MEDICAL GROUND ROUNDS

Parkland Memorial Hospital

April 11, 1968

SELECTED ASPECTS OF THE TREATMENT OF RESPIRATORY FAILURE

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Case I: [redacted]

[redacted] is a 72 y/o [redacted] male who was admitted for the fourth time to [redacted] on [redacted]/68. The patient was lethargic, irrational and in respiratory distress and was unable to give an adequate history. As best as could be determined, the patient had been demonstrated to have diffuse cortical atrophy in 1952 by pneumoencephalograms. That same year a chest x-ray was interpreted as showing severe pulmonary emphysema. He had last been seen in [redacted] in 1956 when he was successfully treated for a right middle lobe pneumonia. During the ensuing years he was known to have a chronic productive cough and rather marked dyspnea. These complaints had been more prominent during the 6 months prior to admission, and the patient had been loosing weight during that period of time. He had been noted by his family to be cyanotic on exertion intermittently for several months, but during the few days prior to admission he had been even more cyanotic, had an even worse cough, and he had perhaps had chills and fever.

On physical examination the vital signs were: T 98.6, P 108, R 28, BP 100/60. He was an emaciated, elderly, stuporous white male with obvious respiratory distress and cyanosis. He appeared wasted and markedly dehydrated. The chest had an increased AP diameter, marked kyphosis, hyperresonance to percussion, expiratory wheezes in the left posterior chest, fine rales in the right base that cleared with coughing, and decreased breath sounds. The heart was not detectable because of the pulmonary findings. The liver was palpated 3 cms below the right costal margin, but it seemed to be depressed from above by a flattened diaphragm.

Shortly after admission arterial blood gas studies revealed: PCO_2 24 mm Hg, pH 7.71, PO_2 18 mm Hg, O_2 saturation 47%. The patient was started on continuous oxygen administered by heated nebulizer with the Venturi set to 40% and flowmeter at 84L/min:he was given intermittent positive pressure driven by compressed air with a 3 liter/minute oxygen bleed in (pressure 25 cm H_2O, flow setting 20, and sensitivity setting 10) every 2 hours. A sputum smear revealed numerous gram positive, encapsulated diplococci, and hence the patient was started on penicillin. Sputum cultures subsequently grew out Diplococcus pneumoniae.

On this regimen the patient's respiratory distress was markedly improved. IPPB was successful in promoting deep breaths and the coughing up of large amounts of greenish sputum. As hydration was carried out, physical findings of bilateral lower lobe consolidation

appeared. Likewise, bilateral lower lobe pulmonary infiltrates became evident by x-ray. The following day the patient had clear-cut evidence of carbon dioxide retention while adequately oxygenated. (See chart).

The patient's hospital course of 23 days was somewhat hectic, but in essence he showed continual, gradual improvement. Carbon dioxide narcosis did not ensue, and an artificial airway or respiratory stimulants were never necessary. By time of discharge the patient's blood gases were close to normal. On subsequent follow up visits in the out patient department, the patient reports that his chronic sputum production is markedly decreased, and his exercise tolerance has improved considerably over that prior to hospitalization. In addition, he has gained 14 pounds since his initial hospital weight.

SELECTED BLOOD GAS VALUES

Date	[redacted]	[redacted]	[redacted]	[redacted]
Inspired gas	Room Air	O_2 mist	?	Room Air
pH	7.71	7.25	7.43	7.46
pCO_2 (mm Hg)	24	76	50	44
CO_2 content (meq/L)	31	34	32	32
pO_2 (mm Hg)	18	124	62	75
O_2 sat. (%)	47	97	91	95
Hgb (gm %)	17.2	13.3		12.6

This case is presented to illustrate three points. (1) Under the marked respiratory drive associated with hypoxia and pneumonia the patient was capable of marked hyperventilation with a lowering of his PCO_2 to 24 mm Hg. After the onset of oxygenation, he reverted to hypoventilation, which was almost surely his chronic steady state before the onset of the pneumonia. It is frequently stated that a patient with a PCO_2 below 50 mm Hg at time of admission will not hypoventilate when oxygenated; this case clearly contradicts that clinical maxim. (2) Despite the hypoventilation during oxygen therapy, the patient did not progress to the point of CO_2 narcosis, and he was adequately managed without respiratory stimulants or an artificial airway. (3) At time of discharge the patient's blood gases were probably better than they had been in several months, and the patient's symptoms were markedly improved over those present for a several month period of time. It is not unusual to observe patients who are dramatically improved after a period of hospitalization and adequate bronchopulmonary hygiene, even though the hospitalization is precipitated by acute bronchitis or pneumonia.

