from a person of one blood group and on being squashed on articles of another person of another blood group may create a false evidence against the latter.

Insects are known to pass blood from the anus just after commencing a feed. The droplets so passed and deposited can be over a millimetre in diameter and give positive chemical, spectroscopical and serological tests. They may yield enough material for grouping tests to be done by those who pride themselves on getting results from microscopical quantities. For medico-legal purposes, however, such acts of supererogation (Sutherland, 1910) cannot be commended (Greval, 1940).

Professional keepers of leeches empty the latter, after application and engorgement, by pricking them near the posterior end and then milking. The blood of a certain group may thus drop accidentally or be squirted intentionally on the articles of another person of another group and again create a false evidence.

Even lice crushed between the thumb nails (the usual method of destruction in India) may

*The volume remaining constant the dose is expressed by dilution.