A British Medical Association Lecture ON THE TREATMENT OF INFANTILE PARALYSIS.

DELIVERED BEFORE THE MONMOUTHSHIRE DIVISION,
DECEMBER 10TH, 1926,

BY

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THERE is no disease more productive of permanent disability in children than infantile paralysis, but fortunately much crippling can be prevented by well guided early treatment. The malady is always among us in a sporadic form, but it is many years since we have had an epidemic comparable to that which has occurred recently at Broadstairs and Leicester. Although infantile paralysis has been recognized for nearly a century, yet most of our knowledge has been accumulated during the last thirty years, mainly through experience gained when the disease has assumed an epidemic form in various countries. In 1905 it spread all over Sweden, and since 1907 there have been periodical epidemics in the United States of America.

Poliomyelitis attacks children in all grades of society and is not necessarily associated with poor housing conditions. There is strong reason for thinking that it is a disease disseminated from person to person, and although the causal agent is not known it is believed to be a minute filterable organism¹ invading the body by the mucous membrane of the nasopharynx. Passing through the mucous membrane the poison gains the blood stream by way of the lymphatics, and, spreading widely, produces a general infection.² The main onslaught, however, is directed against the central nervous system, and the motor nerve cells of the grey matter in particular. These last suffer from a threefold attack²—namely, mechanical pressure due to oedema and cellular infiltration of the surrounding nervous tissue; anaemia through rupture or constriction of their arterioles; and intoxication due to the direct action of the virus. The site, extent, and virulence of the attack determine the prognosis. Bulbar lesions account for many of the fatal cases; in lesions of the cord alone, which are much commoner, the mortality is comparatively low. Recovery of the motor nerve cells and the disappearance of the paralysis will depend upon whether the initial attack causes complete cellular destruction, in which case the paralysis is absolute and irrecoverable, or whether the cells, having survived the first onslaught, are only temporarily unable to function through persistent pressure from haemorrhagic exudate and oedema, the gradual absorption of which brings relief. Should the exudate, however, become organized and replaced by fibro-cicatricial tissue, then some cells may remain permanently functionless from scar pressure. Although there is no means of determining the nature of the pathological lesion and forecasting the ultimate prognosis, yet it can be said that there is in most patients a strong tendency towards spontaneous recovery.

The symptoms of onset, apart from the paralysis, resemble those of other acute specific fevers. The incubation period is probably from four to ten days, during which there may be some sore throat. The onset is sudden, accompanied by fever up to 103° F., sweating, vomiting, sometimes diarrhoea, and general nervous irritability. One or more limbs are paralysed and flaccid. There are various clinical types, which are classified by Müller as spinal, bulbar, cerebral, and abortive. The bulbar form, as already mentioned, presents an unfavourable outlook owing to possible involvement of vital centres. Several fatal cases of this type have been reported by Feiling,³ Collinson,⁴ and others. The cerebral form (polio-encephalitis) may be confused with meningitis, and being an upper motor neurone lesion is characterized by spastic paralysis. The abortive form gives rise to an interesting group, and was first recognized by Caverly⁵ in the Vermont epidemics. Many of the acute initial symptoms are present but paralysis is absent. This form occurs frequently during an

epidemic, but owing to the absence of paralysis is apt to be missed, and thus a patient of this type may become an unchecked carrier. If doubt exists, a lumbar puncture will help to clear the diagnosis. The cerebro-spinal fluid will show white cells from a hundred upwards in each cubic millimetre, three-quarters of them being polymorphonuclear. Organisms are absent and the glucose and chloride content are both normal.

