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POLIOMYELITIS

THE EARLY TREATMENT AND PROBLEMS

by

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It is now ten years since our country has joined the circle of afflicted countries with a growing wave of cases from year to year. Until the epidemic of 1950, this country had a low endemic rate, with average of two cases per month. Already in 1949, the number of cases rose, indicating the change-over from a sporadic disease to an epidemic one, and since then the annual number of cases has been as follows :

Year	No. of cases
1950	1621
1951	918
1952	874
1953	636
1954	785
1955	468
1956	533
1957	57
1958	636
1950-1958	6528

We note that in the year of the first outbreak the incidence was 146 per 100,000. In subsequent years, the annual rate never dropped to lower than thirty per 100,000 except for 1957, which was a year with an expectionally low morbidity in our country and in the world in general. As for age distribution, Israel resembles the neighbouring countries in having a "primitive" pattern,

with eighty per cent of afflicted children under the age of three years. The disease is thus truly "infantile paralysis" with all its attendant problems.

The great outbreak found us insufficiently prepared, concerning both, clinical understanding and therapeutic equipment. This explains certain inexactitudes - as for example the question of non-paralytic cases. Compared to the confidence in making this diagnosis in earlier years, we have learnt in time (as other clinicians have done) that it is easy to overlook minor paresis, and to classify these cases as non-paralytic. This may explain the discrepancies, and wide amplitude present in reports of different epidemics. In a survey of the epidemic in Jerusalem in 1953, it is stated that a thorough examination revealed, that the number of muscles involved in individual cases ranged from 6 to 145, not including facial muscles (an average of thirty-six per case). In a casual examination, or because of inexperience, assessment, especial in mild cases, may be incorrect, and an unjustified optimistic conclusion may be reached. It is worthy of note, that with a young infant (and most of our cases belong to this category) there exists an unsurmountable difficulty in distinguishing between the inability and the unwillingness of the infant to move a certain limb or muscle.

Another difficulty: - In early years the diagnosis was only clinical. In recent years, the co-ordination of clinical and laboratory findings has shown that not a few cases clinically considered as poliomyelitis, have been shown by laboratory investigations to be due to some other virus - either unidentified, or from another family e.g. Coxsakie. Although from the therapeutic point of view, this may make no difference, from the scientific view-point and the future preventive approach, an exact diagnosis is desirable.

Cases of poliomyelitis are hospitalized essentially in two centres in Israel; in the north, in the Rambam Government Hospital, and in the south, at Tel-Hashomer Government Hospital.

Our clinical experience is based on approximately 1,600 cases treated by us at the Government Hospital Bnei-Brak during 1952-1953, and since the summer of 1953 at Tel-Hashomer Hospital. The 1950 epidemic in the south and north, and also the 1953 outbreak in Jerusalem has been described in the local medical literature.

In clear-cut cases we have avoided carrying out a lumbar puncture, since we feel that in many cases it is not of diagnostic assistance, and in addition we consider the puncture as a possible traumatic factor, which is poliomyelitis, it is desirable to avoid as much as possible. This tendency of avoiding trauma and disturbance has found its expression in the concept of sleep above any other activity. The sleep of the child with poliomyelitis, especially in

the acute stage, warrants postponing any activity which is non-essential e.g. feeding, administration of drugs, laboratory procedures, and even examination of the child during the daily ward-round. This attitude to the value of sleep, we feel, is of benefit to the patient, and has a therapeutic significance.

The spinal form of poliomyelitis has not confronted us with any special problems. Obviously, high fever is treated with drugs as required (but not by injection), but no other therapeutic efforts are usually required. After the fever has dropped, and spasm or muscle tenderness, requiring hot packs and rest, is absent, passive movements are commenced. In many cases we have seen muscle atrophy develop within a week, and consider it justifiable to commence early passive movements, to prevent atrophy as far as possible.

Three forms of poliomyelitis threaten the patients' life - one which allows but a stygian outlook by the doctor (apart from routine activities) and two others in which the medical attitude is active and decisive.

The first is polio-encephalitis in which active intervention is of a very modest degree. We exclude completely from this classification cases of facial palsy, and include only cases where the centre of the clinical picture is occupied by hyperpyrexia, disturbed consciousness, sometimes coma, central paralysis, akinetic mutism, etc. There have been cases where deep sleep continued for a week, with a subsequent full return to consciousness. Treatment in these cases did not create any severe problems, as nutrition was maintained either by parenteral infusion, or gastric tube (standard or polythylene).

The other two forms, which sometimes demand maximum therapeutic effort, are respiratory muscle paralysis, and bulbar paralysis. Often these two forms are combined, which increases the difficulties, both as to diagnosis and treatment. Since we have these problems in very small infants (in contrast to other countries) we have not been able to receive detailed instructions, from the overseas literature, and we have been forced to forge our own therapeutic path; undoubtedly also making mistakes as we went along.

