our experience with regard to paralysis of any part of the body. The largest number of recoveries took place during the first three months after onset—at a time (as it happened) when little physical treatment was available. A second though much smaller peak in the recovery rate, which occurred during the 9–12 months period, may have been due in part to the influence of treatment. Although the part played by physiotherapy seems to be small, in that no known treatment can bring about regeneration of grossly damaged anterior horn cells, the prevention of contractures and the maintenance of joint mobility are of great value. When recovery is slow and imperfect the importance of muscle training is especially noteworthy. It is therefore only in the early and complete recoveries that physical treatment seems superfluous.

The care of the victims of this epidemic was and still is the work of a team. In addition to those whose names appear in the text we express our thanks to Prof. A. V. Bernard; to the resident medical staff of St. Luke's Hospital, especially to Dr. O. Azzopardi and Dr. V. Zammit-Tabona; to the physiotherapists, Mrs. K. J. Bell, Miss E. J. Charter, Miss P. Empson, Mrs. M. T. Ensoll, and Miss M. B. Quinton; and to the Reverend Mother Superior and her devoted staff of nurses.

REFERENCE

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ACUTE ANTERIOR POLIOMYELITIS AMONG SERVICE PERSONNEL IN MALTA

AN ACCOUNT OF AN EPIDEMIC

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An account is given of the clinical pathological, and therapeutic findings in a series of 57 cases of acute anterior poliomyelitis occurring among Service personnel during the Malta epidemic of the winter of 1942–3. The epidemiological findings of the outbreak have been fully discussed elsewhere by Seddon, Agius, Bernstein, and Tunbridge (1945). Only 4 out of a total of over 400 cases were recorded among the adult Maltese population and none at all among the Maltese troops. The apparent immunity of the Maltese troops is the more striking because they were often working side by side with United Kingdom troops.

Of the 57 cases 2 were from the Royal Navy, 27 from the Army, and 28 from the Royal Air Force. The incidence was proportionately higher among the R.A.F. than among the Army personnel, being 4 to 1. The duration of service in Malta bore no relation to the disease. The average duration among affected personnel was over 12 months (Table I). The four cases with

TABLE I

Duration of stay in Malta:		Period of service:	
1 month and under	4	Minimum	2 weeks
1 to 6 months	7	Maximum	5½ years
7 " 12 "	4	Average	Over 12 months
13 " 24 "	31		
Over 2 years	11		

less than one month's service in the island arrived after the onset of the epidemic. The average duration of the disease before admission to hospital was 3.6 days, with a range from 12 hours to 15 days.

The Preparalytic Stage

In only two instances was information regarding the preparalytic stage not available. In two further cases no paresis developed, and in five additional cases the exact duration was difficult to assess as there was in each instance a history of illness followed by a period of normality lasting 3 to 12 days before the return of symptoms and the onset of paralysis. The average duration of the preparalytic stage in the remaining 48 cases was 3.4 days (Table II).

The proportion of non-paralytic cases was unusually low, and undoubtedly some cases were missed. On the other hand, the medical officers and all the troops were aware of the prevalence of anterior poliomyelitis, with the result that almost all those complaining of headache and fever during the peak period of the epidemic had a routine examination of the cerebrospinal fluid on admission to hospital. By this procedure the diagnosis was established in the two recorded non-paralytic cases.

TABLE II.—Duration of Preparalytic Period

12 cases 24 hours or less	6 cases 4 days
6 " 2 days	7 " 5–7 days
11 " 3 "	6 " 8 days or more
(Average 3.4 days)	
5 cases with uncertain preparalytic period	
(2 or 10, 5 or 14, 3 or 15, 1 or 4, 3 or 10 days);	
2 cases, no paresis; 2 cases, data not available	

Symptoms.—In 36 cases the onset was sudden and dramatic, the patient presenting some of the symptoms and signs of meningeal irritation. Headache was the most prominent initial symptom, and was recorded in 45 cases (Table III). No new

TABLE III.—Preparalytic Symptoms in 53 Cases

Headache	45	Anorexia	8
Muscle weakness	26	Vomiting	6
Pain in back	21	Lassitude	6
" neck	14	Urinary symptoms	5
" limbs	13	Abdominal pain	4
Malaise	14	Dysphagia	3
Sore throat and coryza	13	Weakness of eyes	2
Shivering attack	11	Miscellaneous	5

symptoms were observed, nor was there a constant clinical picture applicable to the majority of cases.