The spinal type is the commonest paralytic form, and the following remarks apply mainly to this group of cases. There are three clinical periods of the disease—the acute, the quiescent, and the chronic. The acute stage is ushered in by the symptoms indicated. The limbs are painful and tender to touch, and this may last with declining severity for six weeks. It should be added, however, that frequently in the sporadic case no history of an acute onset is obtained; the child goes to bed apparently well, and the next morning the mother finds one limb powerless and hardly any other symptom to suggest an acute infection. Muirhead Little,⁶ in a careful investigation of 115 cases, describes the sudden appearance of paralysis in 43, and in 19 of these the paralysis was the first symptom to attract attention. With the passing of muscular tenderness the second or quiescent stage begins, and with it spontaneous recovery of the muscles. Improvement continues for approximately two years. When recovery comes to a standstill the third or chronic stage has arrived.

TREATMENT.

The virus is present in the mucous membrane of the nasopharynx of infected persons, and therefore contacts should cleanse their throats by gargling three times a day with a 1 in 4,000 solution of potassium permanganate.

First Stage.

The patient should be isolated for four weeks and all the excreta disinfected. In the pre-paralytic stage the intraspinal injection of 5 to 20 c.cm. of human immune serum, obtained from patients who have recovered from the disease, has been advocated, and many cases have been so treated in America and New Zealand. The practical difficulties in providing serum are obvious, although the municipal authority of Melbourne⁷ succeeds in obtaining and storing it for emergency. In this stage, too, urotropine⁸ has its greatest value. The objection to it is its tendency to produce bladder irritation, but this can be obviated and large doses tolerated by keeping the urine alkaline with sodium bicarbonate and potassium citrate, as recently suggested by Hurst.⁹ Urotropine is of doubtful value when the disease is well established.

The pain and tenderness suggest that rest in bed is the best treatment in this stage. The joints should be kept in a neutral position to prevent deformities arising and to relax opposing muscles as much as possible. The child should lie on a flat bed with the thighs slightly abducted, the knees straight, and the feet at right angles to the legs. Sandbags and properly arranged towels will help in maintaining this position. It is a great advantage, however, to put the limbs in splints or plaster gutter casts. A pair of long moulded splints extending from just below the buttock to the back of the heel and then along the sole of the foot will keep the knee and ankle joints in the correct position. A flat bed will help in preventing scoliosis developing if there is paralysis of the spinal muscles on one side. If an upper limb is paralysed the arm should be abducted, the forearm flexed and supinated, and the hand slightly dorsiflexed. These positions of the limbs are the best in the early stages and where it is not yet clear what muscle groups are most affected, but where it is obvious that some muscles are paralysed and their opponents have escaped, then it is well to follow the rule laid down many years ago by Sir Robert Jones¹⁰ and allow greater relaxation of the weakened group—for example, in a drop-foot paralysis with the tendo Achillis unaffected, the foot should be dorsiflexed slightly beyond a right angle and the splint adapted accordingly. No other treatment is necessary during this stage. Massage and electrical treatment of these tender muscles are to be strongly deprecated. They add to the discomfort of the patient and may do infinite harm. But this should be

explained to the parents, who will need reassuring that, having splinted the limbs, the best service to the child is to stand and wait in the early weeks.

Second Stage.

When tenderness has disappeared from the muscles, which is usually somewhere about the sixth week, more active treatment is undertaken. Accompanying the paralysis there are trophic disturbances such as local impairment of the circulation. The muscles are flabby, and the limb is cold and has a blue tinge. The aim of treatment should be to maintain the nutrition of the muscles until restoration of function is established. This is best attained by keeping the limb warm, applying gentle massage and the galvanic current. But it should be clearly understood that neither massage nor electricity has a direct influence on recovery. The most that can be said for either is that they keep the muscles in a state prepared to receive impulses. The splints should continue to be worn and great care taken, during the massage, that the paralysed muscles are not stretched. After two or three weeks of massage the child should begin to be propped up on a bed-rest, and later may be allowed to sit in a chair propped up with pillows. All this is preparatory to getting the child on its feet. By this time the muscles will have shown indications of recovery. Even a small contraction in a muscle can be felt by fingers placed on its tendon, and once a voluntary contraction occurs much can be done to hasten further recovery by graduated exercises. But in doing this great care is necessary to avoid overloading the muscle—for example, if the foot dorsiflexors are paralysed it will not do to let the foot drop and expect the recovering muscle to lift it. The foot should be supported almost to right angles and the patient then asked to dorsiflex. In all these exercises, which are valuable in restoring bulk and tone to the muscles, the burden must be accommodated to the strength of the muscle.

