Treatment of severe respiratory insufficiency in diffuse chronic lung disease by tracheostomy and intermittent positive pressure ventilation (IPPV)

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RESPIRATORY failure resulting from diffuse chronic lung disease has been treated by artificial ventilation for 15 years (Boutourline-Young & Whittenberger, 1951; Mollaret *et al.*, 1958; Munck, Sund Kristensen & Lassen, 1961; Lassen, 1962; Lissac & Pocidalo, 1963; Sluiter, Blokzijl & van Dijn, 1963; Herzog & Engelhart, 1964; Bradley, Spencer & Semple, 1964; Bates *et al.*, 1965; Bydlowski *et al.*, 1965; Castaing *et al.*, 1965; Grendahl & Refsum, 1965; Sadoul, Aug & Gay, 1965). This active form of therapy has gained acceptance in centres equipped with technical and staffing facilities adequate for its successful application, but other forms of therapy and prophylaxis are employed in less well equipped centres, particularly as individual physicians hold widely divergent opinions on the merits of artificial ventilation and the indications for its use. This controversy cannot as yet be resolved on the basis of general principles.

After 1955 (Bjørneboe *et al.*) the treatment of this disease at the Blegdamshospitalet has been centralized in the Department of Epidemiology where IPPV had been extensively used during the poliomyelitis epidemics in 1952-53 (Lassen, 1956); the principles of prolonged artificial ventilation evolved during that period were applied.

Clinical material

Patients were placed in three broad clinical categories because the functional capacity of these subjects prior to the acute exacerbation provided the only significant prognostic criterion as was pointed out previously (Munck *et al.*, 1961). Tables 1 and 2 show that only two out of fifty-six patients with fair respiratory reserve died whereas fifteen of the eighty more severely disabled patients could not be saved.

Group A. Patients able to undertake full or part-time work before the exacerbation that led to respiratory failure.

Group B. Patients unable to work, but able to look after personal requirements such as the household shopping.

Group C. Patients so severely incapacitated as to be

TABLE 1
Distribution according to age at the time of tracheostomy and respiratory reserve prior to acute exacerbation

Age	Group A	Group B	Group C	Total
Less than 50 years	7 (1)	7 (0)	5 (1)	19 (2)
50-60 years	22 (1)	21 (2)	6 (0)	49 (3)
60-70 years	25 (0)	19 (5)	7 (3)	51 (8)
More than 70 years	2 (0)	12 (4)	3 (0)	17 (4)
Total	56 (2)	59 (11)	21 (4)	136 (17)

Deaths during respirator treatment are indicated in parentheses. For definition of groups, see text.

TABLE 2
Survival rates for patients treated by tracheostomy and artificial ventilation for respiratory insufficiency in chronic diffuse lung disease

Time	Total (%)	Respiratory reserve before acute exacerbation		
		Group A (%)	Group B (%)	Group C (%)
Respirator	88(136)	96(56)	81(59)	81(21)
Discharge	75(136)	86(56)	71(59)	57(21)
3 months	65(136)	73(56)	66(59)	38(21)
6 months	54(136)	70(56)	53(59)	19(21)
9 months	51(134)	69(55)	44(59)	20(20)
1 year	44(130)	64(55)	35(55)	15(20)
2 years	36(101)	49(45)	32(41)	7(15)
3 years	39 (85)	58(36)	32(37)	0(12)
4 years	35 (62)	55(29)	26(23)	0(10)
5 years	24 (41)	44(18)	15(13)	0(10)
6 years	15 (26)	23(13)	14 (7)	0 (6)
7 years	14 (14)	40 (5)	0 (6)	0 (3)
8 years	11 (9)	33 (3)	0 (4)	0 (2)
9 years	0 (2)	0 (0)	0 (2)	0 (0)

The figures in parentheses indicate the number of patients observed in the group concerned.

unable to leave their beds or their rooms. A few of these had permanent tracheostomies and a few more used oxygen therapy at home.