Signs.—Pyrexia was noted in 43 cases on admission to hospital, and ranged from 98.8 to 104° F. The fever lasted from 48 hours up to eight days, save in six cases in which the development of secondary infection, respiratory or urinary, prevented an accurate assessment of the duration of pyrexia attributable to the primary disease. Neck rigidity was observed in 20 cases, neck stiffness in 17, Kernig's sign and the spine sign in 12.

The Paralytic Stage

Twenty-six patients complained of weakness of a limb or other part of the body before admission. Muscle tenderness was noted in 30 cases, but in only six did it present with any degree of severity longer than 10 days after the onset of the paralysis. The degree of muscle tenderness bore no relation to the severity of the muscle paresis. In the present series the degree of paralysis was by no means maximal within 24 hours of onset (Table IV).

TABLE IV.—Cases with Delayed Spread of Paresis

Cases		Cases	
2 days	2	6 days	1
3 "	3	9 "	1
4 "	3	10 "	1
5 "	2		
(In 8 cases delayed 4 days and over)			

The spread of the paralysis was descending in 10 and ascending in 3 cases. In 15 cases the development of the paralysis was not continuous, but was of the "jumping" variety, with an interval of at least 12 hours between the jumps. Included in the jumping cases were nine of the descending and three of the ascending type of spread. Accurate assessment of the extent and the degree of paralysis in the initial stages was often a matter of considerable difficulty. In our experience neither the degree of loss of power nor the degree of alteration of the reflexes was of itself an adequate guide to the severity. Some cases with absence of reflexes in the early stages made a dramatic recovery of power, and vice versa. Further, despite repeated and careful examination, muscles apparently unaffected initially, or even after 10 days, showed definite weakness three to five weeks later. The latter feature is of considerable clinical significance and necessitated keeping the patients in bed for at least four, preferably six, weeks. However trivial the initial paralysis may seem, it is only by keeping the patient in bed for such a period that the extent of the paralysis can be fully assessed and the development of deformity minimized.

Case 14.—Sgt. H., aged 21. Admitted to hospital with a two-days history of headache and pain in the neck. The next day he developed weakness of the muscles of the right shoulder, mainly of the right deltoid. Cerebrospinal fluid: 88 cells per c.mm. (poly-

morphonuclears); protein, 50 mg. per 100 c.cm. No further muscle weakness was recorded during the first three weeks of the illness. Allowed up after one month, when he complained of weakness of the legs. Examination showed weakness of the abdominal, hip flexor, and quadriceps muscles of both legs.

The final distribution of the paralysis for all patients is given in Table V.

TABLE V.—*Distribution of Paralysis*

Cranial nerves:						
Alone	..	..	..	..	3	14 (24.5%)
With others	..	..	..	..	11	
Respiratory system (Fatal cases, 11)	..	..	..	..	16	(28%)
Arms:						
Alone	..	..	..	..	7	35 (61%)
With others	..	..	..	..	28	
Legs:						
Alone	..	..	..	..	3	38 (66.7%)
With others	..	..	..	..	35	
Trunk:						
Alone	..	..	..	..	2	28 (49%)
With others	..	..	..	..	26	
Sphincters:						
Bladder	..	..	..	..	10	12 (21%)
Bladder and rectum	..	..	..	..	2	

Respiratory paralysis was observed in 16 cases. The initial symptom was an inability to take a deep breath, and was always complained of by the patient before the detection of any weakness of the respiratory muscles on physical examination. The earliest sign was a weakness of the cough reflex. In at least four cases death was due to central rather than peripheral failure. Patient No. 6 died six and a half months after the onset of the disease, and during the last 12 hours of life had a series of convulsions, each of some minutes' duration, and completely unaffected by the continuous use of the Drinker respirator; necropsy showed no evidence of cerebral abscess or embolus. Analysis of the cases revealed that the later the development of respiratory symptoms after the onset of the disease the greater the prospect of recovery.

