THE PREVENTION AND TREATMENT OF CO_2 NARCOSIS IN A GENERAL MEDICAL UNIT*

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Campbell[1] pointed out that uncontrolled oxygen administration to patients with respiratory failure, during acute chest infections, can be followed by reduced ventilation. This, in turn, can very easily lead to further elevation of the arterial CO_2 tension (PCO_2), resulting ultimately in the state of CO_2 narcosis. He stated: 'Such patients must have oxygen: otherwise many are left to combat pneumonia as inadequately oxygenated as a man on the South Col. On the other hand, incautious administration of oxygen can plunge some patients into acidemia comparable with the most severe diabetic coma.' Apart from the danger of severe hypoxia which will follow subsequent interruption of the oxygen supply, the failure to ventilate the lungs properly, and to cough, causes closure of terminal lung units, which may result in permanent damage, not to mention the danger of prolonged coma, in patients who already have infected lungs. For these reasons, CO_2 narcosis requires early recognition and energetic treatment.

By now the condition should be well known and with proper management should not often occur. Nevertheless, it continues to do so, sometimes despite the utmost care, but often, regrettably, because of ignorance. Acute respiratory infection in the course of chronic obstructive airways disease is one of the commoner causes of admission to beds catering for an ageing population, and some degree of elevation of PCO_2 in this situation is common. However, in the unit in which our cases were treated, respiratory acidosis of a degree sufficient to cause coma has fortunately been rather infrequent, and, indeed, a knowledge of its likelihood is the best prevention in the treatment of respiratory infections.

This unit is one of 3 general medical units in the department, and comprises 40 beds in which there have been 6 cases of CO_2 narcosis during the past 18 months. These have presented in ways which have called forth practically the entire spectrum of therapeutic responses, and have been most instructive in the management of this condition. The results have been as good as can be expected, and it is the aim of this paper to present the relevant details of the cases and to discuss the management of severe respiratory insufficiency in chronic lung disease under the circumstances prevailing in an ordinary general hospital.

These cases were managed in the environment without facilities for sophisticated pulmonary function studies, and therefore an environment reproducible in any general hospital with a reasonable standard of equipment.

CASE REPORTS

These will be presented in therapeutic groups rather than in chronological sequence.

The 2 cases in which either tracheostomy or endotracheal intubation was performed, will be described first, followed by 4 in which intensive conservative measures short of these steps were used.

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Case 1

F.J.T., aged 68, a White male pensioner with a history of chronic bronchitis and repeated chest infections, was admitted on 19 October 1967 with a diagnosis of bronchopneumonia following an increase in sputum, cough and dyspnoea over the preceding 2 days. The sputum had also changed in character from white and mucoid to thick, tenacious and yellow. On examination, he was mildly dyspnoeic at rest, the blood pressure was 150/90 mm.Hg and he was slightly cyanosed. There were no signs of congestive cardiac failure, the apex beat was impalpable and no cardiac dullness was elicited upon percussion. There were 2 rather soft heart-sounds and no murmurs were heard. The chest appeared somewhat voluminous and there were coarse crepitations heard at both bases posteriorly, with expiratory rhonchi heard over the right middle lobe anteriorly. The ECG showed some anterior ischaemia, an axis of $+90°$ and no evidence of cor pulmonale. The X-ray of the chest was delayed some days and then showed some cardiac enlargement, basal congestion of the lungs and an appearance consistent with emphysema.

He did not appear very ill on the evening of admission, and treatment with tetracycline, 250 mg. every 6 hours, was started. During the night he became a little more breathless and was given oxygen by nasal catheter at an unknown flow-rate. The next morning he was disorientated and semicomatose, with warm extremities, a coarse tremor and an aimless picking at the bedclothes. He was still cyanosed. No papilloedema was noted.