Case II: [redacted]

This was the sixth DCHD admission for this 61 y/o WM alcoholic who was brought comatose and apneic by an ambulance to the [redacted] Emergency room. The patient was a known chronic alcoholic with long standing chronic obstructive lung disease. He was last admitted in [redacted], 1967, with pneumococcal pneumonia. On that occasion he had presented to the emergency room awake but became confused and comatose after being given 100% oxygen. On that previous admission the PCO_2 had gone to greater than 95 mm Hg with his emergency room management, but after a hospitalization of 29 days he was discharged with a PCO_2 46 mm Hg, pH 7.47, PO_2 44 mm Hg and oxygen saturation 82%. The only history available for the interim period of time was that the patient's dog had died 12 days prior to admission, and the patient became so depressed that he went to bed and had barely eaten during the 12 days pta. At some time he had developed fever with labored respiration.

Physical examination at time of admission revealed vital signs as follows: T 104, P 110, R 13 (controlled ventilation on IPPB) BP 110/70. The patient was an obtunded, elderly [redacted] male. His chest revealed bilateral rhonchi, dullness to percussion and coarse moist rales in the left lower lung field posteriorly, decreased breath sounds and generalized hyperressonance. The heart could not be heard or percussed. The liver was 2 cms below the right costal margin, but it was felt to be depressed by flattened diaphragms. The patient was felt to be dehydrated.

An oral endotracheal intubation was carried out in the emergency room, and the patient was placed on automatic IPPB as indicated in the accompanying chart. The patient was hyperventilated while automatic ventilation was being carried out, but despite this the patient assumed his own respiratory control and continued to hyperventilate for a period of hours. Subsequently IPPB pressures were appropriately reduced, and hyperventilation was corrected. The oral tracheal tube was in place approximately 96 hours, and was discontinued without difficulty. The patient's pneumonia was treated with Keflin to which his fever rapidly responded. Convalescence was unremarkable, and following 16 days of hospitalization the patient returned to his home at Soul's Harbor. He has not kept subsequent clinic appointments for follow up care.

SELECTED BLOOD GAS VALUES

Date	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
Condition	Auto IPPB Driven on O2 with air mix	Auto IPPB compressed air	Auto IPPB compressed air	Pt cycled IPPB air	Pt cycled IPPB air	No IPPB Room air
pH	7.42	7.58	7.61	7.62	7.42	7.45
pCO_2 (mm Hg)	42	29	26	31	44	40
pO_2 (mm Hg)	69	110	80	54	69	50
Sat O_2 (%)	93	98	97	93	93	86
IPPB Setting						
Pressure	25	32	25	21	20	
Flow	25	20	20	20	20	
Sens.	4	4	4	4	4	

This case illustrates several additional points. (1) On the patient's former hospital admission, CO_2 narcosis was precipitated by the injudicious use of high concentrations of oxygen. (2) Blood gas measurements on the second admission did not reveal hypercapnia, but no measurements were made prior to automatic controlled ventilation. The most likely cause of the initial presentation of the patient with apnea was the administration of oxygen by the ambulance crew on the way to the hospital. Nevertheless, by most definitions this patient couldn't be classified as respiratory failure. (3) The orotracheal tube was easily tolerated for 4 days without sequela. (4) Despite severe lung disease, the patient was easily hyperventilated by IPPB; fortunately, no serious effects were noted from the period of respiratory alkalosis. (5) Soul's Harbor should be equipped with IPPB.

Case 3: [redacted]

This 61 y/o woman, an employee of [redacted], began smoking at least a pack of cigarettes a day in her twenties. She began to have a chronic productive cough with small amounts of early morning, white sputum in the 1950's. Around 1955 she began to have frequent episodes of "colds" during winter months with production of large amounts of yellow sputum; she was seen on numerous occasions in the emergency room because of these episodes of acute bronchitis. An electrocardiogram done in 1955 showed right axis deviation. The first available chest x-ray in 1958 showed pulmonary emphysema with lucent lung fields and a flattened diaphragm. During this period she had the onset of dyspnea on exertion, although the degree is hard to quantitate. By 1962, however, dyspnea was present after walking one block on the level; an EKG showed P pulmonale, right axis deviation and right ventricular overload, and vitalometry revealed a predicted vital capacity of 2.75 L, FVC 1.60, $FEV_{1.0}$ 0.90 and $FEV_{0.5}$ 0.60.