Cranial Nerves.—An unusual feature was the presence of nystagmus in 10 cases. In the non-fatal cases the nystagmus rapidly disappeared and was not present after six weeks. In two of the early cases, although nystagmus was observed and recorded, it was not then considered, to be due to the virus infection—e.g., Case 26, with diagnostic C.S.F. findings, had nystagmus and a mild degree of weakness of the dorsiflexors of the right ankle and slight diminution of the right ankle-jerk as the only abnormal physical signs. The following cranial nerves were involved: 7th (3 cases), 4th and 6th (2 cases), 12th (1 case). Three patients complained of dysphagia and one case of diplopia. In the civilian cases there was also a high incidence of cranial nerve lesions—11% had facial palsy.

Sphincters.—Retention of urine occurred in 12 cases. In eight the retention was of short duration, lasting only from 36 hours to six days. Suprapubic cystostomy was performed in one of the remaining cases; catheterization was necessary in a further two, and in the remaining case hesitancy still persisted after three months. Two cases had incontinence of faeces in addition to retention of urine.

Recovery.—The rate and degree of recovery varied considerably. Owing to the exigencies of the Service it was impossible to record the final disposal of every case, but in all save seven it was possible to assess the degree of paralysis after three months. Table VI classifies the cases according to the severity of the paralysis.

TABLE VI.—*Severity of Cases: Review Three Months after Onset*

	Non-paralytic	Slight	Moderate	Severe	Fatal	Total
Royal Navy ..	—	2	—	—	—	2
Army ..	2	6	—	11*	6	27
Royal Air Force ..	—	6	2	9	5	28
Total ..	2	14	10	20	11	57

* Two severe cases are likely to prove fatal within one year.
Non-paralytic = Positive symptoms and C.S.F. findings; no paralysis.
Slight = Mild paralysis; fit for full duty within three months.
Moderate = Definite paralysis, but ultimately should have no serious disability.
Severe = Cases likely to have permanent disability.

Diagnosis

During the epidemic cases of infective hepatitis, undulant fever, enteric fever, fibrositis, infective neuritis, sciatica, pleurisy, coryza, disseminated sclerosis, diphtheritic polyneuritis, cerebral

abscess, and hysteria were sent to hospital labelled anterior poliomyelitis. By contrast one or two cases were missed, and were referred to hospital only when the paralysis was fully established. The cerebrospinal fluid findings, together with the clinical history and the distribution of the paralysis, were adequate for establishing the diagnosis. During an epidemic of acute anterior poliomyelitis in a subtropical climate great difficulty may be experienced initially in differentiating between sandfly fever, benign lymphocytic meningitis, and a non-paralytic case. The high percentage of lymphocytes on initial examination of the cerebrospinal fluid in the present series of cases suggests that it is necessary to exercise caution in making a differential diagnosis in the above conditions purely from the cytological examination of the cerebrospinal fluid.

Once paralysis is established difficulty may arise in a mild sporadic case in differentiating between muscular dystrophies, localized neuritis of shoulder-girdle distribution (Spillane, 1943), and acute anterior poliomyelitis.

Three cases were of special interest in that the diagnosis was a matter of considerable difficulty.

Case 15.—Pte. S., aged 26. Admitted to hospital for dental treatment while convalescent from enteric fever. Five days after admission he developed bronchitis, and five days later weakness of the right serratus anterior was observed. The diagnosis of anterior poliomyelitis was discussed, but was considered unlikely by the physician in charge and by the surgical specialist. Lumbar puncture was not performed. A plaster abduction splint was applied, and while in the splint the patient complained of weakness of the right elbow and wrist. Removal of the plaster after three weeks revealed weakness of both serratus anterior muscles, the spinati, right triceps, and extensor muscles of right wrist. Prof. H. J. Seddon examined the case and agreed finally with the diagnosis of anterior poliomyelitis.

Case 55.—P/O. T., aged 23. Contracted syphilis seven months before admission. He was having antisyphilitic treatment, and had received nine injections of arsenic. He piloted his plane two days before admission, and the following evening complained of weakness of the right leg; a diagnosis of transverse myelitis, ? syphilitic, was considered. The next day, on admission to hospital, flaccid paralysis of the right leg and weakness of the left leg, with no sensory changes, were observed, and the cerebrospinal fluid showed 170 cells (lymphocytes and monocytes) per c.mm. and 200 mg. protein per 100 c.cm.; second day in hospital, paralysis of both legs and weakness of lower abdominal muscles; third day, paralysis of the left half of the diaphragm (blood Kahn ++); fourth day, all abdominal muscles and left half of chest paralysed, and he was placed in a Drinker apparatus; fifth day, paralysis of both sides of diaphragm. Death occurred on the seventh day. Necropsy revealed oedema of the brain with flattening of the gyri, no naked-eye evidence of haemorrhages into the spinal cord, congestion of the lungs, right-sided heart failure, and slight enlargement of the spleen. The micro-anatomy of the nervous tissues is not yet available. There was no microscopical evidence of any syphilitic changes.