The arterial PCO_2 by Astrup micro-electrode technique was 63 mm.Hg, with a pH of 7·29, a standard bicarbonate of 33·5 mEq./litre, actual bicarbonate of 29 mEq./litre, buffer base 51 mEq./litre and a base excess of +1 mEq./litre. An intravenous infusion of 5% dextrose-water containing 8 ml. of nikethamide in 150 ml. was given over one hour and repeated twice. Oxygen was given via an Edinburgh mask at 1½ litres/min. The effect of the nikethamide infusion was fairly dramatic and he regained consciousness after about 30 min. due to improved ventilation. Additional assistance was provided by the manual operation of an 'Ambu bag' resuscitator and mask for 5 minutes every ½ hour.

After initial improvement, his condition deteriorated despite a further intravenous infusion of nikethamide during the course of the afternoon. By 5 p.m. he had developed obvious under-ventilation and was comatose. Because he had been leading an active outdoor, albeit somewhat breathless, existence on his brother's farm before the present episode, it was decided to perform a tracheostomy and to assist his ventilation with a Bird Mark 8 respirator. After the insertion of a cuffed Portex tracheostomy tube, he was admitted to the intensive care unit.

At first he seemed more comfortable, with his respiration partly controlled by a low sensitivity setting and alternating doses of amylobarbitone, 100 mg. and promazine, 50 mg., one or other being given every 3 hours, but

on the following day the machine was set for patient-triggering so that his own ventilatory efforts were now being merely assisted.

The morning after tracheostomy his PCO_2 was found to be 42 mm.Hg. After 4 days of assisted ventilation and tracheostomy toilet he was weaned from the respirator without difficulty. Three days later the tracheostomy tube was removed, and at the end of a month in hospital the wound had healed and he was discharged. At the time when the tracheostomy was performed, the antibiotic was changed to cephaloridine 1 G every 6 hours. A week later a specimen of sputum aspirated from the tracheostomy was submitted for bacteriological examination and *B. proteus* was cultured, sensitive, among other antibiotics, to ampicillin but resistant to cephaloridine, and the patient was thereupon given a course of ampicillin. One week after removal of the tracheostomy tube an Astrup estimation showed an arterial PCO_2 of 26 mm.Hg, compared with a figure of 32 mm.Hg obtained by the rebreathing method.

Case 2

M.P., a housewife aged 26, had been asthmatic since childhood but had been free of severe attacks in recent years. A few days before admission she had caught a cold and subsequently developed a wheeze. She lapsed into status asthmaticus and arrived in the casualty department on the day of admission asking for an injection. The casualty officer administered Aminophyllin, 0·5 G intravenously, Alupent, 0·5 ml. subcutaneously and chlorpromazine, 50 mg. intramuscularly, and was not unduly worried by her condition. Shortly afterwards she became confused. This was ascribed by the staff to hysteria, until she went into coma a few minutes later, emphasizing the frightening rapidity with which these patients can collapse.

On examination she was deeply cyanosed and unconscious, with a pulse of 120/min., multiple extrasystoles and a blood pressure of 120/60 mm.Hg, and she was obviously under-ventilating; in fact she seemed to be hardly breathing at all. On auscultation she had high-pitched inspiratory and expiratory rhonchi, the latter more marked, and there was hardly any audible air entry. An endotracheal tube was immediately passed and connected to an 'Ambu bag' resuscitator and a supply of oxygen. An arterial blood sample taken at this stage showed a PCO_2 of 100 mm.Hg. In retrospect it seems that extreme bronchospasm had caused cerebral hypoxia with depression of the respiratory centre, allowing an already elevated PCO_2 to become extremely high.

The patient was hurriedly transferred to the intensive care unit, being manually ventilated en route. The original endotracheal tube was too small and allowed some leakage when connected to the Bird Mark 8 respirator, and a larger one had to be inserted rapidly. Intermittent positive-pressure ventilation (IPPV) was now quite satisfactory, with a sensitivity setting such that the patient triggered the machine herself.

Other treament had by now included 500 mg. of hydrocortisone intravenously in several aliquots, 0·5 G of Aminophyllin in 500 ml. 2·74% sodium bicarbonate solution, by intravenous infusion, and the inception of an antibiotic regime. The Bird respirator was initially run off pure oxygen; later the air-mix button was pulled out, giving an approximate concentration of 40%.