Treatment of severe respiratory insufficiency

TABLE 3

A. Oxygen saturation, CO₂-tension and pH of arterial blood before, during and after artificial ventilation

	Group	Oxygen saturation (%)	CO ₂ tension (mmHg)	pH
Shortly before tracheostomy, spontaneous respiration, plus oxygen	A	65.7 ± 3.0(35)	82.5 ± 3.9(36)	7.259 ± 0.017(36)
	B	68.2 ± 2.8(42)	87.1 ± 3.7(43)	7.253 ± 0.021(42)
	C	66.0 ± 4.6(17)	77.2 ± 4.7(17)	7.299 ± 0.026(17)
During respirator treatment	A	92.1 ± 0.7(53)	51.3 ± 1.4(53)	7.441 ± 0.010(53)
	B	92.9 ± 1.0(56)	48.6 ± 1.1(57)	7.438 ± 0.010(57)
	C	93.9 ± 0.8(18)	47.6 ± 3.4(18)	7.450 ± 0.018(18)
After treatment, spontaneous respiration, no oxygen	A	85.3 ± 1.1(43)	47.9 ± 1.2(43)	7.408 ± 0.008(43)
	B	85.4 ± 1.1(35)	50.2 ± 1.6(35)	7.412 ± 0.008(35)
	C	84.0 ± 3.0(10)	47.0 ± 2.7(10)	7.428 ± 0.020(10)

B. Oxygen saturation, CO₂-tension and pH of arterial blood before tracheostomy (spontaneous respiration plus oxygen) in: (a) patients who die during admission or within 6 months after discharge, and (b) patients alive 6 months after discharge

	Oxygen saturation (%)	CO ₂ -tension (mmHg)	pH
Death during admission or within 6 months	64.4 ± 2.5(45)	79.4 ± 3.2(46)	7.275 ± 0.018(45)
6 months survival	69.1 ± 2.7(49)	87.6 ± 3.5(50)	7.254 ± 0.016(50)

The figures are mean values. The standard error of the mean is given. The figures in parentheses are the number of observations.

TABLE 4
Mental state of the patients before tracheostomy

	Group A	Group B	Group C	Total	Deaths during IPPV	Deaths 6 months after discharge
Conscious	3	6	1	10	0	4
Somnolent, quiet	20	28	11	59	9	31
Somnolent, agitated	6	4	1	11	2	4
Comatose	19	17	8	44	5	20
Not recorded	8	4	0	12	1	3

TABLE 5
Presence of oedema, cor pulmonale and obesity correlated to death or survival of the patients

	Deaths during IPPV	Other deaths within 6 months	Patients surviving 6 months	Total
Oedema ≤ 3 months	2	10	14	26
Oedema 3–12 months	1	6	5	12
Oedema ≥ 12 months	5	7	11	23
Cor pulmonale	6	21	31	58
Obesity	3	6	10	19
Total No. of patients	17	45	74	136

The clinical condition during the acute exacerbation, the results of arterial blood gas analysis (Table 3), the mental state of the patients (Table 4) and the presence of cor pulmonale or oedema (Table 5) did not form reliable guides to prognosis and the length of the previous history of the disease did not correlate with the mortality. In fact Table 6 shows

TABLE 6
Duration of lung symptoms correlated to death or survival of the patients

	Deaths during IPPV	Other deaths within 6 months	Patients surviving 6 months	Total
Lung symptoms < 10 years	7	12	20	39
Lung symptoms 11–20 years	4	16	20	40
Lung symptoms > 20 years	6	12	33	51
Not recorded	0	5	1	6
Total	17	45	74	136

that many patients with symptoms for less than 10 years progressed rapidly to a fatal outcome. These findings are similar to those of Sluiter *et al.* (1963).

All 111 patients treated had chronic bronchitis, usually for more than 10 years (Table 6) with varying degrees of pulmonary emphysema and fibrosis. Most of the patients were 50–70 years of age

(Table 1) and there were five times as many men as women. Fifty-eight patients had electrocardiographic evidence of cor pulmonale and more than half were in heart failure when treatment was commenced (Table 5).

