LABORATORY INVESTIGATIONS DURING TREATMENT OF PATIENTS WITH POLIOMYELITIS AND RESPIRATORY PARALYSIS

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This paper is based on the experience gained during the 1952 epidemic of poliomyelitis in Copenhagen, in which there were many more severe cases than in any previous epidemic in Denmark. The management of this epidemic is described by Lassen. We would like to stress that the ideas and the work behind this presentation result from close co-operation and numerous daily discussions between all the physicians involved in spells of duty of 12–16 hours a day for months, ably and enthusiastically assisted by the nurses and medical students.

Our tasks in the laboratory were (1) to control the effectiveness of ventilation; (2) to control the circulatory collapse, the renal function, the fluid balance, etc.; and (3) to assist in evaluating the ventilatory effect and the influence on circulation of the different types of mechanical respirator.

Control of Ventilation

In the control of gas exchange, the *p*H, the CO_2 tension in arterial or in venous blood, and the oxygen saturation of arterial blood were determined.

The CO_2 tension was calculated from the blood *p*H and the total plasma CO_2 according to the following two formulae:

$$pH = 6.1 + \log \frac{\text{conc. } HCO_3^-}{\text{conc. } H_2CO_3}$$

$$\text{total } CO_2 = HCO_3^- + H_2CO_3$$

In the first, which is that of Henderson–Hasselbach, the numerator is the CO_2 carried as bicarbonate, while the denominator is the amount in physical solution. We estimate the total CO_2 content of plasma by Van Slyke's technique. The total CO_2 is usually referred to as serum or plasma bicarbonate, which is incorrect, although the ratio between chemically and physically bound CO_2 is normally about 20 to 1. If the values of *p*H and of total CO_2 are substituted, we have two equations with two unknowns which may be solved with the help of a nomogram expressing CO_2 tension directly as measure of the amount in physical solution.

The *p*H determinations are made directly in heparinized blood from which air is excluded, and which is kept at 37° C. until the examination is done. The effect of glycolysis must be avoided, either by the addition of fluoride or by doing the determination within fifteen minutes of taking the sample. By our measuring technique we obtained a high degree of accuracy and very rarely saw two consecutive measurements with a variation of more than 0.02. If the variation is higher than this there is an error of adjustment, and a new determination must be done.

The normal values for *p*H in arterial blood are 7.38 to 7.46, in venous blood 7.34 to 7.43. The CO_2 tension in normal arterial blood varies from 35 to 43 mm. Hg, and in venous blood from 40 to 53 mm. Hg.

By January 1, 1953, we had done 705 determinations of *p*H in arterial or venous blood, from patients with threatened or manifest respiratory paralysis (Table I).

Most of the *p*H values in arterial blood are between 7.40 and 7.54, and in venous blood between 7.35 and 7.49. The CO_2 tensions are correspondingly low, between 25 and 35 mm. Hg in arterial blood, and between 30 and 45 mm. Hg in venous blood. The highest CO_2 tension we found was 150 mm. Hg in venous blood, and the lowest 9 mm. Hg in arterial blood. There was a general tendency to a slight alkalosis with lowered CO_2 tension, which was usual in the patients under artificial ventilation.

In several patients we found, within a short period of time, colossal changes in *p*H, in the total carbon dioxide in plasma, and in the CO_2 tension.

In a 5-year-old boy, almost moribund on admission, the *p*H of venous blood was 6.99, the total CO_2 in plasma 39 mMol and the CO_2 tension 150 mm. Hg. After tracheotomy and manual artificial ventilation the *p*H had increased in two hours to 7.52 and fifty-five minutes later to 7.65, accompanied by a fall in the total CO_2 content in plasma as well as in the CO_2 tension (Table II).

TABLE II.—*pH and CO_2 Tension in Boy Aged 5*

Date	Hour		*p*H	Total CO_2 (mMol)	Free CO_2 (mm.Hg)	HCO_3^- (mMol)
6/9	11.40 a.m.	Venous	6·99	39·0	150	34·5
	2.10 p.m.	Arterial	7·52	24·4	32	24·5
	3.05 „	„	7·65	15·6	14	15·2

On the other hand, we found only slight changes from day to day in patients who were in a relatively stable clinical condition (Table III). The interesting thing about this case is that it was decided at the conference on September 9 to increase the rate of manual ventilation in all patients from 20 to 30 a minute. On September 10 the *p*H was 7.70 and the CO_2 tension 17. Similar changes were found in all patients, and it was decided to cut down the frequency to 25 a minute. In this patient the *p*H then fell to 7.56 and the CO_2 tension rose to 23.

Naturally the ideal artificial ventilation should tend to keep the *p*H and the CO_2 tension in the blood within normal limits. It was therefore important to correlate clinical and laboratory findings in order to differentiate the symptoms of overventilation and underventilation.

Severe degrees of underventilation always gave rise to serious clinical signs (increasing blood pressure and pulse rate, poor general condition, sweating, etc.), which are generally easy to recognize. The main indication was a change in the blood pressure.

A state of overventilation is, however, often difficult to detect clinically because the patient in this condition also would complain of air hunger, might have a slightly increased blood pressure, and might even be a little cyanosed. One was therefore often in doubt whether these patients were underventilated or overventilated, especially when it was impossible to measure the ventilation in litres a minute. This could be resolved only by blood analysis. A determination of the CO_2 content of alveolar air could also be

TABLE I.—*pH Values and CO_2 Tension*

pH in arterial and venous blood

pH	6.95	7.00	.05	.10	.15	.20	.25	.30	.35	.40	.45	.50	.55	.60	.65	.70	.75	.80	.85	.90	total
a	0	0	1	2	4	5	4	20	52	71	88	70	43	18	10	6	2	0	1		397
v	1	0	0	2	4	2	10	24	48	81	63	35	23	11	2	2	0	0	0		308

CO_2-tension in mm.Hg.