As bronchospasm relaxed, the patient improved rapidly and began to object to the tube, which was removed after 2½ hours. During this time the respirator had been set at a low flow-rate and the minimum pressure consistent with adequate ventilation, to obviate the risk of haemodynamic disturbance and shock from air trapping.

Her condition improved progressively on further parenteral, and later oral, steroids, Aminophyllin suppositories and the occasional use of an Alupent inhaler. She was returned to the general ward 48 hours after admission. The morning following admission to the intensive care unit the arterial PCO_2 was 33 mm.Hg by the Astrup technique and estimated at 42 mm.Hg by the rebreathing method.

Case 3

A.C.-S., a 74-year-old male, was admitted on 2 July 1966 with bronchopneumonia and congestive cardiac failure. He had had chronic bronchitis and emphysema for many years, and during the year preceding the present episode he had required digitalization and diuretic therapy. On examination he was comatose, in gross congestive cardiac failure, with slight cyanosis and warm extremities; no papilloedema was noted. X-ray examination of his chest showed patchy consolidation and small bilateral basal effusions. ECG showed atrial fibrillation and anterior ischaemia. He had been given oxygen by nasal catheter at an unknown flow-rate while awaiting admission and on the way to the ward.

Results of other relevant investigations were as follows: haemoglobin 14·6 G/100 ml., WBC 10,600/cu.mm., blood pH 7·16, arterial PCO_2 105 mm.Hg, standard bicarbonate 22 mEq./litre, actual bicarbonate 36 mEq./litre. Penicillin, 1 million units, and streptomycin, 1 G *b.d.*, were commenced by intramuscular injection, and an intravenous infusion of 6 ml. of nikethamide in 150 ml. 5% dextrose-water was commenced, being given over 1 hour and repeated once. Following this, 2 ml. of nikethamide were given intramuscularly every hour for a further 3 doses. Oxygen was administered via a nasal catheter at 3 litres/min. in the absence of an Edinburgh or Venturi mask, and the patient was given IPPV with a Bennett respirator and a tightly fitting face-mask. This was given for 10 minutes every ½ hour, initially.

The question now arose whether to perform a tracheostomy for better tracheal toilet and more efficient ventilation, but because of the patient's age and the severity of his underlying disability this was decided against. Our conservatism was fully justified, as he improved perceptibly over the next 24 hours. Three days after admission his blood pH was 7·4 and the arterial PCO_2 was 43 mm. Hg. He was now fully conscious and lucid, though he was to remain for several months in hospital under treatment for his congestive cardiac failure before a satisfactory arrangement could be made for him to return to the care of his family and their doctor.

The danger of unrestricted oxygen was once again forcibly demonstrated, but the case also demonstrated that intensive conservative measures can be very successful. It is worth mentioning that he found the IPPV with a mask extremely uncomfortable in his confused and unco-

operative state. This was very distressing, both to him and his attendants, and in retrospect it might have been better to have passed a cuffed endotracheal tube transnasally, under sedation and a relaxant if necessary, and to have assisted or controlled ventilation continuously.

Case 4

J.O., a Coloured male aged 63, had suffered from chronic bronchitis and emphysema for a number of years and was admitted on 28 August 1966 in congestive cardiac failure following a recent chest infection. On examination he was obese and somewhat wheezy, with some cyanosis. The blood pressure was 160/110 mm.Hg. He had a regular pulse with a rate of 110/min., the apex beat was not palpable and the heart-sounds were distant and obscured by rhonchi and crepitations. The liver was felt 4 finger-breadths below the right costal margin and there was dependent oedema. The X-ray of his chest showed some cardiomegaly with a moderate-sized cyst in the right mid-zone, of probable emphysematous origin. The ECG showed right atrial and ventricular hypertrophy.