Pte. C., aged 27.—On admission, after a history of one day's pain in the limbs and in the neck, this case presented the physical signs of a transverse myelitis, the upper level of the sensory loss being D9. Wasting of the whole of the erector spinae muscles and of the muscles of the left arm, with a distribution typical of anterior poliomyelitis, developed after admission. The blood Kahn reaction was repeatedly negative and there was no history or clinical or pathological evidence of a syphilitic infection. Brig. D. MacAlpine at first thought the case might be one of anterior poliomyelitis, but in view of the progress of the patient it was later diagnosed as transverse myelitis, and is not included in the present series.

Pathology

Cerebrospinal Fluid.—Lumbar puncture was performed within 24 hours of admission, and in 24 cases was repeated at least once after 7 to 10 days. In 21 cases lumbar puncture was performed on at least three occasions—on admission, at 7 to 10 days, and again 14 to 21 days after the onset of the disease. No definite relation existed between the cell count, the protein content, and the severity of the disease. The cell count was high in the majority of cases. The cells were mainly lymphocytes and monocytes, but in a few instances the polymorphonuclears predominated. In the 11 fatal cases the count upon admission ranged from 100 to 900 cells per c.mm. On the other hand, two severe cases had initially a count of only 40 cells per c.mm. In the series only three cases had a normal cell count upon admission, and in one of these the protein content was raised. In the two non-paralytic cases the cell counts were 112 and 200 respectively—lymphocytes and monocytes pre-

dominating. Repeated examination of the cerebrospinal fluid confirmed the findings of previous workers that the cell count steadily falls, reaching normal limits in 10 to 21 days, whereas the protein content remains raised or continues to rise for 2 to 4 weeks. In a few instances the protein content fell to within normal values after 10 to 13 days, then rose again. This suggests that the finding of a normal cerebrospinal fluid between the 10th and 14th days in a suspected case of acute anterior poliomyelitis does not necessarily exclude the diagnosis (see Graph).

Graph illustrating the fall of the cerebrospinal fluid protein content and the cell count to within approximately normal limits between the 10th and 14th days of the disease in certain cases.

Leucocyte Count.—Examination of the white-cell count was not made as a routine, and the data available were too limited to permit of any conclusions.

Post-mortem Examination

Necropsies were made in all fatal cases—11 adults and 1 child; the latter is excluded from this report. In nine cases death occurred during the acute stage of the illness, and in the remaining two cases after four and a half and six and a half months respectively from the onset of the disease. The spinal cords and brains from four cases were preserved intact and sent to the Central Laboratory, G.H.Q., Cairo, for micro-anatomical and virus examination. Faeces and urine from one fatal case were also sent. In the nine cases in which death occurred during the acute stage the brain was described as oedematous or congested, and the gyri as flattened. Medullary haemorrhages were reported in six cases, pontine and mid-brain haemorrhages in three.

Cardiovascular system: Right-sided dilatation of the heart and congestion were reported in eight cases. Lungs: Congestion was reported in seven cases, collapse in three, bronchopneumonia in three, and pneumonic consolidation with acute abscess formation in one case. The spleen was enlarged and friable in five cases. The liver and kidneys were stated to be congested in five cases. Tardieu spots were reported in four. The principal finding in the two patients dying after the acute stage was right-sided heart failure. In one the lungs were healthy, and in the other there were mild bronchopneumonic changes. The brains showed no macroscopic evidence of congestion, oedema, or haemorrhage.

Treatment

Ward Management.—All doubtful cases were admitted to one ward, where full precautions were taken. The staff wore masks and gowns; the patients had individual crockery, and all faeces and urine were disinfected. Once the diagnosis was established the cases were transferred to another ward. A portion of the latter was set aside for the treatment of cases with respiratory paralysis. At the end of 10 days all cases not requiring the use of the Drinker apparatus were transferred to a convalescent ward, where no further precautions were taken concerning the possible spread of the disease.

