DIAGNOSIS AND TREATMENT OF ACUTE ANTERIOR POLIOMYELITIS

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The diagnosis and treatment of acute anterior poliomyelitis is a matter of concern to every clinician, for the earliest indications of the disease may be non-specific, and dangerous symptoms may develop rapidly. When this occurs treatment is life-saving, but incorrect treatment only adds to the patient's dangers.

Diagnosis

Minor Illness. In about 40 per cent. of paralytic cases the 'minor illness' is the first symptom of poliomyelitis. It is characterized by malaise, fever, sore throat, headache and catarrh, and usually lasts about 48 hours. It is absent in more than half the paralytic cases, and when present it is so non-specific that it can only be diagnosed in retrospect. The suggestion has been made that this stage represents a period of viraemia, but the evidence is conflicting.

Major Illness. The major illness develops three to ten days after the minor illness, and in the interval the patient usually considers himself perfectly fit, although occasionally the minor illness merges indeterminately into the major illness. In 90 per cent. of cases the major illness develops abruptly. It begins with the *meningitic phase*, characterized by fever, malaise, headache, vomiting and pain in the neck, spine and limbs. Catarrh is rare at this stage, and meningitic symptoms prominent, the neck being stiff and flexion painful. Nystagmus may occur. The cerebrospinal fluid (C.S.F.) though very rarely normal, almost always contains excess cells, varying from about 20 to 500 per c.mm. The majority of the cells are usually polymorphs, and occasionally as much as 80 per cent. polymorphs are found. The polymorphs are replaced within a few days by lymphocytes, which themselves disappear soon after the fever subsides. The protein at first is raised only slightly, and considerably less than in pyogenic meningitis, with a similar cellular reaction, but as the cells disappear the protein rises to 100 or 200 mg. per 100 ml., or higher, and may remain elevated for several

weeks. The glucose content of the C.S.F. is normal throughout. Neither the severity of the meningitic symptoms, nor the degree of pleocytosis in the C.S.F., give a guide to the severity of paralysis that may follow.

The aching in the limbs may be severe or it may be so mild that the patient may be inclined to 'work off his rheumatism' by vigorous exercise with disastrous results. Though the severity of the pain does not enable a forecast to be made about the paralysis to be expected its site gives some indication, for paralysis is more likely to develop where the pain is felt. Muscular fasciculation similar to that in motor neurone disease may be observed, and this is a diagnostic sign and indication that the muscles showing fasciculation will shortly become weak.

The meningitic phase and the minor illness (if it occurs) constitute the whole illness in non-paralytic cases.* Such cases can in general only be diagnosed on probabilities from the nature of the illness and the presence of poliomyelitis in the population. New laboratory procedures, however, are being developed, which make it possible both to demonstrate complement-fixing antibody and to grow the virus on tissue culture from faeces or sputum. Poliomyelitis virus has been cultured from 98 per cent. of paralytic cases, but only from 42 per cent. of cases diagnosed clinically as non-paralytic poliomyelitis. It now seems reasonably certain that a large proportion of the latter were not infected by poliomyelitis virus at all (Weller, 1954).

The paralytic phase of the disease usually begins at the time when the fever and symptoms of the meningitic phase are abating (Fig. 1). There may be a delay of 12 or even 24 hours after the fever subsides before the onset of paralysis, and it is important that the patient continues to rest in bed during this period, even though he feels

* 'Non-paralytic' cases must be distinguished from 'abortive' cases, in which the virus multiplies in the body and is excreted in the stools, but in which symptoms are either absent or non-specific.

FIG. 1.—Diagram to show various patterns which the disease may take (from Russell, 1952).

completely recovered. It is an unfavourable sign if low-grade fever continues or fever recurs after the onset of paralysis, for further paralysis may then occur. This is unlikely when the temperature has been normal for 24 hours. It is unusual for paralysis to spread for more than four or five days, and very exceptional for it to do so more than a week from the onset of paralysis.

The paralysis may affect any of the voluntary musculature of the body, and it is particularly important to recognize immediately the onset of the paralysis which may endanger life, that is involvement of the muscles of respiration or of swallowing. If serious *weakness of the primary muscles of respiration* develops, the respiratory rate rises, the patient becomes restless and anxious and when speaking pauses for breath every few words; the alae nasae move with respiration, and the accessory muscles of respiration, if not themselves paralysed, are brought into use. If the respiratory centre is affected, a similar picture is presented with, in addition, irregularities of respiratory rate and volume. These signs indicate an already serious state of affairs, and if possible a more accurate estimate of the progress of events should be obtained by measuring the vital capacity with a spirometer or, more conveniently, with a dry gas meter.* This should

*e.g. Type C.D. 1 Meter (Parkinson and Cowan Industrial Products, Ltd., London).