The time when a patient may be allowed to stand will vary with each individual case, but in many with lower limb paralysis it will be from three months upwards from the onset of the disease. If the paralysis has disappeared walking should be undertaken by stages, but fatigue avoided. If some paralysis remains, then walking appliances will be necessary, depending upon the disability—for example, with paralysed quadriceps, peronei, and foot dorsiflexors, a calliper with uplifting toe-spring and outside T-strap and wedge on the boot will keep the knee straight, the foot dorsiflexed and everted at the ankle. By this means all the paralysed muscle groups are kept relaxed. The splints first prescribed should still be worn during the night and the graduated exercises and massage continued. At the end of the year recovery slows down considerably, but it may still go on for yet another year. It is very unlikely that any improvement will take place after two years in a patient who has been continuously under well directed treatment from the beginning. But during this time every care should be taken to improve the general health, for this helps in the recovery. Fresh air and sunshine are useful adjuncts in this respect. Children in the second stage treated at the Royal National Orthopaedic Hospital at Brockley Hill react favourably to graduated sun exposure, but whether it is due to the increased metabolism thereby produced is difficult to say.

Third Stage.

When, in spite of treatment, recovery has ceased and the condition has remained stationary for some months, then the last or chronic stage has been reached. The residual paralysis then represents the extent of permanent disability, but two or three years at least from the onset should be allowed to elapse in a well treated case before this disability is accepted as incapable of further non-operative improvement. This, of course, applies only to cases where deformity has been prevented and where the disability is essentially a paralytic one. Where, however, as frequently happens in the neglected case, deformity is present, this should first be corrected and the overstretched paralysed muscles given some months to recover with the joint in a favourable position.

In the lower limb the commonest deformities needing

correction are flexion of the hip and knee, and plantar flexion at the ankle. In flexion of the hip the tightened tensor fascia femoris usually prevents correction, and is best dealt with by Soutter's¹¹ operation. A longitudinal incision three inches long is made in the long axis of the body with its centre two inches posterior to the anterior superior iliac spine. The tensor fascia femoris is exposed and cut transversely. The muscles and fascia are lifted subperiosteally by an osteotome from either surface of the anterior superior iliac spine. The muscles are pushed back leaving the bare spine projecting, which is then removed by an osteotome. The hip is hyperextended and the wound closed. The patient is nursed for six weeks on half mattresses extending from the head to the buttocks, allowing the hip to be hyperextended, or a plaster-of-Paris cast may be applied from the chest to the ankle. Flexion at the knee can be overcome by extension in a bed calliper, but in intractable cases open elongation of the hamstrings may be necessary. An equinus deformity due to the contraction of the tendo Achillis can often be overcome by dorsiflexing the foot under an anaesthetic, but if this fails open elongation of the tendon should be undertaken, allowing the foot to be brought just up to a right angle with the leg. A varus deformity usually responds to manipulation. Splinting, massage, and graduated exercises should follow the correction of deformity to see if any recovery in the affected muscles takes place. If this happens, the treatment should continue until improvement ceases. But before considering further measures it is worth while allowing this type of case also to be up and walking, properly splinted, for some months.

After sufficient time has elapsed to show that further recovery from the paralysis is unlikely, and where any deformity present has been corrected, there still remain means by which function can be improved, although the paralysis is permanent. Each case presents its own peculiar problem, and therefore principles of treatment only can be mentioned here.

The object of operative treatment is to produce joint stability and dispense with the necessity of wearing an appliance. The methods which find most favour for this purpose at the present time are arthrodesis, tendon transplantation, and tendon fixation, and sometimes two of these methods may be combined.

