ASSESSMENT OF RESPIRATORY FAILURE IN POLIOMYELITIS

BY

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The chief danger to life in poliomyelitis is respiratory failure. This may come about in two ways: firstly, by paralysis of the intercostal muscles and diaphragm; secondly, by obstruction of the airway as a consequence of paralysis of the muscles concerned with swallowing, which is the most important feature of involvement of the spinal bulb. The fatal cases most often have both forms of paralysis. At the onset of failure, and often throughout, the lungs are normal. This is in contrast to respiratory failure occurring in chronic bronchitis, when lung compliance and gas distribution and diffusion are affected. The successful treatment of respiratory failure in poliomyelitis calls for an accurate assessment of respiratory function. During the past five years at Ham Green Hospital we have admitted 62 cases of respiratory and bulbar paresis due to poliomyelitis. In treating these cases we have made use of standard methods of studying respiratory function in addition to clinical observation. This paper is based on our experience with these cases.

Clinical Assessment

Failure of swallowing can be best diagnosed by observing the pooling of secretions in the pharynx. Inhalation of these secretions and airway obstruction will occur unless steps are taken to prevent them, and such cases may present with acute dyspnoea and with many rales to be heard in the chest, or as patients whose respiration in the tank respirator is unsatisfactory.

Clinical assessment of failing respiration, apart from airway obstruction, must be considered in terms of respiratory function and not of recognition of abnormal physical signs. The respiratory reserves are large and must be greatly reduced before the patient is distressed. The adult poliomyelitis patient at rest in bed needs only 6-8 litres of ventilation a minute. The maximum voluntary ventilation under test conditions ranges from 120 to 180 litres a minute in normal adults. The eye can only crudely estimate the extent of the thoracic movement, while the diaphragm, whose movement produces three-quarters of the vital capacity (Wade, 1954), cannot be directly observed at all. A function test is therefore required, and we employ the following one on all patients with paralytic poliomyelitis. The patient is asked to take a deep breath and count aloud at the rate of two digits a second as high as he can before taking another. A demonstration by the physician serves to standardize the test. We have found, as have others (Plum, 1952), that when an adult patient can only count up to 20 his vital capacity has fallen to about 2,000 ml., when he can only count up to 15 it has fallen to about 1,500 ml., and so on. Observation of the power of coughing is another less precise function test.

Through function tests alone can give an accurate early assessment of respiratory weakness, they cannot be used with patients under the age of 6. Our experience has shown that if these children have no

definite arm weakness, if they can cry loudly, or sleep soundly, they have no need of artificial respiration.

The value of clinical observation, again, is limited in accurately determining the efficacy of artificial respiration. For various reasons we have overventilated patients for considerable periods to the extent that the arterial carbon dioxide tension fell to 20-30 mm. of mercury (normal 38-42), and they showed no abnormal signs or symptoms. On only one occasion have we seen tetany with tingling of the extremities. Under-ventilation will be quickly recognized by the conscious patient, though he may complain of "not getting enough air" when his arterial blood gas levels are normal. On some occasions underventilation may be suspected when the patient attempts to breathe on his own in between or against the cycles of the respirator. If the ventilation provided by the respirator is increased he may synchronize satisfactorily, his own efforts being no longer stimulated by accumulated carbon dioxide. However, the clinical signs of underventilation—that is, the signs of anoxia and hypercapnia—appear only when underventilation is gross.

Cyanosis can be detected in a good light only when the haemoglobin saturation falls to 70-80% of normal. Owing to the S-shaped dissociation curve of haemoglobin, this fall in saturation is the result of 50% fall in oxygen tension. When oxygen is administered the fall in tension may not occur, although hypercapnia, due to underventilation, may be present. A sudden rise of carbon dioxide tension, if large enough, may produce flushing, sweating, and a rise in blood pressure, but estimation of arterial carbon dioxide tension has shown us that these signs are only a rough guide to the recognition of underventilation. For example, a patient under our care developed a sudden rise of carbon dioxide tension up to 65 mm. of mercury and showed none of the above signs. In addition, flushing, sweating, and hypertension (McDowell and Plum, 1951) may be due directly to the infection.