Serum Therapy.—Serum was not used in the early cases, the work of the Rockefeller Institute and the Australian Commis-

sions having led us to believe that its use was of little value. Serum, however, was forwarded from the Middle East, and 13 cases received convalescent serum in doses varying from 20 to 150 c.cm. The dosage used was 10 c.cm. intravenously and 40 c.cm. intramuscularly daily for three days. No serum reactions were observed.

Sulphadiazine.—In addition to the serum 10 cases received sulphadiazine. The dosage advised by Brig. MacAlpine was 3 g. twice daily for four days and then once daily for two days. The 3-g. dose was dissolved in 10 c.cm. of distilled water and injected slowly over a period of three minutes. Of the 10 cases only three had the full course. Four cases developed frank haematuria after the administration of 9 to 12 g.; one case had red cells in the urine after 18 g.; one case developed severe vomiting after 12 g.; the remaining patient had to be placed in the iron lung, and died before the completion of the course. Of the 10 patients receiving both serum and sulphadiazine two died. The number of cases treated was too small to be of statistical significance, and owing to the development of complications only three had a complete course. No dramatic improvement seemed to follow the use of this therapy, but, in view of the long preparalytic period in some cases, we feel that it is worthy of a more extended trial.

Respiratory Paralysis.—The serious therapeutic problem during the early stages was the treatment of respiratory paralysis. In these stages of the epidemic sufficient mechanical respirators were not available and respiration was maintained manually. None of the recognized methods of artificial respiration (Schäfer, Silvester) was tolerated by the patients. The only effective method was rapid pressure and release (30+ per minute), applied centrally with the palm of the hand on either side of the lower end of the sternum. One of us (R. E. T.), on observing this, reprimanded the orderly for using an unsound method, as he considered that any benefit derived from such a procedure must be psychological rather than physiological. Further experience suggested that this supposition was untrue, for the unorthodox method did relieve cyanosis, which the orthodox methods failed to do because they distressed the patient. We consider that the reason for the anomaly was the painful character of all movement of the chest on account of the tenderness of the affected muscles.

Through the generosity of the civil Government three iron lungs of the Nuffield pattern were lent to the Army. Lieut.-Col. Tanner, R.E., constructed three respirators of the Drinker type, incorporating several minor modifications which made for greater efficiency. Several Bragg-Paul respirators were sent to the island upon the instructions of the War Office. As a result of the experience gained in the epidemic we found that the Drinker type of artificial respirator did relieve cyanosis—even when all other methods had failed—induced physiological respiration, and gave the patient comfort and confidence, whereas little benefit was derived from the use of the Bragg-Paul type, either in the acute stages or in the treatment of cases still requiring the use of artificial respirators after three months. Oxygen was administered in the cyanotic cases, the B.L.B. apparatus being used.

Sepsis.—Respiratory and urinary sepsis were treated with full courses of sulphapyridine and sulphathiazole. For the lung infections postural drainage was attempted, but in the acute stages of the disease proved too distressing to the patient. The local treatment for infection of the bladder consisted of alternate washouts with oxycyanide of mercury (1 in 10,000) and boric acid solutions.

Muscular Paralysis.—The problem from an orthopaedic standpoint consisted in retaining the muscles in a neutral position during the acute stage, in providing varying forms of splintage to prevent stretching of the paralysed muscles and contracture of their antagonists, and in giving suitable remedial treatment in the convalescent stage pending evacuation of the cases from the island.

Acute Stage.—The neutral position adopted was the supine, with a low pillow just raising the shoulders to avoid stretching the abdominal muscles (as in many cases these particular muscles were affected), but not high enough to stretch the paravertebral groups. The head was similarly supported. The upper limbs were placed in the hips-firm position, the fingers resting lightly on the abdomen just above the iliac

crests, with a reasonable degree of abduction of the shoulders maintained by means of sandbags. The legs were placed together and extended, with the feet supported at a right-angle with a foot-board or with short back splints. Behind the knees a low pillow was placed, giving about 10 degrees of flexion—a position in which there is very little risk of stretching the quadriceps and which avoids the graver danger of the development of genu recurvatum. The legs and feet were further protected with a bed cradle. This position was assumed by all patients except those whose respiratory paralysis necessitated the use of an iron lung, in which provision for the best posture for the arms is absent. An iron lung of the Drinker pattern constructed locally was furnished with an adjustable foot-rack, and a second one was projected in which abduction of the arms could have been accomplished, but the epidemic subsided before the machine was completed. It is noteworthy that all patients who had been treated in the iron lung either developed contractures in the pectoral groups of muscles or experienced discomfort in them during the passive abduction. The neutral position was maintained until the pain and tenderness had gone and the convalescent stage was reached.