	<10	10	15	20	25	30	35	40	45	50	55	60	65	70	>70	total
a	1	2	31	45	66	74	39	41	15	14	5	5	4		6	287
v	0	0	10	19	23	60	46	53	29	21	7	10	1		8	350

TABLE III.—*pH and CO_2 Tension in Woman Aged* 30

Date	Hour		pH	Total CO_2 (mMol)	Free CO_2 (mm.Hg)	HCO_3^- (mMol)
1/9	1·00 p.m.	Venous	7·49	24·0	32	24·1
	4.45 „	„	7·47	22·2	31	21·3
2/9	9.15 a.m.	„	7·50	25·0	32	24·2
	10.30 „	Arterial	7·50	25·6	36	24·5
	10.35 „	Venous	7·47	24·8	34	28·8
	2.15 p.m.	„	7·48	26·3	36	25·2
4/9	9.10 a.m.	„	7·55	26·7	30	25·8
6/9	10.55 „	Arterial	7·55	26·2	30	25·3
8/9	12.35 p.m.	Venous	7·56	27·6	31	26·7
10/9	10.10 a.m.	„	7·70	22·1	17	21·6
13/9	10.10 „	„	7·56	21·0	23	20·3

used, and we worked out a rapid method of determining this. Into a 20-ml. syringe were aspirated 5 ml. of a 12 mMol bicarbonate solution containing a small amount of bromthymol blue, and 15 ml. of air, withdrawn from the system at the end of expiration. After agitation for half a minute the colour was compared with that of a series of standards, from which the CO_2 content was estimated. In patients under manual artificial ventilation it was difficult to eliminate the effects of CO_2 absorption in the canister and of soda-lime dust in the tubing and in the trachea. We have no doubt that the estimation of alveolar CO_2 will be the method of choice, and we have attempted to devise a satisfactory technique.

A very constant symptom of overventilation is tetany, which was seen in a number of patients with an arterial blood *p*H of 7.6 or over. In others there may have been a latent tetany, masked by muscular paralysis. It seems possible that the alkalosis in some patients may cause a rise in blood pressure, which is corrected when the *p*H and the CO_2 tension are brought back to normal.

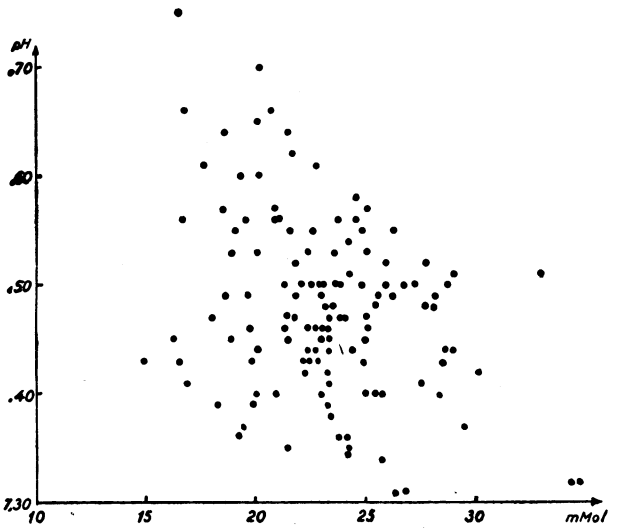

FIG. 1.—*p*H and total CO_2 in patients with less than 70 mg. of non-protein nitrogen per 100 ml. arterial blood.

The alkalosis may affect the oxygen dissociation curve, leading to an abnormally low oxygen tension in the tissues even when the arterial blood is fully saturated. This will be more evident when the artificial ventilation is carried out with air. The symptom of air hunger may be explained by this, but it is also due to habituation of the respiratory centre to a high blood *p*H and a low CO_2 tension. If a normal subject is hyperventilated for 24 hours in a respirator he will continue to hyperventilate for up to 12 hours afterwards, when the blood *p*H will have returned to normal. The cyanosis in alkalosis we explain by the low oxygen tension in the tissues, with a decreased rate of blood flow due to a state of shock or local spasm in the vessels which is known to be produced by a low CO_2 tension.

It was impossible, from determinations of total CO_2 alone, to decide whether there was acidosis or alkalosis (Fig. 1). With blood of *p*H 7.42 we found values of total CO_2 from 15 to more than 30 mMol, and of 20 mMol in samples of blood with a *p*H varying from 7.35 to 7.70.

The patients demonstrated in Fig. 1 probably had no metabolic disturbance of their acid–base balance, unlike some, at least, of the patients on whom the values presented in Fig. 2 were determined. In all from the latter group the

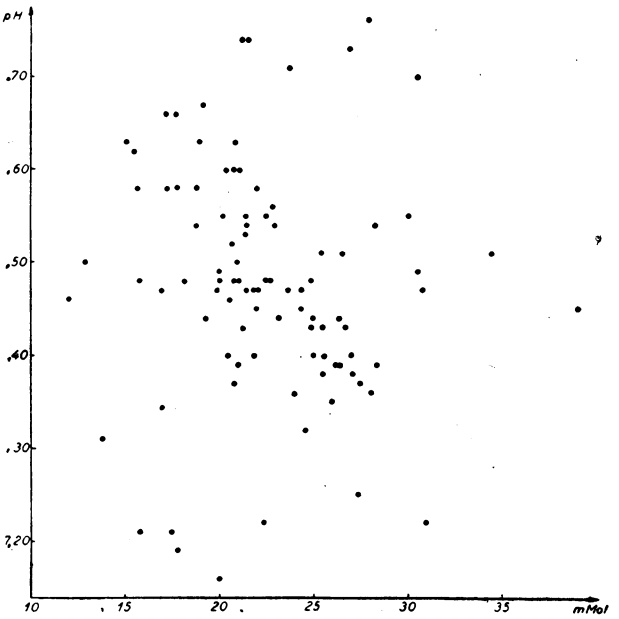

FIG. 2.—*p*H and total CO_2 in patients with more than 100 mg. of non-protein nitrogen per 100 ml. of arterial blood.

non-protein nitrogen was above 100 mg. per 100 ml., probably indicating a more or less pronounced renal acidosis. The figures are spread more than in Fig. 1, and there is no relation between the total CO_2 of plasma and the blood *p*H. In patients with a non-protein nitrogen below 70 mg. per 100 ml., on the other hand, there is moderate correlation between CO_2 tension and blood *p*H (Fig. 3). It is possible to find a normal CO_2 tension, indicating sufficient ventilation, even with a low blood *p*H (Fig. 4).