Laboratory Methods of Assessment

Spirometry.—Of the techniques used to estimate respiratory function in poliomyelitis, spirometry is the most valuable. It is used principally to measure the vital capacity and tidal air. Measurement of maximum voluntary ventilation and related figures (Gandevia and Hugh-Jones, 1957) may provide information of value in some cases. For the measurement of vital capacity we have found the Hutchinson waterbell spirometer most suitable, and use a small one with a 2.5-litre bell instead of the more usual 6-litre bell. The gas-meter spirometer is more convenient to use, but the two commercial types we have tested were inaccurate for small single volumes. We use a small-volume-recording spirometer with variable drum speed for measurement of the tidal air and very small vital capacities. The bell is exactly half the standard size, and so standard recording charts can be quickly interpreted. At the time of writing a small accurate flowmeter designed by Wright (1955) has been introduced by the British Oxygen Company which promises to be very suitable for clinical measurement of vital capacity and tidal air. For measurement of maximum voluntary ventilation we use an apparatus designed by McKerrow (1953). For peak expiratory flow, a related value, another small apparatus has been designed by Wright.

Estimation of Arterial Blood Gases.—We have been estimating arterial blood gases routinely in these cases during the past two years (Walley, 1957). The total carbon dioxide content and pH are measured, and from these values the carbon dioxide tension is calculated. The oxygen saturation is measured by a cuvette oximeter.

Estimation of End Tidal Carbon Dioxide Content.—Under normal lung conditions the carbon dioxide content of the air expired at the end of a normal tidal expiration is the same as that in the alveolar air, which is in equilibrium with arterial blood. The end tidal carbon dioxide tension can therefore, under normal conditions, be measured in place of that in arterial blood, a quicker and simpler procedure. In patients with a tracheotomy end tidal air can be aspirated with a syringe, but in patients without a tracheotomy more complex methods are required, and an electronic infra-red carbon dioxide meter is desirable (Collier and Affeldt, 1954). We have experience of only the simpler method (Walley, 1957).

Chest Radiography.—This is employed in certain cases daily in the acute stage for the recognition of collapse and infection of the lung. Radiographic screening in the convalescent stage allows diaphragmatic weakness to be assessed.

Pressure Measurement.—Respirators used in the treatment of poliomyelitis are always fitted with manometers for the measurement of the pressures being produced. With normal lungs and ventilation in a resting patient the pressures needed are of the order 8–12 cm. of water.

Assessment of Respiration in Practice

The questions to which we have sought an answer are three: (1) Does this patient need artificial respiration and/or measures to clear his airway? (2) When he is receiving assistance, is he being ventilated sufficiently? (3) When he starts breathing on his own, how long can he be expected to breathe at a time, and what is his residual disability?

Need for Assistance

When an adult develops weakness of the respiratory muscles without any weakness of swallowing, ventilation can be assessed by the counting test or, more accurately, by measuring the vital capacity. If these tests are performed on all cases of paralytic poliomyelitis on admission and twice daily, failing ventilation can be detected easily and its speed of development judged. When the vital capacity falls to about a quarter of the predicted normal we usually start respiratory assistance. This is the practice of American workers (Plum, 1952). Normal values for adults are well known. For children down to the age of 5 years we have collected our own figures from measurement of 180 normal convalescents (see Fig.). They agree closely with two British studies (Jones, 1955; Kennedy *et al.*, 1957) and are lower than American figures (Ferris *et al.*, 1952). When the vital capacity falls to this degree the blood gas levels are still normal, or even show evidence of over-ventilation (Dickinson *et al.*, 1953). However, it is important to have the patient confident and co-operative in the tank respirator should the vital capacity continue to fall, as is often the case. Assessment in children below the age of 5 or 6 years is a difficult matter. We have found tracings taken with a face-mask and recording spirometer helpful in a child of 18 months. Rebreathing for a short period increased the tidal air

Range of vital capacities of 180 normal convalescent children aged 5½ to 14 years.

considerably. If there is doubt it is best to artificially respire such children. If they need assistance they will co-operate; if they do not co-operate it is probable that they do not need assistance.