Treatment in the Convalescent Stage

All the muscles of each patient were investigated methodically, and as nearly as possible in the clinical examination the power of each individual muscle was assessed and classified according to the M.R.C. nomenclature at 7-day to 10-day intervals. The majority of cases showed paralysed muscles or muscle groups, occurring in all limbs and the trunk indiscriminately. All the cases in which the trunk and lower limbs were affected were maintained in the neutral position, and, where possible, were permitted use of the arm—either unsupported, if the muscles had escaped paralysis, or suitably splinted according to the muscle groups involved.

A notable feature of many cases was paralysis of the abdominal muscles, including the lateral abdominals, the asymmetrical affection of which is so potent in producing a gross scoliosis unless preventive measures are undertaken. Two such cases, in which there was no respiratory paralysis, were immobilized in plaster jackets preparatory to evacuation from the island. Plaster beds would have been preferable, as physiotherapy could have been continued, but they are difficult to adapt for air travel. No patients with lower-limb weakness were allowed to walk save where such weakness was confined to the anterior tibial group, and for these a short walking calliper with drop-foot springs was supplied.

In some patients the paralysis was confined to muscles of the shoulder girdle, and these were given abduction splints. One patient whose main weakness showed in the rhomboids derived considerable benefit from wearing a figure-of-8 bandage, applied as in the treatment of a fractured clavicle. Arm weakness in the patient kept in the supine position on account of trunk and lower-limb paralysis was countered by maintaining abduction at the shoulder with light metal trough splints or plaster splints slung to a transverse bar at the head of the bed. Wrists were supported by short cock-up splints combined, in cases showing paralysis of the intrinsic muscles of the hand, with a knuckle-duster attachment; where the long finger extensors were affected the splint was continued to finger-length. If the thumb was also affected the digit was held into the palm to avoid stretching the muscles of opposition. In one patient the intrinsic muscles of the foot were involved and the toes began to assume a clawed position. For this deformity a pair of wooden sandals were supplied with four slits cut out at the front end, and the proximal phalanges of the toes were bandaged to the intervening prongs.

No limb was completely immobilized, and during the convalescent period light massage and appropriate active exercises were given daily. No active movements were carried out until muscle tenderness had completely subsided, and only such passive movements were used as were unavoidable during nursing. Once active movements had been started particular care was taken to stop them short of fatigue.

All the patients, with the exception of those who required treatment in the iron lung, carried out gymnastic exercises for all muscle groups in warmed water in a specially constructed seven-feet-long pool (funnel-shaped to provide the maximum of room for exercise in the minimum of water in order to

economize in fuel for heating and to reduce the rate of heat loss) furnished with rigid head-rests and a supporting pelvic sling on a crane. These water exercises were performed three times weekly, and were very popular with the patients because they seemed to be effortless, a rough idea of progress could be measured by the patients themselves, and the much greater excursion of voluntary movement while in the water was psychologically stimulating.

For those patients who had respiratory paralysis—and in all of them the paralysis of the remainder of the trunk and the limbs was widespread—a suspension frame for the carrying out of active exercises was constructed to imitate the effect of a swimming-pool so far as was possible. The upper limbs were supported on a light metal trough, with troughs for the upper arm and forearm, leaving the shoulder and elbow free, while the wrist was supported by chaining wooden hand-grips to the forearm troughs. These troughs were suspended from the frame by selected cords, attached to the frame by springs made of coiled piano wire. Resistance to the movement was minimal, and the springs afforded considerable resilience to such movements as could be executed. An extension of the frame enabled the lower limbs to be treated similarly, and the active pressing down of the lower limb against the supporting springs proved to be particularly good for exercising the gluteal muscles. In one case a further addition to the frame was a hand-pulley device for exercising the feet by pulling down the hinged foot-pieces of the lower-leg troughs against the counter-springs.