The patient was first admitted due to chest complaints in [redacted], 1964, at which time she had pneumococcal pneumonia in the lingula. On that admission her pCO_2's ranged between 48 and 54 mm Hg, and her PO_2 on room air breathing was 45 mm Hg (O_2 saturation 83%). The patient was treated with continuous heated mist, IPPB with bronchodilators, and penicillin; she responded promptly without complications, and she was discharged after 14 days. Vitalometry at time of discharge revealed an FVC 1.70 and $FEV_{0.5}$ 0.50.

The patient did not follow any consistent treatment regimen during the ensuing months, and her symptoms remained essentially those noted above. She was next admitted in [redacted], 1966, at which time she again had pneumococcal pneumonia; on this occasion it was in the left lower lobe. On admission her blood gases were PCO_2 48 mm Hg, PO_2 59 mm Hg, pH 7.42 and oxygen saturation 89%. Her vitalometry on admission was FVC 0.80, $FEV_{0.5}$ 0.50 and $FEV_{1.0}$ 0.70. During that hospitalization she was treated in much the same way as during the previous hospitalization, and there were no complications nor overt carbon dioxide narcosis. Her PCO_2 got as high as 66 mm Hg, but by discharge it was 54 mm Hg. Discharge vitalometry revealed FVC 1.25, $FEV_{0.5}$ 0.55, and FEV 0.80.

The patient's next admission in [redacted], 1967, was precipitated by several days of severe gastroenteritis which caused marked systemic dehydration, thick sputum, and increasing respiratory difficulty. During the first 36 hours of hospitalization the patient was re-hydrated with systemic fluids, and she was given heated mist and IPPB with bronchodilators. She improved markedly. However, on her second hospital evening she was given 500 mgms of chloral hydrate for sedation.

The next morning she was disoriented and dyspneic, and blood gases at that time revealed PCO_2 74 mm Hg, pH 7.31, PO_2 36 mm Hg and oxygen saturation 50%. She was started on continuous intravenous Ethamivan which improved her mental status, although she continued drowsy and needed encouragement to take deep breaths by IPPB and cough effectively. Although she improved on this regimen, for reasons that are not entirely apparent her progress was not considered satisfactory, and naso-tracheal intubation was carried out. She was placed on continuous IPPB with automatic cycling; the machine was driven on compressed air with an oxygen bleed-in resulting in a PCO_2 57 mm Hg, pH 7.46, PO_2 69 mm Hg and oxygen saturation 93%. By the next day the patient was more alert and she was allowed to trip the machine herself. This resulted in a PCO_2 72 mm Hg, pH 7.34, PO_2 118 mm Hg, and O_2 saturation 97% during the first hour of patient triggered ventilation, but after several additional hours she had a PCO_2 of 55 mm Hg at a time that her PO_2 was 72 mm Hg. By the next day she was able to maintain roughly equivalent blood gases without the use of intermittent positive pressure, and she was extubated. The rest of her hospital course was uneventful. At time of discharge she had PCO_2 50 mm Hg, pH 7.46 and PO_2 55 mm Hg, FVC 1.40, $FEV_{0.5}$ 0.60 and $FEV_{1.0}$ 0.80.

The patient's next admission was approximately a year later on [redacted] 1967. This admission was precipitated by 6 days of nausea, vomiting, and malaise presumably due to an injection of influenza vaccine. On admission the patient's blood gases revealed a PCO_2 54 mm Hg, pH 7.35, PO_2 35 mm Hg and O_2 saturation 65%. She was started on continuous heated mist, deep breathing by IPPB with bronchodilator, postural drainage, encouraged coughing, and intravenous fluids. Not only did she not improve on this regimen, but the next day she seemed to be somewhat more lethargic, and her PCO_2 had risen to 67 mm Hg. She was started therefore, on a continuous infusion of Ethamivan which resulted in marked mental clearing, much better mobilization of secretions, and a fall in the PCO_2 to 58 mm Hg. The range of Ethamivan infusion was 6 to 8 mgms/minute and she became toxic on 10 to 12 mgm/min. After approximately 24 hours of IV Ethamivan, it was discontinued without deterioration of her blood gases. The rest of the hospital course was one of gradual improvement, and her blood gases at time of discharge were PCO_2 48 mm Hg, pH 7.44, PO_2 62 mm Hg and O_2 saturation 93%.