In the bulbar case, when swallowing fails, measurement of vital capacity is more difficult, for the presence of secretions in the pharynx appears to inhibit the patient from taking a deep breath. A face-mask must be used in place of a mouthpiece in such cases, for air escapes past the paralysed palate. In fact, the difference between the vital capacity measured with a mask and with a mouthpiece provides a useful test of palatal function in such cases. Partial collapse of the lung may occur in these cases, and this increases the ventilatory requirements and decreases ability. To detect this we make use of routine chest radiography in cases with swallowing weakness. On occasions when other methods give no help, blood gas analysis may provide useful information. The following case illustrates these points.

Case 1.—A girl of 7 years was admitted to hospital with gross pooling in the nasopharynx and so many moist sounds in the chest that pneumonia had been previously diagnosed. She was very restless and quite uncooperative throughout the acute illness. Though treated by postural drainage, a chest radiograph taken on the following day showed segmental collapse of the lung. At this time her chest and arm movements appeared powerful, though her epigastric movement suggested diaphragmatic weakness, and on occasions her colour was poor. To clarify assessment of her respiratory state blood gas analysis was done. This showed an oxygen saturation of 75% and carbon dioxide tension of 60 mm. of mercury. She recovered after tracheotomy and positive-pressure respiration, but she was left with a nasal voice, marked facial weakness, and a diaphragm which radiographic screening showed was very weak.

The possibility of damage to the respiratory centre must be considered in a case of bulbar poliomyelitis. This is difficult to detect with certainty. Irregularity in rate and depth of breathing are the features usually chosen to support this diagnosis (Baker *et al.*, 1950), and

these can be demonstrated with a recording spirometer. But these signs also appear with partial airway obstruction and this is difficult to exclude with certainty. However, if it can be shown that the patient breathes irregularly and develops carbon dioxide retention when unattended but when encouraged breathes regularly and blows off the carbon dioxide, as happened with a case (Sarnoff *et al.*, 1950), then the diagnosis is well established. We have not seen such a combination of events, for signs of peripheral respiratory failure and failure of swallowing have always confused the picture.

Assessment of Artificial Respiration

Radford *et al.* (1954) published a nomogram relating ventilatory requirements of patients at rest in bed to their body weight. Experience at Ham Green has confirmed that ventilation according to this nomogram produces normal blood gas levels in patients whose lungs are normal. The patient who suffers from respiratory weakness alone and can be successfully treated in a tank respirator comes into this category. If the tidal air, measured in these patients with a recording spirometer, is a little more than that predicted by the nomogram, it can be assumed that ventilation is adequate. A more direct method of assessment in these cases is the measurement of end tidal carbon dioxide concentration with an infra-red meter (Collier and Affeldt, 1954). It is possible to use such a meter as part of a servomechanism regulating a tank respirator (Saxton and Myers, 1957). Complexity and expense appear to rule out the routine use of this meter.

Patients who, in addition to respiratory failure, develop weakness of swallowing require a tracheotomy to maintain their airway. Even so treated, they are liable to develop lung collapse and infection, and then require 50–100% more ventilation than predicted by the nomogram. To supply these volumes, pressures of 25–35 cm. of water are needed. In these cases it is necessary to measure oxygen saturation and carbon dioxide tension of arterial blood to obtain a true assessment of respiration (Walley, 1957). End tidal carbon dioxide tension is always below that of arterial blood, and this difference often enables one to diagnose collapse of the lung.