Discussion

The most striking feature of the epidemic was the almost complete absence of cases among the adult Maltese population. This fact, coupled with the high incidence among Maltese infants, would be compatible with the view that the strain of virus responsible for the epidemic was one for which the adult Maltese had some degree of immunity, but not the adults from the United Kingdom. The above facts are also of interest in the elucidation of the origin of the epidemic. Before the outbreak Malta had been besieged, and contacts with the outside world were limited to the movements of a small number of Service personnel. As a result the question of the epidemic having been introduced by the Service had to be considered. Prof. Seddon, after a careful analysis of all the cases, concluded that there was no evidence to support the view that the epidemic was introduced by Service personnel.

The case incidence was high—approximately 150 per 100,000—but the mortality rate was not abnormally high either for the epidemic as a whole or for the adult cases. The unusual clinical features were the high incidence of cranial nerve lesions in both the civilian and the Service cases and the presence of nystagmus in 10 of the latter (17%). Further, in view of the recent controversy concerning the early treatment of acute anterior poliomyelitis, the frequent finding of selective muscle weakness as late as the fourth week after onset suggests that the age of the subject should be taken into consideration before generalizations concerning therapy are made. As a result of the experience gained in the epidemic described, we consider that for an adult case a minimum period of six weeks' absolute rest is advisable.

Treatment of the patient with respiratory paralysis raised many problems. Of the 16 cases with respiratory paralysis 13 were treated in a mechanical respirator. Of the 13 so treated 10 died, and two further cases have such a serious degree of paralysis that they are likely to succumb to a secondary respiratory infection within two years. The distressing appearance of such patients often leads the physician to make frantic demands for a mechanical respirator on misguided humanitarian grounds. In a considerable proportion of cases, more particularly those in which the paralysis begins within five days of the onset of the disease, the respiratory failure is central in origin and no form of artificial respiration is of avail. Morphine, used judiciously, would seem to be the treatment of choice for such cases.

The mechanical respirator, however, has a value in the treatment of cases in which the paralysis is peripheral. In our experience the later the onset of the respiratory paralysis the more likely was it to be peripheral in distribution. In the treatment of respiratory paralysis two varieties of mechanical respirator were used—the Drinker cabinet respirator and the

Bragg-Paul pulsator. In our series the former proved to be the only effective method of artificial respiration. We attribute the failure of the Bragg-Paul pulsator to: (a) the limited area of application, which prevents the machine from completely dominating the respiratory action; (b) the tenderness of the affected muscles, which induces spasm; (c) inability of the machine, when it cannot dominate respiration, to adapt itself to the constantly varying respiratory rhythm; (d) the faulty principle of the pulsator, in that inspiration is not purely passive; (e) the noise made by the bellows, which is such that the patient tends to expire when he should inspire. Although the Drinker respirator was of value, the constant use of the apparatus required the continuous attention of skilled mechanics and an unfailing supply of electricity. Further, since cases with respiratory paralysis often had widespread muscle involvement the long-continued use of the ordinary type of cabinet respirator prohibited the treatment necessary for prevention of contractures of the upper limbs.

In view of the above facts and the difficulty of transporting artificial respirators it would seem advisable to centralize treatment. It is our considered opinion that for a small outbreak it would be preferable to transfer patients in the initial stages, if necessary by air, to a centre adequately supplied with respirators and full physiotherapeutic facilities rather than to transport respirators.

Summary

The clinical and pathological findings and the early treatment of a series of 57 adult cases of anterior poliomyelitis occurring among Service personnel during the Malta epidemic 1942-3 are described.

There were 11 deaths—a mortality rate of approximately 19%.

In 2 cases no paralysis was noted. In 26 (45%) cases there was no serious final disability.

In 14 (24%) cases the cranial nerves were involved; in 16 cases there was respiratory paralysis; and all the deaths resulted from respiratory paralysis, despite the use of a mechanical respirator of the Drinker cabinet pattern; in 12 (21%) cases there was disturbance of the sphincters.

Convalescent serum and sulphadiazine were used in a small number of cases without any apparent benefit.

The onset of the paralysis was often delayed, and in a few instances was not apparent until the fifth week.