The results of other investigations were: haemoglobin 14 G/100 ml., WBC 6,000/cu.mm., serum urea 54 mg./100 ml., serum cholesterol 184 mg./100 ml. The sputum culture revealed no organisms. Treatment consisted of tetracycline, 250 mg. every 6 hours, frusemide, 20 mg. intravenously on alternate days, and chlorothiazide, 500 mg. daily. In addition he was digitalized. He responded slightly to treatment, but on the morning of 4 September was found to be semicomatose though he could be roused by loud speech and mildly painful stimuli. He had warm extremities and did not appear particularly cyanosed. There was no history of his having been given oxygen during the night, but a cylinder was at his bedside and he may well have helped himself. Signs of left lower-lobe pneumonia were found upon clinical examination and confirmed radiologically; the plasma pH was 7·24 and the arterial PCO_2 81 mm.Hg. Clinically, the appearance was that of CO_2 narcosis apparently precipitated by further infection of his chest. An infusion of 6 ml. of nikethamide in 150 ml. 5% dextrose-water was given over approximately 1 hour and repeated once. He was given erythromycin, 250 mg. every 6 hours, to which Orbenin, 500 mg. every 6 hours, was subsequently added. In addition to the nikethamide infusion his ventilation was further assisted for 5 minutes every ½ hour during the first day by means of an 'Ambu bag' resuscitator and mask.

It had been decided not to perform a tracheostomy on this patient because the severity of his preceding disability had been such that he had been too breathless to leave the house.

The next morning the arterial PCO_2 was 95 mm.Hg and it was 48 hours before he regained a fully conscious and lucid state. During this time the 'Ambu bag' was used for a short period approximately every hour, and, as he improved, he was encouraged to breathe deeply several times during each day. He was discharged 10 days later, only to be readmitted on 30 May 1967 in congestive cardiac failure, despite follow-up and supportive treatment as an outpatient. On this occasion he suffered a massive pulmonary embolus and died on his third day in hospital. This case is another example which justified a more conservative approach under these circumstances, as evidenced by his good response to initial treatment but his subsequent short survival.

Case 5

J.C.L., a White male aged 60, had fallen while carrying a kitchen knife and sustained a severe orbital laceration. He also suffered from severe obstructive airways disease of long duration and was employed in a sedentary capacity. He had never had an episode of congestive cardiac failure, but over the preceding 2 years he had become progressively more short of breath, until dressing himself had become an effort and he was being driven to work every morning by his wife. It had become clear that his disability was such that he would shortly have to resign from his job.

While undergoing a light general anaesthetic for the repair of his orbit, he sustained cardiac arrest, but was successfully resuscitated by open cardiac massage via an abdominal incision. As his pulse persisted at a rate of 140/min., he was digitalized. He was also given an intramuscular preparation of tetracycline. ECG showed sinus tachycardia, an axis of +90, but no specific evidence of cor pulmonale, while the X-ray of his chest showed a normal-sized heart, emphysematous lungs, a small left basal effusion and a flattened right diaphragmatic dome. On 21 March 1967, 15 days after admission, he became more breathless, and transfer to a medical ward was requested by the ophthalmologist. On examination his blood pressure was 160/90 mm.Hg, there were no signs of congestive cardiac failure, and he was moderately cyanosed and dyspnoeic at rest. No clubbing of the fingers was noted. The heart was obscured by voluminous lungs, and a moderate left basal pleural effusion was detected clinically.

Aspiration of the chest yielded 1,400 ml. of straw-coloured fluid. Aminophyllin suppositories, ampicillin, 500 mg. every 6 hours for 5 days, choline theophyllinate, 200 mg. *t.d.s.*, and breathing exercises were added to the previous treatment. His condition remained unchanged until 17 April, when he became even more breathless and cyanosed and developed severe bronchospasm which was not relieved by several intravenous injections of Aminophyllin. Prednisone was accordingly administered in a dose of 40 mg. daily, with the intention of tailing the dosage and withdrawal as soon as possible. With no help forthcoming from a sputum culture, another course of ampicillin was started, and he was given oxygen at 2½ litres/min. by nasal catheter. Though this promptly relieved the cyanosis, he became progressively more drowsy and the arterial PCO_2, measured by the Astrup technique, was 81 mm.Hg. An infusion of 8 ml. of nikethamide in 150 ml. 5% dextrose was given over a period of 1 hour, repeated once and followed by 2 ml. of nikethamide intramuscularly every hour for a further 3 doses. An 'Ambu bag' resuscitator and mask were applied for 5 minutes each half-hour, and IPPV with a Bennett respirator and mask, with Alevaire solution in the nebulizer chamber, was carried out for 3 sessions of approximately 10 minutes each in the first 12 hours. The patient resisted the latter 2 measures and was therefore placed in a tank respirator throughout the night. This appeared to im-