If the arterial blood gas content, end tidal carbon dioxide concentration, and tidal air and inflating pressures are estimated at one time and the chest is radiographed, it is possible to find the ventilation and inflating pressure sufficient to provide normal blood gas levels. So long as routine radiography or clinical observation provides no evidence of changes in the lungs, measurement of the tidal air will then suffice for accurate assessment of respiratory state. In this way two to three blood gas estimations will be enough in the average case. The place of clinical observation lies in the detection of gross changes in the lungs, in oxygen saturation, or in carbon dioxide levels, which threaten life and require immediate action before more precise methods can be used.

Recovery from Respiratory Failure

In many moderately severe cases the vital capacity does not fall below 500–700 ml., figures which are well above the resting tidal air. Such patients can breathe easily on their own when the acute state is over and their temperature falls to normal. Their vital capacity will be found to rise rapidly to double that figure. We have

found with others (Affeldt *et al.*, 1957) a qualification which must be made to these statements. Those patients who have marked diaphragmatic paralysis may have greater difficulty with their breathing than might be expected from their vital capacity. The following cases illustrate this point.

Cases 2 and 3.—E. H., aged 24, was left with a vital capacity of 3,700 ml., but became dyspnoeic after slight exertion, while G. M. E., aged 26, with a vital capacity of 2,700 ml., was not dyspnoeic after similar exertion as one would expect. E. H. had marked weakness of the diaphragm, which moved paradoxically when inspiring sharply. E. H.'s functional disability was better illustrated by measurement of his maximum voluntary ventilation and peak expiratory flow. He recorded 43 and 260 litres/min. respectively, while G. M. E. recorded 84 and 310 litres/min.

These case histories also illustrate the value of radiographic screening, and measurement of maximum voluntary ventilation and peak expiratory flow in assessing residual respiratory disability.

With more severely affected patients it is difficult to say how long any one patient may breathe on his own. Measurement of the vital capacity is the only guide we use at present. Others have shown that, with a vital capacity equal to the tidal air, at rest a patient will be able to breathe for periods of from 15 minutes up to a few hours. These and similar figures (Austin *et al.*, 1952; Spencer, 1954; Ferris *et al.*, 1955) we have been able to confirm, but they are general and not particular statements. Ferris *et al.* (1955) have made use of a fall of vital capacity during unassisted breathing as a guide, although we have not found this of much use. Possibly measurement of other function tests may yield useful information; but at present, apart from measurement of vital capacity, we are limited to encouraging the patient to breathe as long as he can while watching for the appearance of any clinical evidence of respiratory distress.

In conclusion it should be mentioned that even a mild upper respiratory infection may set a patient back considerably. For example, the vital capacity of a female patient four months after the onset of poliomyelitis was 2,300 ml. With an upper respiratory infection she became relatively short of breath. Her vital capacity was found to have fallen to 1,300 ml., although no signs were to be heard in the chest and the radiograph was normal.

Summary

The successful treatment of respiratory failure in poliomyelitis calls for an accurate assessment of respiratory function. The methods used in the management of 62 cases of respiratory and bulbar paresis due to poliomyelitis are discussed. The application of these methods to the assessment of the respiratory function of these cases is described.

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ALTERATIONS IN THE DIURNAL EXCRETION OF ELECTROLYTES DURING INTERMITTENT POSITIVE- PRESSURE RESPIRATION

BY

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The diurnal variations in the excretion of water and electrolytes by the kidney have been studied extensively, the most important contributions to the literature having been listed by Stanbury and Thomson (1951) in an account of their own observations. These authors found the excretion per minute of water and of sodium, potassium, chloride, and bicarbonate ions falls during the night together with the urinary pH, whereas the output of ammonia and titratable acid rises. These diurnal excretory rhythms are extremely resistant to attempts to alter their pattern. Thus, starvation, water deprivation for three days, salt deprivation for nine days, the sustained action of deoxycortone acetate and antidiuretic hormone, and the temporary disturbance of the sleep rhythm do not abolish the excretory rhythm (Stanbury and Thomson, 1951).