The commonest cause of the acute exacerbation (Table 7) was infection; other causes were lung

TABLE 7
Causes of deterioration necessitating tracheostomy

	Group A	Group B	Group C	Total	Deaths during IPPV	Total deaths 6 months after discharge
Total	56	59	21	136	17	62
Acute bronchitis; acute pneumonia	38	35	10	83	12	37
Asthma	2	2	0	4	1	1
Morphine	8	2	1	11	0	2
Oxygen	8	7	4	19	2	11
Other causes	7	16	9	32	3	17

infarcts, bleeding gastroduodenal ulcer, alcohol intoxication, pneumothorax, diabetic precoma, hepatic coma, barbiturate intoxication, pancreatitis, tachycardia and heart failure. In several cases, the cause of the acute exacerbation could not be determined. In a number, incorrect diagnosis such as lung oedema for which opiates were administered, excessive oxygen therapy due to inexact flowmeters and insufficient supervision and other similar avoidable factors contributed to deterioration. Finally, a number of patients who are not considered further in this report were treated by oral intubation for 24-48 hr, aspiration of endobronchial secretions and intermittent positive pressure ventilation, and these patients avoided tracheostomy. In most of the patients who required tracheostomy well-supervised conservative treatment had failed to prevent deterioration.

Clinical condition before tracheostomy

The clinical condition during the acute episodes was so grave, that survival was considered improbable without artificial ventilation. In ten cases the mental state of the patients was relatively unaffected but one-third of the cases were in coma, and the remaining patients were somnolent (Table 4). Their arterial blood gases (breathing oxygen-enriched mixtures via a nasopharyngeal catheter) are presented in Table 3. The mean values were very similar in groups A, B and C with oxygen saturations between 66% and 68%, $Paco_2$ about 80 mmHg, and pH between 7.25 and 7.30. We see from this that

mental changes and arterial blood gas alterations formed the major indications for treatment by artificial ventilation.

Treatment by tracheostomy and IPPV

Tracheostomy was usually performed by an ear, nose and throat surgeon with the aid of cyclopropane or halothane anaesthesia administered by an anaesthetist. All patients were intubated with an orotracheal tube and ventilation was controlled with the aid of a muscle relaxant during the operation of tracheostomy. A transverse skin incision was preferred and the thyroid gland was divided if necessary. Fenestration of the anterior wall of the trachea was made at the level of the second or third tracheal ring and a large size Rsch tube was used. Arterial hypotension occurred at this stage in a number of patients and most often responded to treatment by volume-expanders (Dextran) and occasionally, vasopressors.

A stomach tube was passed initially in most cases as abdominal distension was not infrequent, and fluid could usually be aspirated from the stomach. This abdominal distension disappeared 12-48 hr after tracheostomy: the patients regained consciousness and were allowed to drink and eat. A Lundia respirator which is volume-cycled and functions at a preset rate was used to ventilate the lungs with 40-60% oxygen and a minute volume of 5-6 litres. After some hours, ventilatory volumes were increased and oxygen concentration reduced. Blood pressure, pulse rate and respiratory minute volume were monitored hourly and the efficiency of ventilation was estimated by measuring arterial pH, standard bicarbonate, Pco_2 and oxygen saturation. The patients were shifted from the supine position several times a day and lung physiotherapy was carried out twice a day. Drainage of the tracheo-bronchial tree in head-down or prone positions was not used as the patients responded badly to these positions. Tracheal secretions were aspirated with sterile catheters when necessary and the appropriate antibiotics were administered when infection and fever were present. Antibiotics were never used prophylactically.

Early mobilization of the patients was considered essential and they were sat out of bed from the 2nd day after tracheostomy. They were weaned off the respirator as early as was feasible by allowing increasing periods of spontaneous respiration. When adequate spontaneous respiration was possible, the cuffed tracheostomy tube was replaced by a silver cannula. The inspired atmosphere was humidified and in some instances enriched with oxygen. The cannula was stoppered for increasing periods and when there was sufficient evidence that secretions could be expectorated without the aid of endotracheal aspiration, the cannula was removed.

Results

One hundred and thirty-six episodes of respiratory failure occurred in 111 patients; the number of

patients treated has increased and since 1960 about twenty tracheostomies are carried out yearly (Table 8). Twenty-one patients needed repeat tracheostomies (Table 9). Seventeen patients died in the acute episode and three became respirator-dependent.