On admission of a child with poliomyelitis, we pay special attention to the state of respiration, even in a straight spinal case. We pay special attention, if deltoid involvement is present. With one deltoid paralysed, co-existing diaphragmatic weakness may be present, but with both deltoids paralysed, diaphragmatic weakness is certain to be present, due to the overlapping innervation of deltoid and diaphragm from the cervical cord.

In mild respiratory muscle paralysis our treatment is to give antibiotics and oxygen, either with a small tent, which contains the infant's head, or by nasal catheter. On starting this treatment, it is essential to keep a constant

watch on the child. The first sign of worsening condition indicates that oxygen is insufficient, and more active measure, i.e. the Iron-Lung is required. To decide when to put a child into the iron-lung is not easy. The use of counting-up to ten etc., as used in adults is impracticable in infants. Clinical signs that we depend upon include shallow breathing, sometimes jerky, with the expanding alae nasi, mild facial pallor with an expression of fear, sweat appearing on the forehead. The combined picture, and not any individual sign caused us to decide that the moment for putting the iron-lug into action had arrived. With this, the attitude has developed that it is inadvisable to wait too long, and certainly not to wait until the final evidence of anoxia, i.e. cyanosis has become manifest. Putting the child in the respirator at a late stage, when he is exhausted and anoxic, worsens the chances for recovery. We, therefore, feel that the indication for putting a child into the iron lung exists from the moment we commence to weigh up whether it is advisable to use the respirator or not. Since we have put cases in early, before they are "in extremis" and before irreversible anoxic damage may have occurred, we have had better results. We have never considered the "advisability" of this step, even if the child is completely paralysed. This question is a vexed one, and we have not permitted ourselves to make the ultimate decision, although well aware of the comments of Walford and others, that "patients may be saved who would be better dead".

During the first two years, all difficult and complex cases were put in the iron-lung, without any fine clinical diagnosis. However, afterwards a change for the better in our diagnostic approach developed. The iron-lung is suitable for the child with the "dry" form of respiratory paralysis (Lassen). The presence of secretions in the pharynx ("wet" paralysis) is a contra indication to using the iron lung for the treatment of respiratory paralysis.

The first evidence as to the correctness of putting the child into the iron lung and the efficiency of this step, is in many cases the settling-down of the child and his falling asleep. A child who does not feel well in the lung does not fall asleep nor does he become sedated in the lung. The change in the habitus of the small patient from restlessness and fear to repose and sleep, is prominent and convincing. The harmonic co-ordination between the rhythm of the machine's action and respiratory rate of the child is important. We have noted that an increase in the amplitude is more important than stepping-up the rate of the machine.

With the entry of the child into the iron-lung, a dangerous feeling arises, that "everything" has therefore been done for the child. The monotonous noise of the machine at work sedates not only the patient who

requires it, but also dulls the diagnostic-therapeutic consciousness of the practitioner. It is necessary to know this, in order to avoid costly mistakes or neglect.

The child in the iron lung requires constant care and attention. The disease may progress even when the child is in the lung, and a "dry" child may become "wet", thus denying the validity of the continual therapeutic use of the machine. Also peripheral paralyses may progress leading on to deformities and contractures. Thus careful observation and early treatment is advisable, e.g. correct posturing, change of posture, passive movements, etc., while the child is still in the lung. Special care is required with feeding. The prone position is preferred, in order to avoid aspiration. If the child is in supine position, nursing attention is required for some period after feedings. We have seen one case like this with fatal outcome, and it is vital to avoid "accidents" such as these.

While in the iron lung the child may require special treatment such as intravenous infusion, antibiotics, oxygen. A slight increase of pharyngeal secretion requires suction and the use of the "Trendelenburg position".

Sometimes we have seen the child grow so accustomed to the machine, as to achieve a "symbiosis" with consequent difficulty in weaning. Even a child who is not dependent on the machine, finds it difficult to do without it. Sudden stoppage of the machine may result in grimaces and anxiety, or crying, whereas opening window of the machine (cancelling the pressure effect) while in action, may have no effect on the good condition of the child. The cuirass (Monaghan) respirator intended as stepping-stone to weaning did not give encouraging results in infants although older children managed to do with it. Weaning is usually possible in gradual stages, with longer intervals between use of the machine, although here too there is no definite pattern. Sometimes after a child has been out of the machine, a second period of use may be required for a few days.

Altogether in 1953 - 1958, 120 children were put into the iron-lung at Tel-Hashomer Hospital, with a sixty-one per cent mortality. In the first great epidemic the mortality for the north was sixty-nine per cent. (forty-two children)and in the south eighty per cent. (seventy children). On an average every tenth child required the use of the iron-lung.