On the other hand, hyperventilation for periods of 15 to 40 minutes disrupts the excretory rhythm, causing an increased excretion of sodium, potassium, and bicarbonate ions with little change in the excretion of chloride (Brown, Campbell, Elam, Gollan, Hemingway, and Vischer, 1949; Stanbury and Thomson, 1952). This last observation suggests that the nocturnal reduction in the excretion of these electrolytes may be due to the raised alveolar CO₂ tension which develops at night (Mills, 1953). On the other hand, Longson and Mills (1953) found that the maintenance of a raised alveolar CO₂ tension by the inhalation of 5 to 6½% CO₂ for three hours in the morning failed to prevent the matutinal rise in the excretion of electrolytes. Hence, though it is tempting to attribute the variations in the excretory rhythm of electrolytes to changes in respiration, the evidence is at present conflicting.

The maintenance of human subjects on intermittent positive-pressure respiration (I.P.P.R.) for long periods

during the treatment of respiratory paralysis has provided an opportunity for further investigation of this problem, the results of which are presented here. This method of artificial respiration permits respiration to be maintained at a constant rate, pressure, and tidal volume by day and by night for long periods; hence the variations in these factors which constantly occur during normal respiration are eliminated. Because patients being thus treated are usually recumbent and variations in the renal excretion of electrolytes during acute changes of posture have been reported (Wilkins, Bradley, and Friedland, 1950), observations on the effects of prolonged recumbency on the diurnal excretory rhythms are reported first.

Material and Methods

Observations on the effects of recumbency were made serially on one subject before, during, and after recumbency, and single observations were made on four other subjects, two of whom were totally paralysed by motor neurone disease, after varying periods of recumbency. The effect of I.P.P.R. was observed in six subjects after varying periods of artificial respiration. The urine was obtained by inserting an indwelling catheter and collecting urine at midnight, 6 a.m., 8 a.m., 10 a.m., 12 noon, 2 p.m., 4 p.m., and 12 midnight. The patients were recumbent and capable of little physical exertion apart from slight movements of the arms. They kept a normal ward routine, sleeping from about 10 p.m. to 6.30 a.m. and taking food by mouth or by nasal tube at the usual meal-times. The respiratory minute-volume was maintained at a level suitable for the patient, and its constancy was checked at frequent intervals throughout the period of collection of urine by passing the expired air through a gas-meter. The sodium and potassium ionic content of the urine was estimated by flame-photometer, and the chloride by a potentiometric method (Sanderson, 1952).

Effect of Recumbency

A male patient aged 28 years with an anxiety state, who complained of intolerable fatigue, was glad to lie recumbent for 21 days. He remained flat in bed, lying on his back, or on either side, but not sitting up. Two estimations, one of which is shown in Fig. 1, were made before he was confined to bed, and showed a normal diurnal pattern for the excretion of sodium, potassium, and chloride. Estimations were made on days 1, 2, 4, 7, 9, 13, and 21 of the recumbent period, but no alteration in the diurnal pattern was observed (Fig. 1). Two further estimations after he resumed activity again showed a normal pattern.

In view of the absence of change with a short period of recumbency, single estimations were made on four other patients. K. T. had disseminated sclerosis and had been in bed for 21 days. D. C. had a prolapsed intervertebral disk, and estimations were made on the 14th and 21st days of recumbency; and J. C. and M. had motor neurone disease and had been in bed and totally paralysed for 17 months and 2 years respectively. All showed normal diurnal excretory patterns (Fig. 2).

Effect of I.P.P.R.

In only two of the six patients on I.P.P.R. was it possible to observe the type of diurnal excretory pattern of electrolytes before treatment was instituted. Both had normal patterns, and, as the remaining four patients