TABLE 8
Number of tracheostomies per year

Year	No.
1955	1
1956	1
1957	2
1958	5
1959	10
1960	18
1961	14
1962	23
1963	18
1964	23
1965	21
Total	136

TABLE 9
Number of tracheostomies and re-tracheostomies

	Male	Female	Total
No. of patients with one tracheostomy	73	17	90
No. of patients with two tracheostomies	17	1	18
No. of patients with three tracheostomies	2	0	2
No. of patients with four tracheostomies	1	0	1
Total	93	18	111

Total number of tracheostomies 136.

TABLE 10
Survey of patients who died during respirator treatment

Year	Group	Age (yr)	Sex	Duration of respirator treatment (days)	Cause of death
1955	B	63	M	2	Sudden and unexpected death. Autopsy showed acute and subacute pancreatitis, cysts in kidneys and liver and a small lung infarction.
1956	B	66	M	52	Pneumonia.
1958	C	62	M	19	Numerous lung abscesses with <i>Staph. aureus</i> and <i>P. aeruginosa</i> .
1959	B	67	M	70	Slow deterioration. Increasing oedema and falling blood pressure. Two weeks before death a tracheo-oesophageal fistula. No autopsy.
1959	C	47	M	15	Septicemia with <i>E. coli</i> and <i>Klebsiella</i> . Severe asthma-bronchitis; pyonephrosis with calculi.
1960	B	70	M	12	Pneumonia and cardiac decompensation.
1961	C	65	M	31	Pneumonia with atrial fibrillation and hypotension.
1962	B	57	M	13	Pneumonia with <i>P. aeruginosa</i> and <i>Staph. aureus</i> .
1963	A	56	M	21	<i>P. aeruginosa</i> pneumonia with empyema, shock, anuria and emboli to the basilar artery.
1963	B	65	M	153	Slow deterioration. Autopsy gives no definite explanation.
1964	A	44	F	10	Tracheostomized the day before admission at another hospital, where she developed brain stem attacks. Autopsy showed severe hypoxic brain lesions.
1964	B	74	M	5	Sudden and unexpected death after tracheal suction. No definite explanation of death at autopsy.
1964	B	55	M	1/3	Hypotension after second tracheostomy.
1965	B	77	M	24	High fever at tracheostomy. After tracheostomy prolonged arterial hypotension and various cerebral symptoms.
1965	B	72	M	23	Large lung embolus.
1965	B	72	M	9	<i>P. aeruginosa</i> pneumonia and septicemia.
1965	C	62	M	1/8	Atrial fibrillation and tachycardia before tracheostomy. After tracheostomy extreme hypotension. Autopsy showed pneumonia.

In the other 116 episodes the patients were weaned off the respirator. As tracheostomy and artificial ventilation were employed in life-threatening situations, the immediate results of the techniques must be considered satisfactory and their adequacy is reflected in the normal results of arterial blood gas analysis during treatment (Table 3).

The causes of death during treatment are shown in Table 10. In two instances sudden unexpected death occurred for which no definite cause was found at autopsy, three patients became hypotensive and died shortly after tracheostomy and the most common cause was lung infection usually with *Staphylococcus aureus* or *Pseudomonas aeruginosa*. Most patients were weaned off the respirator rapidly (Table 11)

TABLE 11
Duration of respirator treatment

	Group A	Group B	Group C
< 1 week	26 (0)	13 (3)	5 (1)
1-4 weeks	24 (2)	29 (5)	7 (2)
> 4 weeks	6 (0)	17 (3)	9 (1)
Total	56 (2)	59(11)	21 (4)

No. of deaths during respirator treatment is given in parentheses.

but thirty-two patients required assisted ventilation for at least part of the day for more than 4 weeks. Four of these died and three are respirator-dependent cripples but the other twenty-five were eventually weaned off their ventilators.

Complications during treatment

A list of complications is presented in Table 12.