prove him greatly and he was fully conscious by the next day, and though a PCO_2 estimation was not repeated at this stage, under-ventilation and stupor did not again become a problem. Two or 3 10-minute sessions with the Bennett respirator and a tightly fitting mask were now given every day, and with familiarity and training the patient found this more acceptable. He claimed marked subjective benefit and this was continued for about a week.

He was gradually mobilized and spent, in all, a further 6 weeks in hospital. He was, eventually, able to maintain a pink colour at rest, but undue exertion occasioned cyanosis, suggesting a severe ventilation-perfusion imbalance. He has remained an outpatient since, now unable to work and using a home supply of oxygen at a low flow via an Edinburgh mask, after any slight effort.

Case 6

B.T., a White male aged 28, was admitted on 3 September 1967. He had had bronchiectasis since early childhood and had undergone a left lower lobectomy on 30 May 1965, which appears to have been somewhat ill-advised because he had been very breathless ever since, and appeared to have marked obstructive airways disease and copious sputum. Since the operation he had been in hospital for treatment of chest infections on 2 occasions before the present admission. On the second of these admissions, signs of congestive cardiac failure had been present and, following his discharge from hospital, he had been more or less confined to his bedroom. The present admission had been precipitated by contracting a cold which had occasioned a change in the character of his sputum from white to a tenacious yellow.

On examination there were no signs of congestive cardiac failure, his blood pressure was 110/60 mm.Hg, the apex beat could not be felt, there were 2 rather soft heart-sounds heard in all precordial areas, and no murmurs were heard. His chest had a hyperinflated appearance, with hyper-resonance on percussion. On auscultation there were râles and high-pitched, mainly expiratory, rhonchi throughout. ECG showed right atrial and ventricular hypertrophy, and the X-ray appearance of the chest was consistent with severe emphysema. The haemoglobin was 16 G/100 ml., and the WBC 7,400/cu.mm., with a normal stained film. He was given tetracycline, 250 mg. every 6 hours, choline theophyllinate, 200 mg. *t.d.s.*, and Aminophyllin suppositories, and oxygen was administered at 1½ litres/min. by Edinburgh mask. He improved slightly, but on 16 September his sputum again became thicker and more tenacious. *B. proteus* and a *Staphylococcus pyogenes* were grown, sensitive, among other antibiotics, to streptomycin and cephaloridine, respectively. Cephaloridine, 1 G every 8 hours, and streptomycin, 1 G *b.d.*, were commenced. His condition deteriorated and he was having great difficulty getting rid of his sputum. On 18 September a bronchoscopy was performed and much tenacious sputum was aspirated, with temporary improvement. Later that day he became drowsy and rather belligerent, but could be roused and responded to normal speech. The arterial PCO_2 was 60 mm.Hg and the oxygen saturation 85%. An infusion containing 8 ml. of nikethamide in 150 ml. 5% dextrose-water was instituted and given over a period of 1 hour. It was repeated and was followed by hourly intramuscular injections of 2 ml. of nikethamide for a further 4 doses. The Bennett respirator and a mask were tried, but he appeared quite unable to tolerate these, and he was accordingly given several sessions, each of some hours, in the tank respirator during the next few days. He improved slowly, becoming lucid and co-operative over the next 2 or 3 days, returning eventually to his usual intercurrent grade of disability. He was finally returned to the care of his family and their doctor and has not, at the time of writing, required readmission.