TABLE I
VITAL CAPACITIES IN NORMAL SUBJECTS

Weight (lb.)	Vital Capacity (litres)
35	1.3
70	1.8
105	3.0
140	4.2
175	4.6

Approximate ventilation required for patients of different weights. The minute volume should be increased 10 per cent. when the patient is awake and 5 per cent. for every 1° F. of fever (adapted from E. P. Radford).

be done repeatedly for any patient who has severe paralysis of the upper limbs or who is suspected of respiratory weakness, and if the vital capacity falls to one third or one quarter of the normal (Table I), artificial respiration is likely to be required. If no spirometer is available, the patient should be made to take a deep breath and count aloud as far as possible. The normal person can count considerably more than 50, and if 15 is all that can be achieved in a single breath, artificial respiration will probably be necessary.

Paralysis of swallowing, often loosely referred to by the more general term 'bulbar paralysis,' is as dangerous as weakness of respiratory muscles, but the two conditions must be clearly distinguished since the treatment is entirely different. The danger lies in the risk of pulmonary damage due to inhalation of secretions, food, or most disastrous of all, vomit. Weakness of swallowing may be obvious to patient and observers, or manifest itself as choking over food or medicine. Development of a nasal voice may herald difficulty in swallowing. Children, however, may refuse to take food if they know they cannot swallow it, and if this is dismissed as anorexia, a most significant observation is missed. A patient with a pool of secretion in the pharynx may find that in order to prevent it running into his trachea he has to breathe shallowly and rapidly. If this increase in respiratory rate is attributed wrongly to weakness of respiratory muscles, the patient may be put into a tank respiratory, and this will inevitably lead to the inhalation of the secretions with disastrous results.

Investigations

The principle investigation in a case of suspected poliomyelitis is lumbar puncture, and the C.S.F. changes have already been described. Controversy flares up from time to time whether lumbar puncture is indicated in poliomyelitis. There is

no evidence that lumbar puncture is harmful, and the general principle should be applied that an investigation should be done if, and only if, it is likely to provide useful information. In the meningitic phase the diagnosis of poliomyelitis is always presumptive, and but for the existence of controversy it would seem hardly necessary to stress that lumbar puncture may be essential to distinguish between poliomyelitis and bacterial meningitis, and that the early diagnosis of bacterial meningitis is of the first importance. On the other hand, when paralysis has developed, the diagnosis may be so clear that lumbar puncture would be pointless.

Differential Diagnosis

The minor illness is indistinguishable except in retrospect from other non-specific fevers. The meningitic phase of the major illness has to be distinguished from other causes of meningitis. The insidious onset of tuberculous meningitis and the rapidity with which drowsiness and disorientation follows on frank symptoms of meningitis contrasts with the abrupt onset of the meningitic phase of poliomyelitis and the remarkable preservation of consciousness in that disease. If doubt exists, search should be made for lesions in the chest, tubercles in the retina and acid-fast bacilli in the C.S.F. When polymorphs predominate in the C.S.F. the distinction from pyogenic meningitis is difficult. A source of infection in the chest or middle ear must be sought and the C.S.F. thoroughly examined for bacteria. Diminution in the glucose content of the C.S.F. is much in favour of pyogenic meningitis. Benign lymphocytic chorio-meningitis may only be distinguishable with the aid of complement-fixation tests. Mumps meningitis is rarely confusing unless the parotitis has been minimal. Acute toxic polyneuritis is distinguished clinically by the symmetrical nature of the paralysis and by the presence of sensory loss which may be predominantly posterior column in type. It is further distinguished by the high protein content of the C.S.F. without increase in the cells, but this distinction is lost one or two weeks after the onset since in poliomyelitis the cells disappear and the protein rises at this stage. Other diseases which have been confused with poliomyelitis include neurosyphilis, brain abscess, intracranial and spinal tumour, cerebral haemorrhage, pneumonia, influenza, rheumatoid arthritis, rheumatic fever, scurvy and hysteria (Blattner, 1954).