TABLE 12
Complications during respirator treatment

	Group A	Group B	Group C
Total No.	56	59	21
Fall of blood pressure to less than 90 mmHg systolic	7	23	6
Anoxic kidney damage	1	1	1
Twitchings or convulsions	11	8	7
Bleeding from tracheostomy	3	1	0
Subcutaneous emphysema	3	2	0
Gastric retention or abdominal distension	5	12	1
Haematemesis/melaena	2	3	2
Pneumonia	6	10	4
Atelectasis	2	0	0
Lung infarction	1	3	0
Thrombophlebitis	3	1	0
Stenosis of trachea	2	1	0
Dilatation of trachea	0	1	0
Tracheo-oesophageal fistula	0	1	0

Systemic hypotension immediately after tracheostomy and paralytic ileus particularly in patients with poor respiratory reserve were the commonest complications. Twitching occurred but convulsions were rare if overventilation was avoided initially.

Results of long-term follow-up

Patients were returned to medical wards for rehabilitation after the tracheostomy was closed. About half the patients were eventually discharged with the same respiratory reserve as before the acute exacerbation but in the rest there was marked diminution of the reserve. Seventeen patients deteriorated during their stay in the medical wards after closure of the tracheostomy and eventually died in cardio-respiratory failure for which IPPV was not considered indicated.

Table 2 shows that 50% of group A patients survived after 2-4 years whereas 50% of group B patients died within 9 months and only 50% of group C survived the admission.

Discussion

Proper artificial ventilation is very effective in resuscitating a patient with chronic lung disease from the life-threatening condition of severe respiratory insufficiency.

The treatment with tracheostomy and IPPV ventilation as described in this paper has been chosen as a standard treatment, when conservative regimen is inadequate. In the practical organization of therapy there are definite advantages in adhering to one fairly simple method, familiar to all members of the staff. Further conditions for tracheal suction and effective ventilation are optimal.

The procedure of tracheostomy entails in itself a very small risk, but undoubtedly the unavoidable bacterial colonization of the stoma represents a serious risk of lung infection, particularly with endemic hospital flora of *P. aeruginosa* and *Staph. aureus* (Gotsman & Whitby, 1964). The larger the ward, the greater the risk of cross-infection.

The other significant complication—hypotension in the tracheostomy period—was usually mastered, and should be avoidable in most cases by close supervision and application of the measures mentioned. Both of these complications, as well as the fatalities during the tracheostomy period, were mainly seen in patients with a small respiratory reserve.

Might a less drastic therapy bring about equally satisfactory results? In recent years the use of analeptics (Sadoul, 1965) and acetazolamide (Gay & Cross, 1965) has had several advocates as a valuable improvement of the conservative therapy. Our own experience does not allow us to comment on the use of these drugs.

Tank respirators are still used by some authors (Grendahl & Refsum, 1965) who stress the advantages of avoiding tracheostomy. However, in patients who are comatose and present problems of secretions in the airways, this treatment is hardly suitable. Furthermore the ventilatory capacity of most commercially available tanks is limited, and even with free airways a sufficient ventilation of patients with low compliance may prove impossible. The mobilization of the patient—an essential part of the treatment—is difficult as is nursing and last, but not least, treatment with tank respirator is more uncomfortable than tracheostomy and IPP ventilation.

IPP ventilation on a mask has given excellent results, especially in the hands of French teams (Sadoul *et al.*, 1965). The series of patients treated seem to differ somewhat from those in the present series in comprising few comatose patients. We are not convinced that mask-ventilation would have been effective in our patients, but we intend to undertake a trial of the method, and we hope that our scepticism will be unjustified.

Recently the use of oro-tracheal or naso-tracheal intubation has been recommended in the management of respiratory failure, instead of tracheostomy. The newer types of soft plastic tubes seem to be well tolerated, even for longer periods, and the results of this treatment in larger series of patients will be met with interest.

Finally, tracheostomy or fenestration without intubation or cannulation, performed by a special technique (Rockey *et al.*, 1961), has been advocated. The avoidance of foreign bodies eliminates the inherent risks of dilatation and stenosis of trachea and supposedly reduces the risk of infection. On the other hand, at least in our type of patients, it is to be feared that prolonged periods of hypoventilation with weak cough reflexes would present equally severe risks.