The oxygen saturation in arterial blood was determined by Brinkmann's reflectometric technique. Determinations of oxygen tension should have been made also, but we did not work out a satisfactory technique until the spring of 1953, a technique which may be of future benefit.

In 25 patients with untreated respiratory insufficiency we found oxygen saturations varying from 80 to 96%. This oxygen lack produces the symptom of air hunger, which diminishes or disappears when oxygen is administered with-

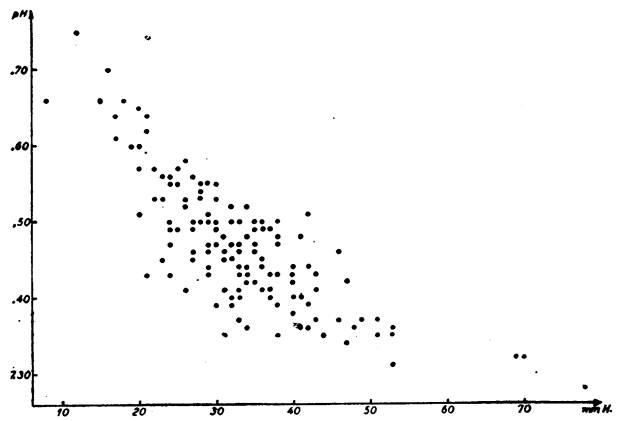

FIG. 3.—*p*H and CO_2 tension in patients with less than 70 mg. of non-protein nitrogen per 100 ml. of arterial blood.

out increased ventilation. Retention of CO_2 is not found until later. In cases with decreased oxygen saturation there were already clinical signs of respiratory insufficiency, so pronounced as to call urgently for artificial respiration of some kind. The degree of oxygen saturation is thus of little importance as an indication for treatment.

A decrease in oxygen saturation may occur also as a consequence of atelectases or of uneven ventilation in a respirator, and this is probably the field in which determinations of oxygen saturation have greatest importance.

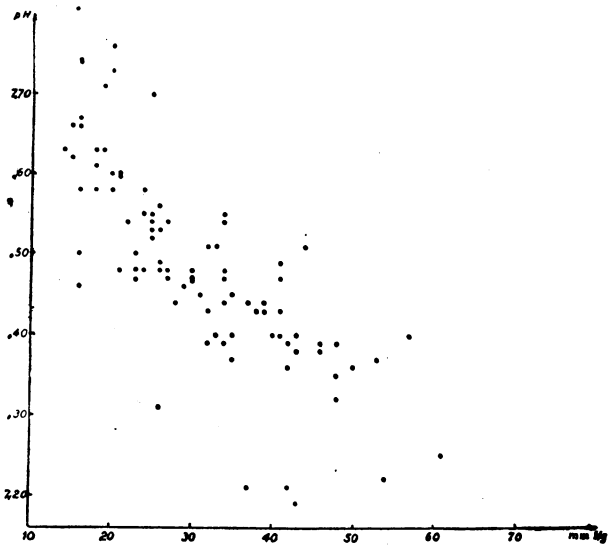

FIG. 4.—*p*H and CO_2 tension in patients with more than 100 mg. of non-protein nitrogen per 100 ml. of arterial blood.

It must be stressed that lowered oxygen saturation may coexist with over-elimination of CO_2. The blood returning from underventilated parts of the lung will have a lowered oxygen saturation and a moderately raised CO_2 tension; in the blood from overventilated parts there may be a considerable lowering of CO_2 tension, though the oxygen saturation can be raised very little above that of normal arterial blood. We have seen numerous examples of this.

Conclusions on Control of Ventilation—(1) Measurements of blood *p*H and of CO_2 tension have proved practicable in large groups of patients. They are of value in deciding whether a patient is overventilated or underventilated with respect to CO_2. (2) A simple and accurate method of estimating CO_2 in alveolar air, as a bedside test, would be valuable. (3) The degree of oxygen saturation of arterial blood is of little value as an indication for artificial ventilation, but a fall may suggest uneven ventilation of the lungs in a respirator or the presence of

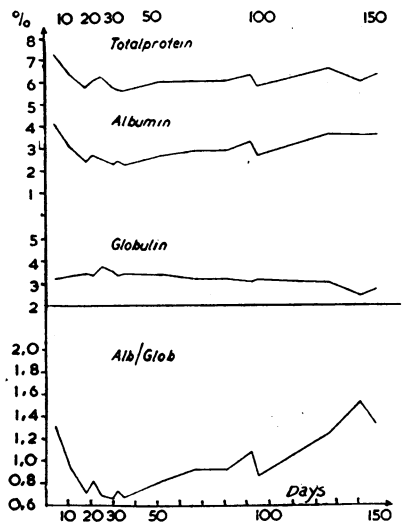

FIG. 5.—Albumin and globulin values and A/G ratio in a patient.