Treatment

Meningitic Phase

During the meningitic phase it is probable that the fate of the lower motor neurones hangs in the

balance, and therefore anything which increases their susceptibility to the virus must be avoided. It has been shown (Russell, 1949, Horstmann, 1950) that exercise during the meningitic phase increases the probability of severe paralysis in the exercised limbs, and therefore during an epidemic of poliomyelitis anyone suffering from symptoms suggestive of the meningitic phase should rest in bed until the fever and symptoms have subsided for 24 hours. In deciding whether a patient must rest or not, meningitic symptoms and pains in the back and limbs are especially significant, whereas catarrh in the absence of those symptoms is very rarely an indication of poliomyelitis.

Paralytic Phase

Treatment in the paralytic stage has two distinct objects, first the preservation of life, and second the prevention of deformity.

Preservation of Life. Deaths due to poliomyelitis are almost all due to respiratory failure or pulmonary complications, but the mechanism of these disorders varies, and since the treatment differs widely according to the nature of the trouble, it is essential to distinguish clearly between them.

In primary respiratory failure the intercostal muscles and diaphragm are weak, or the respiratory centre fails to perform its rhythmical function, and in either case, adequate ventilation is not achieved. Artificial respiration in a tank type of respirator is then required. The patient must not be left until he is grossly underventilated and cyanosed before he is put into the respirator. When his vital capacity has fallen to one third or one quarter of normal, he should be told that at this vital time it is necessary to rest his muscles and that therefore a machine will be used to breath for him. He can then be put into the respirator without hurry, and his co-operation can be obtained both in making him comfortable and in synchronizing his own respiration with that of the machine. A confused and anoxic patient on the other hand, may exhaust his remaining strength struggling against the machine.

When a patient first goes into a respirator it is wise to give him some deep respirations to show him that good reserves are available and to ensure that any accumulation of carbon dioxide is eliminated. Prolonged hyperventilation should, however, be avoided, since it introduces circulatory dangers in the acute stage, and subsequently makes weaning from the respirator difficult. A negative pressure of 16 cm. water at a rate of 16 respiration/minute may be satisfactory, but wide variation from this figure occurs. To determine the actual ventilation occurring, the expired air should be measured. To do this a

FIG. 2.—Apparatus used for measuring ventilation of a patient in a tank respirator.

face mask is required to which are attached two short wide-bore tubes. These tubes are fitted with low-resistance valves so that inspired air enters through one and expired air leaves through the other, and is collected in a spirometer or passed through a dry meter (Fig. 2). The size of each breath (tidal air) and, since the rate of respiration is known, the minute volume is measured in this way. The machine should be adjusted to provide the minute volume appropriate to the weight of the patient (Table 2), an additional 10 per cent. being allowed when awake and 5 per cent. for every 1°F. rise in temperature. Minor degrees of hypoventilation are often shown by rise in blood pressure due to carbon dioxide retention. Chronic hyperventilation is very common in artificial respiration, but it may be minimized if the *ph* of the urine is estimated daily, preferably on an early morning specimen. The urinary *ph* is variable, but is usually 6.0 to 6.4 and frequent estimations about 7.0 suggest a respiratory alkalosis. This may be confirmed by estimating the plasma bicarbonate which should not be less than 20 m.Eq.l.

When the patient is temporarily removed from the respirator artificial respiration can be maintained by inflation through a mask. This can be done manually or, more conveniently, with a

TABLE 2

Body Weight (lb.)	Respirations (minute)	Minute Volume (litres)
14	40	1.4
35	28	2.8
70	22	4.2
105	16	5.0
140	14	5.9
175	14	7.0

respiration pump that provides intermittent positive pressure. It enables nursing, including care of the skin at the neck seal, to be performed without hurry.

When *paralysis of swallowing* is present treatment is urgent, for if the patient vomits his life is in danger. The treatment is postural drainage. The patient is turned into the prone or semi-prone position and the foot of the bed is raised so that vomit or secretions run out from his mouth. If a tipping bed is not available the bed-legs at the foot can be placed on two wooden chairs, or in the case of a child an inverted 'V' frame, as used for treating bronchiectasis, may be employed (Fig. 3).

FIG. 3.—Two methods of providing postural drainage for a patient with paralysis of swallowing (from Russell, 1952).

FIG. 4.—Apparatus for prolonged artificial respiration by intermittent positive pressure. A, Radcliffe respiration pump. B, Humidifier. C, Stott expiratory valve. D, Pressure gauge. E, Nosworthy connector. F, Tracheal tube. G, Gas meter.