In 1955, we began to be aware of the "defects" in this treatment. The child with "wet respiratory" paralysis or bulbo-respiratory paralysis, became worse within the lung and results were no satisfactory. We noted the increasing harm done by the "uncompromising effort of the tank respirator".

With increasing mortality, it was necessary to find a more efficient mode of therapy for this type of case. Pure bulbar cases were treated with tracheotomy and suction.

Usually the swallowing reflex returned within a week or two. Feeding was either intravenous or by gastric tube. In one case a girl of eight years learnt to carry out suction by herself. In mixed bulbar and respiratory paralysis, this treatment is ineffective and within the respirator, with absence of swallowing or cough reflex, pharyngeal secretions are sucked into the chest with each air entry, causing the patient to "drown in his secretions". Being thus aware of the limitations of respirator and tracheotomy respectively, we commenced positive pressure respiration (I.P.P.R.) in the summer of 1956. The matter was not easy. Although aware of the interesting example of Lassen, little experience with infants had been recorded. Of 345 severe cases in Copenhagen in 1952 only six children were under the age of a year. We began this therapy with no experience, little experience and a necessity to improvise. Altogether we have treated thirty-three cases with this therapy with a mortality of seventy-two per cent. We feel that the difficulties in treatment are greater in the infant than in the adult. The tracheotomy was done with local anaesthesia and bronchoscopic cover. A standard silver cannula was used and the leak of air around the cannula was not considered a disadvantage (preventing too great a rise in the inspiratory pressure, and allowing for further removal of CO_2). The bag ventilation apparatus found in surgical theatres was used. Either air or oxygen was used according to need. When the number of children requiring therapy at the same time rose to seven, special arrangements were made. Two compressors were installed, and "air on top" was made available for each child, allowing a generous supply of air to a number of children, without being dependent on outside balloons, which involved a financial outlay and technical difficulties.

To overcome the problem of supplying the necessary humidity to the air stream, improvisation was resorted to. The air was passed through a polyethylene tube, two metres long, containing a number of holes, which was folded up inside a bottle of water. The bottle was kept in a bucket of hot water. Despite these measures we encountered thickened bronchial secretions. We had to resort in these cases to lavage by injecting 1-2cc of saline solution through the broncheostoma, followed by a few positive pressure respirations, and afterwards to "squeezing and tapping" of the chest.

Manual ventilation was used with a short inspiratory phase, and prolonged expiration. Respiratory rate - thirty per minute. This work was carried out by untrained staff, which included some of the parents of the children

and medical students from the Hebrew University, Jerusalem. Our experience was, that untrained personnel including even parents, are capable of doing good and responsible work.

We used the Engstrom Respirator, but our impression was that manual ventilation was more flexible, with a better individual adaptation to the needs of the child.

Nutrition was maintained by intravenous infusion and afterwards by an indwelling polythylene gastric tube. In one child, aged two years, mother herself daily inserted the gastric tube.

Chest physiotherapy, frequent suction, Trendelenburg position as required, antibiotic administration, laboratory procedures, including biochemical investigation (blood and urine) and bacterial cultures (nose, throat, trachea), as well as regular follow-up, x-ray examinations, were included in the medical attention of the children.

The period of positive pressure ventilation ranged between 1 and 138 days, with an average of forty days.

Our experience made us convinced that this type of therapy is essential for the "wet respiratory" or bulbar respiratory cases. Its use does require neither expensive nor a lot of equipment. The apparatus is maintained in readiness in the ward, and can be put into use within seconds of the tracheotomy.

We have described the "life threatening" forms of the disease which demand an active therapeutic approach. We have omitted describing the full clinical picture, which is pleomorphic, and justifies considering the disease as a systemic one, and not confined to the nervous system.

Increasing experience reveals new facets, requiring increased awareness, and a broadening of the diagnostic acumen. The improved and deepening viral "work-up" allow for more precise clinical definition of the paralytic episode, and the revealing of other etiologies in poliomyelitis-like disorders.

We have also found that prognosis is almost impossible during the acute stage of the disease, especially when fever is still present. We have seen dramatic changes in both directions within a few hours, and feel that in all cases it is advisable to give the parents an extremely guarded prognosis.

Isolation procedures carried out by us, have been extremely modest consisting of hand-washing and wearing a gown. The children were treated in separate rooms, but in adjacent rooms (with a common corridor) children with other diseases were hospitalized. The therapeutic staff was the same for the whole ward, and passed freely from one room to the next. <u>During all the years, not a single case of contagion was seen in the ward.</u>

Nine years after the outbreak of the first epidemic in this country, and two and a half years after nation-wide Salk vaccination programme, marked drop in the morbidity of poliomyelitis has been noted. We are optimistic about the future, but at the same time consolidating the clinical experience acquired in the treatment of this disease and which perhaps may still be required, we trust, but for few cases in the future.