From the description of our patients it will be seen that tracheostomy and IPPV was used in many patients with a small respiratory reserve. In fact, most patients admitted to the department with life-threatening respiratory failure were treated by this means. It is remarkable that it was possible to a large extent to bring these severely incapacitated patients back on spontaneous respiration. It also appears from the report that the condition of a number of these patients later again deteriorated, and in some cases respirator treatment was not re-instituted. The decision to be taken in those cases is dependent on whether or not the condition of the patient can be regarded as reversible or not. In the first case every effort has to be made to tide the patient over a life-threatening situation. In the latter case such effort may be an unjustified intervention that prolongs the intolerable ultimate phase of an incurable disease.

Between these two extremes a category of patients

who might benefit from permanent or intermittent respirator assistance, and who might be able to lead a tolerable life in spite of their dependence on assisted ventilation, is emerging. Such treatment might be imagined along the lines followed in the care of patients with respiratory paralysis after polio (Engberg, 1961). The social and medical aspects are complicated, and the care of such patients will add to the responsibilities of medical personnel engaged in the treatment of patients with chronic lung disease.

Summary

Data are given concerning 136 consecutive episodes of severe respiratory insufficiency in 111 patients with chronic diffuse lung disease, treated with tracheostomy and artificial ventilation with a Lundia respirator. The importance of tracheal intubation prior to tracheostomy, large cuffed tubes, low initial ventilatory volumes with oxygen-enriched air and early mobilization is stressed. The results of treatment are given, and it is shown that the outcome depends on the respiratory reserve before exacerbation and not on the age or clinical state of the patient at tracheostomy, nor on laboratory data such as blood gas analysis, ECG, etc. Two patients among fifty-six with some working ability before tracheostomy died during respirator treatment, while fifteen out of eighty more incapacitated patients died. The causes of death and the non-lethal complications during treatment are surveyed. Other possibilities of active treatment are reviewed and discussed briefly.

A follow-up study showed 50% survival after 2-4 years in patients with some working ability before the exacerbation. The corresponding figure was 9 months in the group unable to work, but able to manage personal requirements. In the third and most disabled group of patients one-half died in hospital.

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Discussion to the paper by H. Sund Kristensen, O. Jessen and K. Rasmussen

FINNEGAN. I would like to illustrate the respirator treatment of acute or chronic lung disease at Whiston Hospital in the period from May 1962 to January 1966.

Twenty-two men and three women developed acute cardiorespiratory illnesses and became moribund despite

acute illness consisted of mechanical ventilation through a tracheostomy, antibiotics and metabolic care.

The mean duration of ventilation was 10.6 days and the range was 12 hr to 35 days. Nine patients died during the period of respirator treatment; the causes of death are listed in Table A2.

Four patients died within 1 year and three patients within 2 years of treatment. Nine of the original group are still alive, six with moderate disability and three with severe disability (Table A3).

TABLE A1
Respirator treatment of acute-on-chronic lung disease (May 1962 to January 1966)

Age (years)	No. of patients
40-50	6
50-60	11
60-70	8
Total	25 (22 men + 3 women)

TABLE A2
Deaths during respirator treatment

Causes	No. of patients	Days of treatment
Failure of resuscitation	2	1
Massive pulmonary infarcts	1	3
Undiagnosed carcinoma of bronchus	1	6
Perforated duodenal ulcer	1	24
Renal failure	1	11
Cross-infection	2	10 and 11
Bronchopneumonia	1	13
Total	9	

antibiotics, controlled oxygen and chest physiotherapy. All the patients had acute-on-chronic cor pulmonale due to chronic bronchitis, bronchiectasis or pulmonary thrombo-embolism (Table A1). The treatment of the

TABLE A3
Follow-up of nine patients surviving for 6 months to 3 years

Disability	Grade of dyspnoea	No. of patients
Moderate	1-2	6
Severe	3	3

The criteria for selection, the management and the complications are similar to those reported by Dr Kristensen.

ROBERTSON. I note that Dr Kristensen has had some complications with tracheal stenosis after IPPV, and wonder whether this was because the cuff of the rubber tube was blown up too tight, perhaps by the nursing staff, since this has been the experience of Mr Peter Gibson of Perth, Australia.

Finally, may I ask whether anyone in the audience has any properly controlled evidence that either methyl- or acetyl-cysteine is effective? I have heard many speakers praise its usefulness, but I am more impressed by the absence of any properly controlled trial.

SCARROW. Pulmonary angiography was employed to