Arthrodesis was introduced by Albert of Vienna in 1878, when he deliberately ankylosed a flail knee, but now the only common site for the operation is the tarsus; a flail shoulder is occasionally stiffened, but only where there are good scapular muscles to ensure later good movement of the combined humerus and scapula. Arthrodesis of the knee and ankle are seldom performed. For a flail foot where instability is in front of or below the ankle, at the astragalo-scapoid and astragalo-calcaneal joints, the so-called triple arthrodesis is valuable. The astragalus, os calcis, cuboid, and scaphoid are converted into one bone by the removal of their opposing articular cartilages, thus allowing ankylosis to take place by encasing the foot in plaster-of-Paris for eight weeks. But this operation is preferably done at the age of 12 years or over, when the tarsal bones are well ossified. Where the instability is at the ankle-joint, or in cases where the tendo Achillis and tibial muscles are paralysed, producing a calcaneo-valgus deformity of the foot on standing, the best result is obtained by Whitman's operation,¹² which consists of performing astragalectomy and pushing the foot backwards until the fibula overlaps the calcaneo-cuboid joint; the foot is held in a slight equinus position and encased in plaster-of-Paris in this position for three months. A boot with a long heel part to the upper will be required afterwards. One of the cases shown here to-day had a Whitman's operation performed on the left foot for a severe paralytic calcaneo-valgus deformity and a triple arthrodesis for a right flail foot. The patient now walks quite well without appliances, notwithstanding that the right quadriceps is very weak.

Tendon transplantation was first introduced in 1881 by Nicoladoni. Operating upon a case of paralytic calcaneus, he cut the peronei and implanted their central ends into the tendo Achillis. The success of tendon transplantation depends upon the muscle selected for transference and the

manner in which it is grafted. The chosen tendon should be a normal one of good power—a weak muscle is useless. The tendon should be brought down along a gradual path to its new insertion; a sudden twist or turn in its course is fatal to its working efficiency. Where possible it should be inserted into bone. One of the most successful transplantations is the transference of the tibialis anticus to an insertion into the cuboid to act as an evertor of the foot where the peronei are paralysed. [In one of the cases shown the peroneus brevis had been grafted into the tibialis posticus to aid inversion of the foot in paralytic valgus.] The transplanted tendon should be relieved of all strain for at least two months, and if the appliance has been worn this should be continued for a little while longer.

Tendon fixation originated in 1898 with Tilanus of Amsterdam when operating upon a girl with paralytic equino-varus. The tendo Achillis was cut subcutaneously, the extensor proprius hallucis was then cut by open section and the central end attached to the extensor longus digitorum, and the peripheral end to the periosteum of the tibia. Codivilla extended the method further, and latterly Gallie¹³ of Toronto has brought it prominently forward. The paralysed tendons are converted into ligaments to correct the disability¹⁴—for example, in paralytic varus the paralysed peronei are permanently fixed to the external malleolus so as to check the deformity. Two grooves are gouged—one on the anterior and the other on the posterior aspect of the external malleolus. The tendons are pulled taut, correcting the deformity, and are buried in these grooves, the longus in front and the brevis behind. The periosteum is brought over and sewn to the tendons, and the foot is encased in plaster-of-Paris for two months. The cast is then removed and the patient allowed to walk without an appliance.

In some cases the paralysis is so extensive that an instrument is a permanent necessity, for no operation is likely to succeed in dispensing with it. One child seen here to-day had not walked for four years owing to complete paralysis of the lower limbs with fixed hip and knee flexion. Soutter's operation was performed on each hip and the knees were straightened by extension. This enabled her to be fitted with callipers and so to walk about. There are exceedingly few cases so badly paralysed that they cannot be made to walk. Even if three or four limbs are paralysed, provided there remains sufficient power in the hands to grasp a pair of crutches, walking is still possible by means of artificial support.

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THE TREATMENT OF CONGENITAL SPASTIC PARAPLEGIA BY SYMPATHETIC RAMISECTION.