Between each of these hospital admissions for respiratory failure, and since the recent hospitalization, the patient has always returned to work, and she has been able to perform her duties as an admission clerk in a completely satisfactory manner.

This particular case illustrates at least the following points:

(1) The patient continues to work as an effective and productive employee approximately 4 years after the onset of chronic respiratory failure as evidenced by her blood gases. (2) The last two episodes of respiratory failure were precipitated by systemic dehydration without any evidence of respiratory infection. (3) The patient has been successfully managed both by nasotracheal intubation and by Ethamivan. (4) At least one of the episodes of respiratory failure was markedly worsened by the injudicious use of a sedative.

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3. Filley, Giles F.: Pulmonary insufficiency and respiratory failure. Lea and Febiger, Philadelphia, 1967.

These three books are excellent general reviews of the entire subject of respiratory failure. Each can be read in a few hours time. The symposium on respiratory failure held by the New York Academy of Sciences brought together a large number of experts in this field, and it is interesting to read their various approaches to the same problem. It is apparent that there is considerable disagreement, and the discussion following each of the sections brings this out clearly.

The book by Bendixen and his associates is well written, and the chapters on therapy cover many of the small but important problems that arise in this type of patient; it is obvious that the authors have an extensive personal experience in the care of patients with respiratory failure. In addition, the book has an extensive bibliography covering not only the authors' many studies but also the work of other investigators.

The book by Filley and the group at Colorado is particularly well written in the clinical physiology sections which are more easily understood than the same sections in Bendixen's book. However, the sections on therapy are not as well done as in the former book. Nevertheless, Filley's book supplements Bendixen's book in that it contains more about patients with chronic obstructive lung disease, whereas Bendixen deals largely with patients more likely seen in a surgical unit.

PROGNOSIS OF CHRONIC OBSTRUCTIVE LUNG DISEASE

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The death rate of patients with chronic obstructive pulmonary disease is well in excess of that expected for a standard U. S. population of the same age and sex. In the study by Mitchell, Webb and Filley the findings which appeared to have the greatest adverse influence upon prognosis were a history of right heart failure, secondary polycythemia, severe initial ventilatory impairment, arterial oxygen saturation less than 90% and evidence of right ventricular hypertrophy by the electrocardiogram. However, the progression of the disease is extremely variable, and many patients can lead a useful life for years. Mitchell's statistical projection in essentially untreated patients indicated that at least 25% of the patients were able to walk 500 feet without dyspnea 10 years after the first symptom of dyspnea. In a small group of 12 patients with chronic airway obstruction who had been treated for acute respiratory failure, Ebert found that the average length of survival at follow-upwas 5 years.

CLINICAL PICTURE OF RESPIRATORY FAILURE

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16. MacDonald, Frank M.: Respiratory acidosis. Arch. Intern. Med. 116:689, 1965.

17. Williams, M. Henry, Jr.: Ventilatory failure. Medicine 45: 317, 1966.

These are representative articles which include the clinical picture of patients with ventilatory failure and the pathophysiology of respiratory failure. Most such articles define ventilatory failure in terms of abnormal blood gas measurements. Although some authors feel that the definition should include patients with abnormally low arterial oxygen tensions, most investigators rely entirely on hypercapnia as the single defining characteristic of ventilatory failure. This seems reasonable, since hypoxia can occur in a variety of circumstances in which the respiratory apparatus has not failed in terms of adequate alveolar gas exchange. The arbitrarily determined limit of the arterial pCO2 indicating ventilatory failure is usually taken to be 50 mm Hg, although some (e. g. Kilburn) insist on the more rigorous criteria of 60 mm Hg.