Of these 6 cases, one underwent tracheostomy, and in one endotracheal intubation was performed. All were given some form of ventilatory assistance. All 5 of the cases with chronic obstructive airways disease were given nikethamide, and there is no doubt as to its value in the over-all therapeutic effort. Tracheostomy and continuous IPPV are by no means always indicated, as will be discussed presently, other avenues of ventilatory assistance often being of considerable help. These are therefore worth exploring.

DISCUSSION

As the uncontrolled use of oxygen is often the most important factor in the evolution of hypercapnia and coma, it is perhaps best to begin any discussion on the prevention and treatment of CO_2 narcosis by considering briefly the methods by which oxygen administration may be controlled.

The Venturi mask[2] is designed so that a 2-litre flow of oxygen/min. entrains 50 litres of air which gives a concentration of oxygen in the inspired air of 24%. Correspondingly higher oxygen flows will give, of course, higher oxygen concentrations. A cheap and disposable mask on this pattern is manufactured by the Oxygenaire Company, known as the Ventimask, and this is available with different vent sizes, giving different oxygen concentrations on a 4-litre flow/min., which range from 24 to 33%.

Alternatively, one may use the cheaper Edinburgh mask,[3] which will control the oxygen concentration within the range of 22 - 41% according to the following table, supplied by the manufacturer.

TABLE I. OXYGEN CONCENTRATIONS

O_2 flow/min. (litres)	*O_2 concentration in inspired air %*
½	22 - 25
1	24 - 28
2	27 - 31
3	29 - 41

The advantages of this mask are its cheapness and simplicity, but it suffers from the drawback that it depends partly on the current set up by inhalation to entrain sufficient air. If the patient is under-ventilating at the outset, dangerous concentrations of oxygen can still develop.

These masks work according to the principle of high air-flow oxygen enrichment (HAFOE). However, in the absence of a mask of this design, a nasal catheter can be used and still has a place in oxygen therapy in chronic respiratory disease. Oxygen flows of 4, 6 and 8 litres/min. will give approximate oxygen concentrations of 30, 40

and 60%, respectively.[4] Flow-rates below 4 litres/min. can therefore often be administered safely, though it must be remembered that wide variations in alveolar and arterial oxygen enrichment have been shown using nasal catheters, and that from the clinical standpoint, therefore, great vigilance is needed if oxygen is to be used in this way. Finally, it is necessary to condemn the practice of giving intermittent oxygen in the early stages of respiratory failure. In the acute stage with hypercapnia, withdrawal of oxygen means that blood arrives in the lungs with a high PCO_2 and discharges the excess CO_2 into the alveoli, and this causes a further and dangerous decrease in an already reduced alveolar oxygen tension. Hutchison *et al.*[5] found that continuous controlled oxygen therapy may be required for a week or more in these cases.

Campbell's practice at Hammersmith Hospital[6] deserves the closest consideration. The arterial PCO_2 is measured rapidly by means of the rebreathing method in any patient admitted with a respiratory infection. If the PCO_2 is normal, there appears to be little danger of under-ventilation. If it is raised (say 50 mm.Hg or more), the patient is given 25% oxygen and is observed closely. Should no progressive hypoventilation occur, the O_2 concentration is increased to approximately 30%. If, however, progressive under-ventilation develops, arrangements are made for tracheostomy, while more intensive conservative measures are carried out. CO_2 narcosis might well have been prevented in cases 1 and 3 of this series had we paid more attention to the initial oxygen concentrations on first admission, and these 2 cases have been a sharp lesson that bears out Campbell's remark[7] that many of the studies which have been performed described patients who were much more ill than they ought to have been allowed to become before treatment was applied.

Lal[8] described 18 patients in respiratory failure during an English winter-period of 7 months. All had arterial CO_2 tensions of less than 80 mm.Hg on first admission. Lal felt that higher levels in other series might have resulted from the injudicious use of oxygen, while McNicol and Campbell[9] pointed out that, when breathing air, hypercapnia is unlikely to become dangerous. In fact, they presented physiological arithmetic to show that it was impossible for the arterial PCO_2 to rise much above 80 mm.Hg while breathing air alone. These authors quoted 81 cases which bore out their prediction.