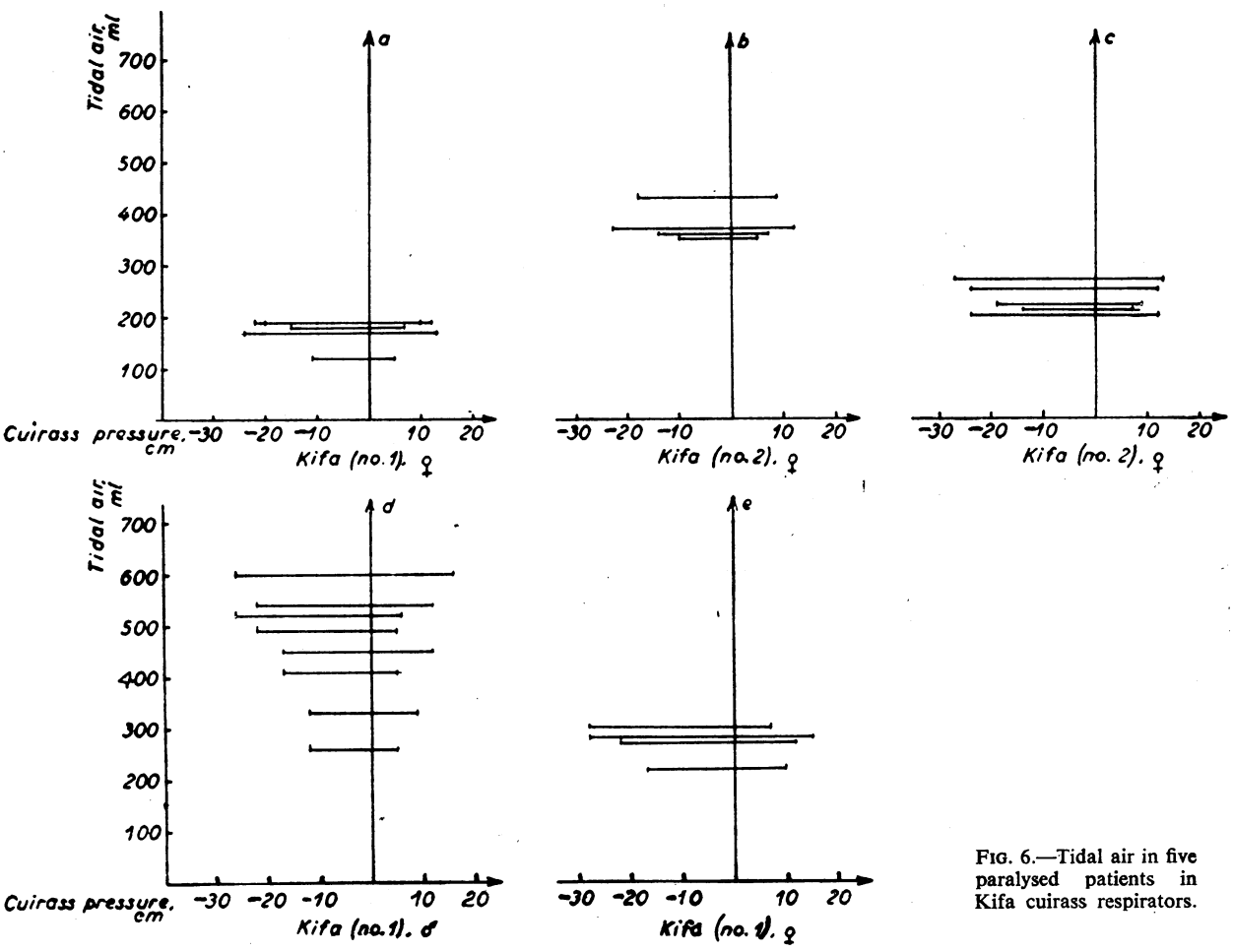

FIG. 6.—Tidal air in five paralysed patients in Kifa cuirass respirators.

atelectases. The result should be considered in relation to the CO_2 tension. (4) Determination of the oxygen tension of arterial blood is likely to be of value.

Other Laboratory Investigations

In about 200 patients with poliomyelitis we did fractional estimations of serum proteins: in about 100 of these we followed the changes for some time. In aparalytic patients and in patients with pareses of light or moderate degree, without respiratory insufficiency, we found practically no change except a small decrease in albumin. In patients with respiratory insufficiency there was a tremendous fall in albumin in the first few days after starting artificial ventilation.

Fig. 5 shows the values for albumin and globulin and the A/G ratio in one patient. In the first two weeks there was a heavy fall in albumin, the globulin being little changed. This picture was found in nearly all patients under artificial ventilation—in the great majority, manual positive-pressure ventilation.

We feel that this change is due to a depression of liver function. In about 50 patients the bromsulphthalein test was usually positive, often to a high degree. Urobilinuria was frequent, and in 10 patients there was manifest jaundice. The disturbances of liver function are probably caused by changes in circulation, brought about by the positive-pressure ventilation. This type of ventilation tends to decrease venous return and cardiac output (T. Bennike, in press).

The serum amino-acid nitrogen was found to be normal in 19 patients. In 12 patients with paralyses paper chromatographic examination showed no pathological changes in quality or quantity of urinary amino-acids. In five there was an increase in output of taurin, probably related to increased tissue breakdown. In 14 patients the daily nitrogen excretion was found to rise a few days after the onset of extensive paralyses, reaching a maximum of as much as 45 g. in the second week and falling again to normal by the third or fourth week.

The increased nitrogen output is paralleled by an increased excretion of potassium, often to more than three times the normal, due to liberation of potassium in the destroyed muscles.

In a number of patients repeated determinations of serum potassium were made. In the majority the values were normal, but in a few cases we found hypokalaemia, calling for parenteral administration of potassium. Hyperkalaemia was found in some patients with azotaemia.