Unfortunately many authors do not divide patients with acute respiratory failure and a rapidly mounting pCO_2 from those patients in a stable state of compensated carbon dioxide retention. This makes it difficult to compare the results of therapy in different series, since some groups of patients are clearly more ill than others. For example, 18 of Williams' 122 patients had chronic CO_2 retention for which they were admitted and treated, but they were no more hypercapnic on admission than they were before or after hospitalization. At first thought it would be attractive to use the severity of the accompanying acidosis as an index of the rapidity of rise in PCO_2, but many patients with acute deterioration and worsening CO_2 retention have only a mild decrease in the arterial pH.

In older literature (not included here) it is emphasized that acute bacterial chest infections are the most frequent precipitating causes of respiratory failure in an otherwise compensated patient. However, our experience parallels that of Williams in that many of our patients reveal little evidence of pyogenic infection. It may be that viral infections account for the increasing airway obstruction that preceeds frank ventilatory failure. However, it is likely that many of these admissions are due to ineffectual bronchial hygiene over a period of days or weeks leading to retained respiratory secretions.

The mental and neurological dysfunctions in patients with ventilatory failure include disorientation, confusion, incoherence, somnolence, obstreperousness, combativeness, stupor, coma, fine tremors, asterixis, myoclonic jerks, sustained myoclonus, and seizures. Such symptoms may

in part be due to hypoxia and in part due to hypercapnia. It is well known that the exact level of the blood gases does not correlate well with symptoms in a particular patient. It has been suggested that the pH of the cerebral spinal fluid is the most important determining factor in the genesis of symptoms due to carbon dioxide. Almost surely local vascular disease in the brain detrimental to cerebral perfusion also determines the severity of the disease before symptoms develop.

BLOOD GAS DERANGEMENTS AT THE OUTSET

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When a patient with respiratory failure presents to the emergency room it is unlikely that the arterial pCO_2 will be above 70 to 80 mm Hg, unless the patient has been previously breathing oxygen or has been given a sedative. The arterial pH will be 7.19 or higher unless there is a superimposed metabolic acidosis;usually the pH will not be this severely depressed (>7.30) due to renal compensation. The PO_2 will be 20 mm Hg or higher and the oxygen content of arterial blood at least 5.5 ml/100 ml; values below this level are probably incompatible with life. In Refsum's series of 129 patients, no case of coma occurred at PCO_2 values below 75 mm Hg, PO_2 values above 35 mm Hg or oxygen content values above 11.0 ml/100 ml. The highest PCO_2 associated with consciousness was 89 mm Hg, the lowest PO_2 20 mm Hg, and the lowest O_2 content 5.5 ml/100 ml.

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Virtually all investigators agree that the first therapeutic aim in the patient with acute respiratory failure is adequate oxygenation. Unfortunately, no one can agree on what constitutes adequate oxygenation. In his earlier articles Campbell suggested that as long as the PO_2 of arterial blood was over 20 mm Hg the patient would likely be maintained alive, and as long as the PO_2 was over 30 mm Hg (corresponding to an oxygen saturation of approximately 50%) the patient was safe from death from hypoxia. More recently, Campbell has increased the lower levels of acceptable hypoxia to a PO_2 in excess of 40 mm Hg. However, others have chosen a PO_2 of 50 mm Hg as the lowest tolerable level, and still others feel that the PO_2 should be brought to near normal values (PO_2 of 80 mm Hg or higher). There are two basic problems in determining the goals of oxygen therapy. The first problem relates to the fact that there is very little good experimental or clinical evidence to determine the critical levels of tissue oxygenation that cause damage to cells. Dr. David Nicholson will conduct a Grand Rounds on this subject in the near future, and hence I shall not deal further with it here.

The second problem relating to the goals of oxygen therapy is the inability of the physician to measure or estimate all of the critical parameters of tissue oxygenation in the patient in respiratory failure. To assume that good tissue oxygenation is assured at a particular level of PO_2, oxygen saturation or both is an extreme over simplification. These measurements are not sufficient to allow one to calculate the oxygen content of arterial blood; the hemoglobin concentration is also a critical factor.

PO_2 x 0.003 = mls. O_2 dissolved/100 ml of blood.
1.34 x grams hemoglobin x % O_2 saturation = ml O_2 carried by hemoglobin/100 ml blood.