This emphasizes the absolute necessity for controlled oxygen therapy to be the first form of oxygen therapy used on any patient with underlying chronic obstructive airways disease. HAFOE masks are therefore now a much commoner sight in our wards than they were a year ago.

It must not be imagined that tracheostomy and continuous assisted ventilation are an easy answer. Tracheostomy requires a surgeon and an operating theatre, and the condition in which these patients are sometimes presented to the surgeon is far from encouraging to even the stoutest-hearted anaesthetist. The complications can be frightening, and apart from those associated with the mechanics of the tracheostomy itself, these include serious infection which can spread to other patients, and gross intrathoracic haemodynamic disturbances from air-trapping in emphysematous lungs. This may lead to circulatory collapse, though this often responds promptly to intravenous volume expanders. Apart from these complications, the proper care of such a patient requires the expenditure of staff, time and equipment on a lavish scale. This hospital has recently opened an intensive care unit with ample allocation of trained nursing staff, sometimes at the expense of other parts of the hospital. With a shortage of sufficient medical staff, such cases can be a severe tax on available doctors, often also somewhat to the detriment of other patients.

Bearing all these factors in mind, it is essential to exercise some selection. Sund-Kristensen *et al.*[10] divided their cases into 3 groups:

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;

Group C—patients so severely incapacitated as to be virtually incapable of leaving their rooms.

Follow-up showed that approximately 50% of group A survived between 2 and 4 years, whereas 50% of group B died within 9 months and only about half of those in group C survived the admission. Some of these included respirator-dependent cripples. It seems therefore hardly justifiable to subject patients in group C to tracheostomy and IPPV, but this decision is one of the most difficult in clinical medicine. The fact that all 6 cases survived in the small series here reported, speaks, however, for the efficacy of other forms of treatment.

Tank respirators have recently still been used by some workers[11] which is perhaps some justification for the use of this method in 2 of our patients. However, this is very uncomfortable for the patient, and good nursing of a heavy adult is almost impossible inside an iron lung. IPPV on a mask has given excellent results in some hands and is certainly worth a trial in patients not suited to tracheostomy, or as a preliminary treatment to try to avoid this, though many clinicians remain sceptical.

Finally, the use of prolonged nasotracheal intubation has been advocated, and Kuner and Goldman[12] compare their mortality rate of zero in 20 cases with an approximate average mortality rate of 3% directly attributable to tracheostomy in a number of series. Some tubes were left *in situ* for up to 14 days in their series. This work deserves attention, and results of further work are awaited with interest.

The essence of management therefore amounts to recognition of the cases in which CO_2 narcosis is a possibility, and great care with the use of oxygen in these patients apart from other measures such as antibiotics and bronchodilators. Should under-ventilation and progressive CO_2 retention develop, intensive conservative treatment should be adopted and a decision taken as to the extent of further measures. In borderline cases there can be few more difficult decisions.

SUMMARY

The dangers of uncontrolled oxygen therapy are once again emphasized. Six cases of CO_2 narcosis are presented, all of whom survived their admission. One patient underwent tracheostomy, in another endotracheal intubation was employed and all were given some form of ventilatory assistance. In 2 cases

a Bird Mark 8 respirator was used and in 3 others a Bennett respirator and mask. A tank respirator was also used in 2 of these 3 cases. In one case an 'Ambu bag' resuscitator was the only device employed for ventilatory assistance. Nikethamide infusions were found useful and certainly aided recovery in those cases not subjected to intubation or tracheostomy. Finally, the prevention and treatment of the established condition are discussed, with special emphasis on methods of controlled oxygen therapy.

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ADDENDUM

Cases 3, 5 and 6 had all died within 18 months of the admissions described here, i.e. after 18, 10 and 6 months respectively. It was the poor prognosis in each of these cases which guided the choice of treatment. Their short survival and severe incapacity would appear to have justified the more conservative approach in that they were not subjected to tracheostomy.

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