The concentrations of sodium and chlorides in serum were usually normal or subnormal. The salt balance was followed by daily estimations of urinary chlorides by the Fantus test. We tried to control the intake of salt so that the daily urine contained a few parts per thousand of chloride. Overdose of sodium chloride is dangerous, and gave rise to a few cases of pulmonary oedema in the early period. The fluid balance was followed on special records of intake and output.

Haemoglobin estimations were done four to six times a day, to reveal, as early as possible, haemoconcentration indicating the development of a state of shock.

Kidney function was evaluated through many, often daily, determinations of non-protein nitrogen in the blood, using the microtechnique. In the majority there was an increase in N.P.N. in the first few days after the beginning of positive-pressure ventilation : in 105 patients to more than 70 mg. per 100 ml. and in no fewer than 53 to more than 100 mg. per 100 ml. We feel this increase is due to a transient or, in certain cases, a permanent depression of renal function due to shock, combined with an accumulation of nitrogen-containing substances from tissue destruction.

About 130 patients had some bleeding and were examined for haemorrhagic diathesis. The examinations done were prothrombin, thrombocyte count, fibrinogen, coagulation time, bleeding-time, and capillary resistance. A few cases showed a moderately decreased prothrombin content in plasma ; otherwise all the findings proved normal apart from the capillary resistance. Where there was a real increase in the bleeding tendency there seemed to be a definite relation to decreased capillary resistance. It is too early to say anything about the reason for the change in capillary resistance—it may be due to anoxia (T. Bennike and F. Grandjean, in press). In the other cases the haemorrhage was presumably traumatic, due to injury of the bronchial mucosa by repeated suction of bronchial secretions.

Evaluation of Different Types of Artificial Ventilation

The pronounced variations from normal we have described above in patients undergoing artificial ventilation may be accounted for partly by the disease, as such, and partly by the effects on the body of the artificial ventilation. We will therefore endeavour to evaluate the effectiveness of the ventilation and the effects on the circulation of the different methods employed.

Cuirass Respirators

In the first period of the epidemic patients died in spite of treatment in a cuirass respirator and of administration of oxygen. There is no doubt whatsoever that the cause of the fatal outcome was CO_2 retention in the body with alteration of *p*H towards the acid, in other words, a respiratory acidosis. Unfortunately we have only a few measurements in these patients, but in all cases we found a low blood *p*H, a very pronounced increase in the total CO_2 in plasma, and a marked rise in CO_2 tension. The characteristic feature of these patients was that they did not do well in a respirator without administration of oxygen ; whereby the anoxaemia was overcome but the ventilation was too small to remove the CO_2. If a patient in this type of respirator does not get on without oxygen it is a good indication that the ventilation is insufficient, leading to the risk of death from acidosis.

Later measurements with a cuirass respirator indicate that ventilation was, in fact, too small. In this connexion it must be remembered that patients in the acute phase of the disease have a high temperature, with a metabolic rate increased by at least 50%.

Fig. 6 shows the tidal air in five paralysed patients, chosen at random, with a well-placed Kifa cuirass. In only two is the tidal air above 300 ml., which, at a rate of 22–25, gives a minute-volume of 6–8 litres. It is thus obvious that the Kifa respirator will not give enough ventilation without spontaneous co-operation by the patient. Further, like other cuirass respirators, it tends to pro-

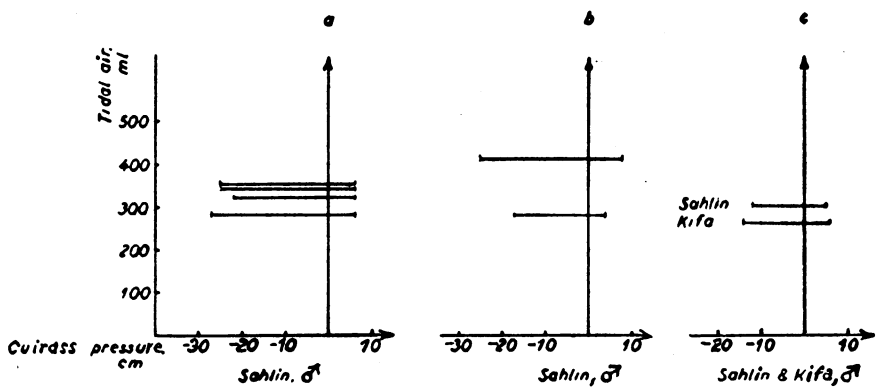

FIG. 7.—Tidal air in three patients in Sahlin cuirass respirators.

duce uneven ventilation, the bases of the lungs especially being underventilated, which we have confirmed at necropsy in a number of cases.

Similar measurements, from three patients in Sahlin cuirass respirators, are shown in Fig. 7. The tidal air is here about 300–400 ml.

Tank Respiration

The tank respirator is capable of producing a much greater ventilation than the cuirass (Fig. 8), up to 18 litres a minute having been measured, and there is no doubt that it ventilates the lungs more evenly. However, in two cases the surroundings, examination, physiotherapy, and general nursing of the patient become difficult or impossible to carry out.

Manual Positive-pressure Method

Manual positive-pressure respiration was used in most cases, and was carried out by medical students. As we have already mentioned, practically all patients were overventilated in respect of CO_2. One day we measured the pressure at the proximal end of the tracheal tube with a simple water manometer, suitably damped, and counted the frequency. We did not tell the students what we were measuring.