To confirm that the airway remains clear, the trachea must be auscultated with a stethoscope, or a laryngeal microphone relaying to a loud-speaker may be kept permanently in position. Occasionally tracheotomy may be required to maintain a clear airway. Paralysis of swallowing almost always recovers in about ten days, but during that time constant supervision by an experienced nurse is essential.

When *primary respiratory paralysis is combined with paralysis of swallowing*, the outlook has until recently been exceptionally bleak, for in a tank respirator, even if the patient is nursed head down and prone (which is only possible with the most modern respirators) the uncompromising inspiratory effort of the machine causes secretions to be inhaled, and it is impossible to keep the lungs healthy. It is therefore necessary to adopt the method introduced in 1952 in Denmark (Lassen, 1953). A tracheotomy is performed and a short cuffed tracheal tube is inserted.* The cuff performs the dual purpose of preventing secretions or vomit from reaching the lungs and of enabling

the lungs to be inflated by intermittent positive pressure. Inflation should be carried out mechanically by a pump of proven reliability (Russell and Schuster, 1953; Crampton Smith, Spalding and Russell, 1954), but in an emergency it can be done by squeezing an anaesthetic bag. Since the inspired air does not pass through the upper air passages, it is essential to use a humidifier (Marshall and Spalding, 1953) to warm and moisten it and prevent secretions from forming crusts which cannot be aspirated. The apparatus required for this form of artificial respiration is shown in Fig. 4.

Artificial respiration provided in this way is relatively simple and leaves the limbs and trunk free for nursing care and physiotherapy. Detailed management has been described elsewhere (Crampton Smith, Spalding and Russell, 1954), but it should be stressed that the essential requirement is adequate care of the chest. For this the basic necessities are that the patient should be turned two hourly, and receive percussion and vibration to the chest once or more daily. Secretions should be completely aspirated from both sides of the chest through the tracheal tube before and after turning, and at other times as required.

*A suitable tracheal tube can be made by cutting a Magill endotracheal tube immediately below the cuff and 1.5 cm. above it.

Apparatus intended for this form of artificial respiration must include facilities for measuring expired air, and the control of ventilation is governed by the principles enumerated above.

Though patients with tracheostomes have been successfully treated in tank respirators (O'Brien *et al.*, 1954; Affeldt, 1954), the simplicity of positive pressure respiration and the full access to the patient that it provides, makes it the method of choice whenever a tracheostome is present. A tracheostome only adds to the difficulty of making a neck seal if a tank respirator is used.

Prevention of Deformity. To provide the best functional recovery, deformities must be prevented, and in particular, watch must be kept to prevent foot-drop, limitation of movement at the shoulder, flexion contracture at the hip and knee, and kyphosis. The advice of an orthopaedic surgeon may be valuable in this connection. Splints are generally unnecessary and inadvisable, but if used should be as light as possible. There is, however, scope for 'live' splints and other appliances in which elastic or springs are made to take the place of a paralysed group of muscles (Affeldt, 1954). This prevents deformity and allows the patient to keep the joint mobile and to exercise the muscles antagonistic to the springs. Passive movements should be given from the beginning to maintain the mobility of the joints. There is no evidence that after the active stage of the disease is over any object is achieved by restricting active movements, and on the contrary a good increase in power has followed early active movement (Russell and Fischer-Williams, 1954). Active movements may therefore be begun a week after the fever settles and be steadily increased. Heat, whether in the form of dry heat or hot packs, may soothe the patient or help to loosen a stiff joint, and if it does so it should be used. Hot packs, however, have no curative properties and should not become such a fetish that their application interferes with other treatment or the patient's rest.

Summary

Poliomyelitis consists of a non-specific 'minor illness' and of a 'major illness' characterized by a meningitic and a paralytic phase. The features and differential diagnosis of these stages are detailed and the danger of exercise in the meningitic phase is emphasized.

Treatment of acute paralytic poliomyelitis has two objects, to save life and to prevent deformity. There are three combinations of paralysis which may cause death. Paralysis of the respiratory muscles should be treated with a tank respirator, paralysis of swallowing by postural drainage, and the combination of the two by tracheotomy with artificial respiration by intermittent positive pressure through a cuffed tracheal tube. Correct treatment can be life-saving, but incorrect treatment is likely to add to the patient's dangers.

To obtain the best functional results, deformity must be prevented. Management aimed at producing the best functional result is outlined.

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