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THE ERYTHROCYTE SEDIMENTATION RATE IN INFECTIVE HEPATITIS*

BY

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In recent years a number of references to the erythrocyte sedimentation rate in infective hepatitis have been published. Ziegler (1941) found that a rise in the E.S.R. often occurred in children in the second or third week after the onset of icterus. Penman (1943), reporting hepatitis in tuberculosis patients in a sanatorium, stated that in about one-half of a series of patients the E.S.R. was markedly slowed when the jaundice was at its worst, but had returned to approximately the pre-jaundice level by the next monthly examination. Any change for the better or worse could not be attributed to the tuberculous process. More recently Wood (1945) gave figures for a small series of cases in the Mediterranean area showing readings below 10 mm. (Wintrobe) in 85% in the first 10 days of the disease and later rising.

In the present investigation the E.S.R. has in 80 cases of infective hepatitis been followed throughout their stay in hospital, and observations on a few other cases seen in the pre-icteric phase are reported. Wintrobe tubes were used and the mixed ammonium-potassium oxalate anticoagulant of Wintrobe and Landsberg (1935). Corrections were made for variations in the volume of packed erythrocytes, using the chart published by Whitby and Hynes (1938).

In 40 of the 80 cases the E.S.R. was raised above 10 mm. at some stage of the disease, and it was noticed that in these cases the E.S.R. was low in the acute stage except in a few very mild cases, and then became raised as the patient recovered. Figs. 1 and 2 are graphs in which the bilirubin level and E.S.R. are shown throughout the course of the disease in two severe cases. No relation between the maximum height of the E.S.R. and the clinical severity of the case could be found.

Two Service cases were seen in the pre-icteric phase, and both these had raised sedimentation rates. One of them developed jaundice in hospital, and as his jaundice increased his E.S.R. fell, to rise again to a maximum of 29 mm. at the time when the serum bilirubin was about 1.5 mg. per 100 c.cm. (Fig. 3). Six further pre-icteric cases had sedimentation rates measured in the pre-icteric phase. In four of these the rate was raised. Two of this last group were tested again after they became jaundiced: one, who had become moderately jaundiced, showed a normal E.S.R. in the icteric phase; the other, with only conjunctival icterus, did not show any fall to a normal E.S.R. Of the six cases with an increased rate in the pre-icteric phase, two were raised above 30 mm. in one hour. In five cases the E.S.R. was raised at the time of admission. In one of these the highest recorded bilirubin was 2.3 mg. per 100 c.cm. at the time of admission, and his sedimentation rate of 35 mm. on the same day was the highest recorded.

Particulars of the case of Bdr. C., who was admitted in the pre-icteric phase, are shown in Fig. 3. His bilirubin never rose above 4.8 mg. per 100 c.cm., and his lowest recorded sedimentation rate was 12 mm. in one hour. The other three cases were also clinically mild, having 3, 5.75, and 8.5 mg. per 100 c.cm. as their highest bilirubin, and their E.S.R. continued to rise while the bilirubin was falling.

The mean sedimentation rate of all readings at various bilirubin levels has been estimated in those cases which had a raised E.S.R. at some stage in the disease, and the results are shown in Table I. It can be seen that in this series there is a

TABLE I

Serum Bilirubin in mg./100 c.cm.	Number of Readings	Mean E.S.R. (mm. in 1 hour)
10+	20	5.30
8-1-10	18	8.39
6-1-8	12	8.00
4-1-6	18	10.56
2-1-4	29	14.31
0-2	34	18.94

rise in the E.S.R. as the serum bilirubin falls. The coefficient of correlation is -0.57. The highest recorded E.S.R. in the cases showing some rise, after correction for differences in haematocrit, varied between 11 and 42 mm. in one hour.

The existence of a raised sedimentation rate at least as often in the pre-icteric as in the post-icteric phase in this series, and the very marked negative correlation between the serum bilirubin and the E.S.R., suggest strongly that the normal E.S.R. in the icteric phase is due to some factor inhibiting sedimentation. On the other hand, the sedimentation rate is not so closely bound to the bilirubin figure as to suggest that the normal figure for the E.S.R. in the icteric phase is entirely due to the raised bilirubin (see, for instance, Fig. 3). The following experiments were therefore carried out to discover whether the most obvious abnormalities in infective hepatitis—increased amounts of bile salts and bilirubin in the plasma—were capable of inhibiting the raised E.S.R. in patients with tuberculosis, rheumatic fever, and infective arthritis, or those convalescent from infective hepatitis.

Methods

Oxalated blood was taken, using a minimal amount of Wintrobe's ammonium-potassium oxalate mixture, and cells

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