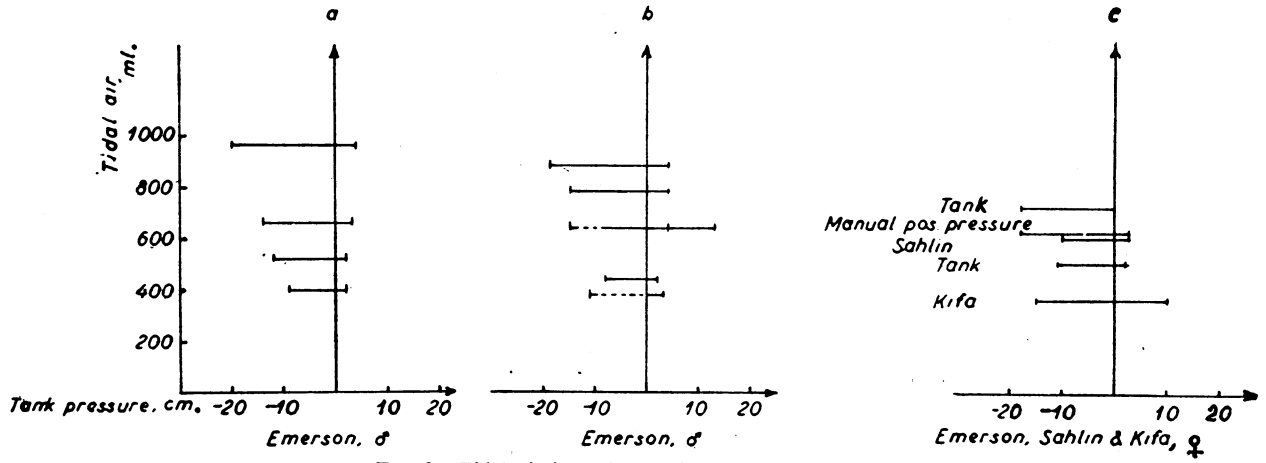

FIG. 8.—Tidal air in patients using different methods.

—and we used tank respirators very little—we found a considerable decrease in oxygen saturation of arterial blood where there was overventilation in respect of CO_2, indicating uneven ventilation. The tank has been used with good results in other epidemics, either alone as a negative-pressure chamber or in combination with some method of positive-pressure ventilation. However, the tank has practical disadvantages: because the patient is excluded from

The inspiratory pressure is demonstrated in Fig. 9, measured in 27 individuals in two different wards on the same day. There are noticeable variations in the inspiratory pressure during the period of observation, which is only a few minutes. Some students worked very constantly, while with others the pressure varied by as much as 10 cm. of water. There is no striking difference between male and female patients and children. The average inspiratory pressure is from 12 to 25 cm. of water. We have put down the average frequency during the few minutes of observation. At this time the students had been instructed to ventilate at a rate of 20–22 a minute. The tendency to exceed this rate is quite obvious, and is probably explained by the fact that most patients get used to a big ventilation and keep asking for more air. The pressure during the expiratory phase was also recorded: in most cases it was zero, according to instructions. In no fewer than 4 of the 27, however, it was not allowed to drop to zero, but was held at from 1–4 cm. of water.

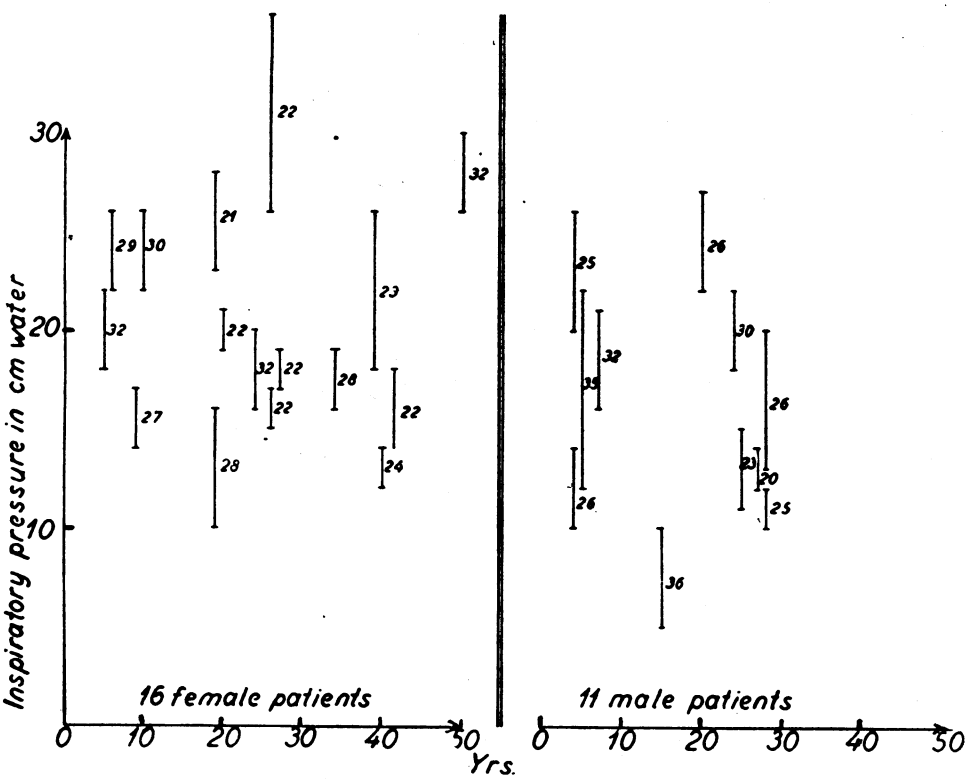

FIG. 9.—Inspiratory pressure and ventilatory frequency in 27 patients.

On another day we measured the ventilation in a series of patients. The pressure and the rate used by the student were noted, a gasometer was shunted in, and the ventilation was measured under the same conditions (Fig.