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DR. N. D. ROYLE has contributed to the *Medical Journal of Australia* the full text of a paper on the treatment of congenital spastic paraplegia by sympathetic ramisection. By arrangement with the editor of our contemporary the following abstract appears simultaneously here.

It had occurred to Dr. Royle that in spastic paralysis the resistance offered to the moving group of muscles by its antagonists was due to a fault in reciprocal innervation; and when, subsequently, the discovery was made that the

voluntary muscles have a dual innervation, he carried out a series of experiments on goats to ascertain whether any deductions could be drawn as to the part played in the causation of the spasticity by the sympathetic element in this dual innervation. The results were published in the *Medical Journal of Australia* in January, 1924.

EXPERIMENTAL.

It was found from these experiments that animals from which the left abdominal sympathetic trunk had been removed could walk well after a short interval, but exhibited the following peculiarities. When the animals were placed on the back both lower limbs were at first held extended, but after an interval the left limb gradually fell into a more flexed and abducted position than the right. The relaxation time of the left knee-jerk was found to be much shorter than that of the right; the relaxation and extension times were, on the operated side, approximately equal, and the whole movement was more quickly performed than on the right side. In addition, resistance to passive flexion and extension was greater on the normal side. These differences were more evident when the animal was placed under light anaesthesia; and when partially anaesthetized the animals were unable to support weight on the limb from which the sympathetic had been removed. The ability to maintain posture voluntarily in the operated limb, however, was retained, and only disappeared under anaesthesia. If the operation were performed on a decerebrate animal similar changes were observed; and further, the extension of the limbs peculiar to the decerebrate condition, although usually present, was not well maintained on the operated side.

In order to obtain independent testimony as to the accuracy of his conclusions, Dr. Royle called in the aid of an experienced neurologist, Dr. Campbell, who examined one of the goats operated on and fully confirmed Dr. Royle's observations and added several details to those mentioned above. He noted that the muscles of the operated limb were softer, the relative lack of tension being particularly noticeable on pressing the tendo Achillis against the bone; further, although the difference in the knee-jerks was as Dr. Royle had described, the least degree of excitement in the animal immediately caused them to become similar in character on the two sides. Some investigators have failed to verify Dr. Royle's results, but it is to be noted that they have experimented on small animals only, in which the changes are less readily detected than in goats; the results, however, have recently been corroborated by Kuntz and Kerper, who experimented on dogs.

THE FIRST CASE.

These experiments confirmed Dr. Royle in his opinion that the sympathetic system was responsible for the occurrence of spasticity, and he considered it justifiable to apply the operation in the human subject. An apparently suitable case came under his notice in September, 1923.

The patient, a man aged 30, had received a gunshot wound seven years before. The bullet, entering above the left ear, caused extensive laceration in the region of the left central sulcus, and the wound extended across the middle line to the opposite hemisphere. The patient was unable to speak for three months, and unable to walk for two years, after the injury. On examination there was found to be right spastic hemiplegia affecting both limbs, with spasticity in the left leg and foot. On the right side the knee-jerk and ankle-jerk were exaggerated and there was patellar clonus; on the left side there were exaggerated ankle-jerk and ankle clonus. The Babinski reaction was present in both limbs. The patient could walk, but had great difficulty in controlling the right leg, and when bearing on it exhibited a coarse, uncontrollable tremor. He could walk forwards, but not laterally or backwards. Occasional tonic fits occurred, but were readily controlled. The genito-urinary system was normal, but the patient suffered from chronic constipation.

This patient exhibited a change for the better within six hours after the operation. While the right foot immediately fell into plantar flexion after being passively dorsiflexed, the return in the left foot was slow and gradual. The clonus on the right side became progressively less, while persisting on the left. There was an immediate improvement in voluntary movement; the range increased and muscular control was more effective; the patient was able to support himself on the right leg, an impossibility before the operation, and he could walk readily both backwards and forwards; there was also an improvement in balance, and the tremor affecting the right leg disappeared. The limbs showed an increase in size and power, and resistance to fatigue gradually became greater. The chronic constipation was permanently relieved.