10). In most adults the ventilation was 10–15 litres a minute. In Fig. 10 tidal air is compared to inspiratory pressure in some of the patients. It can be seen that tube pressure and tidal air show pretty good correlation, without much spread, in the adult group. The most usual pressure, of 12–25 cm. of water, gives a tidal volume of 300 to 700 ml. A frequency of 15–20 should therefore suffice to give a normal ventila-

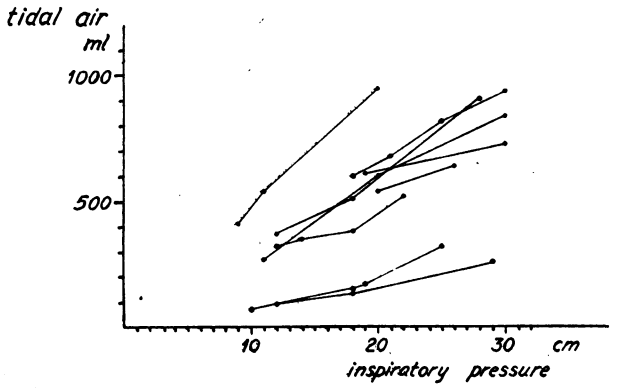

FIG. 10.—Inspiratory pressure in 9 patients.

tion of 6–8 litres a minute. With a rate of about 25 respirations a minute one usually obtains a ventilation of 10–15 litres.

Conclusions on Types of Ventilators.—(*a*) Cuirass respirators are of no use in the acute stage of the disease, because the ventilation is too small. (*b*) Tank respirators give an adequate volume of ventilation, but this may be uneven, and they have certain disadvantages as regards examination, physiotherapy, and general nursing of the patient. (*c*) The manual positive-pressure method gives sufficient and even ventilation when done correctly. In practice there is a tendency to over-ventilate.

Effects on the Circulation

Although it has not been possible to demonstrate any harmful effect from alkalosis and a fall in CO_2 tension due to overventilation (except for the tetany), the problems have by no means been solved just by offering the patients with respiratory paralysis a sufficient ventilation or an overventilation. We still have to deal with the very important question of the influence of artificial ventilation on circulation; and the relative value of the different types of artificial ventilation depends exclusively on this, if they are at all capable of ventilating the patient sufficiently.

The great importance of this problem is that circulatory problems of any importance had not been seen until the introduction of the positive-pressure breathing, which corrected the ventilatory insufficiency from which the earlier patients died. As a result, most patients who died during this epidemic died from shock or the consequences of shock.

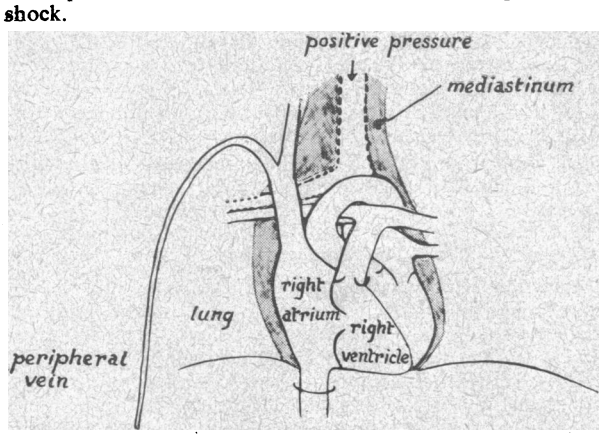

FIG. 11.—Diagram showing how positive pressure is transferred.

A considerable decrease in venous return to the heart, from any cause, leads to a fall in cardiac output and a state of shock. It is essential to realize that, in certain conditions, positive-pressure ventilation may cause or contribute to the development of shock by this means. During the phase of inspiration (Fig. 11) the pressure is raised in the bronchi and lungs, and a high fraction of this positive pressure is transferred to the mediastinum and from there to the right atrium and the central veins. This produces a fall in the pressure gradient from the peripheral veins to the central veins and the right atrium, and the venous return is decreased while the pressure rises in the peripheral veins. This peripheral pressure rise will increase the venous return during the expiratory phase, when the intrapulmonary pressure is falling, because the atrial pressure falls before the venous pressure. If this phase of falling pressure, plus the expiratory pause, is long compared with the inspiratory phase, venous return and cardiac output will be kept up.

Werkö (1947) has shown experimentally that the right ventricular output will fall if the expiratory phase is too short compared with the inspiratory. He also found that the output fell if the mean pressure in the mask was high —that is, more than 5 mm. Hg—irrespective of the type of respiration. We have measured the mean right atrial pressure in one patient under positive-pressure ventilation and found that it takes a very considerable rise of mean pressure in the tracheal tube to give relatively small changes in the mean atrial pressure, indicating a lowered effective filling pressure in the right heart. We measured the venous pressure in some cases and found a rise when the expiratory phase was shortened, and especially when the expiratory pressure did not fall to zero.

At higher venous pressures there must be some passage of plasma into the tissues, due to increased capillary pressure. This will decrease the circulating blood volume and lead to a further reduction of venous return.

There can be little doubt that manual positive-pressure ventilation—which in the circumstances was the only way to keep the patients ventilated—has given rise to many cases of shock. Many of the patients were in very poor condition before tracheotomy and had anoxia and hypercapnia, both factors shock-producing, just as damage to the vasomotor centre by the virus, with accompanying vasodilatation, has undoubtedly been present in some cases. When the circulation in such a patient is exposed to another load, such as positive-pressure ventilation, the chances of a fatal outcome are high even if the ventilation is carried out correctly, and especially so if this is not the case.

Conclusions on Effects of Artificial Ventilation.—For these reasons we have come to the following conclusions. (1) All forms of positive-pressure ventilation, including the tank respirator, which works essentially in the same way, produce unphysiological circulatory conditions because the negative inspiratory pressure gradient from the outside of the body to the thoracic cavity does not occur. (2) The least harmful method of positive-pressure ventilation with regard to the circulation, should have (*a*) a low frequency, (*b*) a short inspiratory positive-pressure phase, and (*c*) a long expiratory phase with a rapid fall of pressure to zero, where it remains until the next inflation. This form of ventilation will probably do no harm to the cardiac output of a healthy person. (3) Overventilation is harmful, the extent of ventilation being roughly proportional to the inspiratory pressure (Fig. 10).

Mechanical Ventilators

It was difficult to carry out mechanical ventilation correctly, the tendency being to overventilate at the demand of the patient and to hold a positive pressure during expiration. It was therefore clear at an early stage that it would be desirable to introduce a machine capable of being adjusted to the necessary requirements.

Several types of such machines were tried out in the Blegdams Hospital. The only one which has functioned satisfactorily as regards both the respiratory and the

circulatory systems is the Engström (1953) respirator. This machine has the advantage that it combines positive-pressure inflation with a mechanical compression of the lower aperture of the thorax during expiration. Because of this, a lower inspiratory pressure is effective, which is an advantage for the circulation. Bang's respirator shows some promise, but our experience is not yet sufficient to evaluate its usefulness during the acute phase.

There is no doubt that the Engström respirator should be used in the acute stage of respiratory insufficiency to minimize the risk of shock. This risk is especially high in the first week of the disease. Later it seems the circulatory system becomes adjusted to compensate for the positive pressure. If an Engström respirator is not available, correctly performed manual positive-pressure ventilation should be used.

After the acute phase a change may be made to any of the other methods of artificial ventilation.

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THE ANAESTHETIC MANAGEMENT OF PATIENTS WITH POLIOMYELITIS AND RESPIRATORY PARALYSIS

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In 1952 Denmark suffered from a severe epidemic of poliomyelitis. Not only was the number of cases high but the incidence of bulbar cases was higher than in any previous epidemic.

In the years 1934–44 76 patients were admitted with respiratory paralysis due to poliomyelitis and 80% of these died. At least 51 had bulbar poliomyelitis, and 48 (94%) of these died. The mortality was equally high in the following years.

In 1952 5,722 cases were notified, and 3,722 of these passed through Blegdams Hospital. In the period July 7, 1952, to March 2, 1953, 349 cases of respiratory and/or swallowing insufficiency were treated. Up to August 26 31 patients with respiratory paralysis were treated, and 27 of these died, 19 within three days of admission. This state of affairs, and also the fact that the hospital had only seven respirators and the epidemic was just beginning, made the situation catastrophic. It was necessary to look for new methods of treatment, and, at the suggestion of the anaesthetists, manual intermittent positive-pressure ventilation was introduced. This was by far the most commonly used method during the whole epidemic. The introduction of this type of ventilation was the beginning of large-scale co-operation between epidemiologists and anaesthetists in Denmark.

At an early stage the following measures were adopted in order to intensify the treatment and also to improve the results: (1) Patients who were likely to develop respiratory complications were transferred to special wards for observation, and recording of blood pressure, pulse, respiration, etc. (2) Tracheostomies were done under general anaesthesia and cuffed tubes were used to seal the trachea from the pharynx. (3) Manual intermittent positive-pressure ventilation was carried out as a supplement to the respirators or instead of them. (4) Secondary shock was treated effectively.

The measures we adopted to solve our problems are not claimed to be ideal, but they may be of interest to others faced with similar circumstances.

Treatment of the Typical Patient with Respiratory Paralysis

It soon became evident that active treatment of the severely ill patient must begin before he arrives at the centre—the fever hospital—because these patients do not tolerate transport in the acute stage of the illness. Several times within the first months it happened that patients came to Copenhagen from provincial hospitals and had to be tracheotomized and ventilated before removal from the ambulance. From the experience of these desperate cases we agreed that the logical thing was to do the tracheostomy before transport, so that ventilation and aspiration could be done during transit. For this reason a team, consisting of an otologist, an anaesthetist, and a nurse, was ready at any time to fetch a patient to the centre by car or plane, with or without tracheostomy. All equipment for resuscitation, anaesthesia, and operation was ready, packed, in the admission centre of the hospital.

The *period of observation* may vary from a few seconds to several weeks. As we have mentioned, in some cases tracheotomy had to be done before they reached the centre; in other cases at the moment of arrival in hospital. At the other extreme were patients who never had to have tracheotomy, but who had to be watched closely in the observation wards for long periods.

The patients transferred to observation wards were those with (*a*) respiratory insufficiency, (*b*) pharyngeal paralysis, (*c*) a combination of these, and (*d*) rapidly ascending paralysis, in which respiratory or swallowing insufficiency could be expected to develop in a matter of minutes.

The aim of the minute-to-minute observation was to correlate clinical findings with laboratory values and thereby to obtain indications for the different types of treatment which could be offered—namely, postural drainage, mechanical respirator (tank or cuirass), tracheotomy, and tracheostomy with manual intermittent positive-pressure ventilation.

Where there was difficulty in swallowing we looked for secretions in the pharynx, and also got the patient to swallow a teaspoonful of water from time to time.

Respiratory insufficiency presents itself in the form of paralytic cough, weak or paradoxical excursions, and, in the later stage, excitement, confusion, elevation of blood pressure, and cyanosis. Further secretions may accumulate in the airways and atelectasis occur. It was of some value to follow the changes in vital capacity with a small spirometer, which could be used at the bedside.

The correlation between clinical observations and laboratory findings is mentioned in detail by Astrup *et al.* at page 780 of this issue.

The Tracheostomy

Indications for tracheostomy differ. In the deeply asphyxiated patient it is done as an urgent measure. Tracheostomy is carried out in the bulbar type with vagal paralysis if the airways cannot be cleared by suction and drainage; in the bulbo-spinal type with swallowing paralysis, and stagnation of secretions; in encephalitis, when the patient is excited or comatose or the respiration irregular with long periods of apnoea; and in respiratory paralysis,