Thus, in a patient with a normal hemoglobin of 15 grams %, a pH of 7.40, and a PO_2 of 50 mm Hg the arterial oxygen saturation will be 84%. The amount of oxygen dissolved in plasma will be 0.15 ml/100 ml

of blood, and the amount of oxygen carried by the hemoglobin will be 16.88 mls/100 ml of blood; the O_2 content of arterial blood will be 17.03 ml/100 ml of blood. However, if the patient has a PO_2 of 50, a pH of 740 and an arterial saturation of 84%, but the hemoglobin concentration is only 7 grams %, the total oxygen content of the arterial blood would be only 8.03 ml/100 ml of blood.

Moreover, tissue oxygenation cannot be assessed by the arterial oxygen content alone. The total amount of oxygen leaving the cardiopulmonary apparatus is determined not only by the oxygen content of arterial blood, but also by the cardiac output. If the patient is not capable of increasing his cardiac output sufficiently for metabolic needs, tissue hypoxia may result in the presence of a normal arterial oxygen content. Finally, the local regulation of blood flow to vital tissues is also critical. Patients with coronary or cerebral arteriosclerosis may suffer catastrophic local tissue damage despite normal perfusion of the rest of the body with well oxygenated blood.

The decision of the extent of oxygenation necessary is not an academic one, because the higher the concentration of inspired oxygen (and hence the higher the arterial oxygen tension) the more likely a patient with respiratory failure is to decrease his effective ventilation and hypoventilate more. Because of worsening hypoventilation with oxygen therapy, it has been recommended that oxygen be given intermittently as a method of preventing progressive carbon dioxide retention. It is reasoned that withdrawal of oxygen restores the anoxic stimulus to breathing and causes the retained carbon dioxide to be blown off during the oxygen free intervals. This is clearly an extremely dangerous mode of oxygen administration, and its results may be more disasterous than the use of no oxygen at all. Farhi and Rahn determined that the stores of oxygen and carbon dioxide in the body are very different. Campbell first pointed out that if the inspired air composition is changed the oxygen concentration in the body changes to a new level in one to two minutes, but that of carbon dioxide takes 10 to 20 minutes. The persistence of a high PCO_2 in the blood coming to the lungs dilutes the alveolar air and reduces alveolar oxygen tension, and thus arterial hypoxemia may temporarily be even more severe following oxygen administration than if oxygen had never been given at all. The soundness of this reasoning has been demonstrated by Massaro, Katz and Luchinger.

It is noteworthy that in all of the arguments concerning the mode of administration of oxygen not a single author notes the importance of adequate humidification of the inspired gas. Virtually all of the methods of oxygen administration reported in these references utilize an oxygen hunidifier which causes the moisture content of the inspired

oxygen to be only 20% of the moisture necessary to saturate the oxygen once it has been heated to body temperature. Thus, all of these methods of administration of oxygen are drying to the respiratory mucosa.

ASSISTED OR CONTROLLED VENTILATION

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49. Sukumalchantra, Yosvi, Sung S. Park and M. Henry Williams, Jr.: The effect of intermittent positive pressure breathing (IPPB) in acute ventilatory failure. Am. Rev. Resp. Dis. 92:885, 1965.

50. Block, A. Jay and Wilmot C. Ball, Jr.: Acute respiratory failure, observations on the use of the Moerch piston respirator. Ann. Int. Med. 65:957, 1966.

51. Kilburn, Kay H.: Shock, seizures, and coma with alkalosis during mechanical ventilation. Ann. Int. Med. 65:977, 1966.

If adequate oxygenation of the patient depresses respiration causing a significant rise in PCO_2, oxygen must be continued while some

other mode of therapy is instituted to prevent further hypoventilation. Most commonly this is done by assisting the patient's ventilation with an IPPB device. The attachment of the patient to an IPPB machine, however, does not insure adequate ventilation. If the patient's respiratory drive is diminished by the oxygen received from the machine, each tidal volume may be adequate, but the respiratory rate may be so slowed that continued CO_2 retention occurs. In such a case completely controlled instead of assisted ventilation may be necessary. Conversely, if the patient continues with a rapid, but ineffectual, respiratory rate, it may be difficult to mate the machine to the patient and hence adequately assist ventilation. In such a circumstance, it may be necessary to depress the patient's respiratory drive with sedatives so that effective assisted or controlled ventilation can be carried out.

Although some authors such as Cullen, Brum and Reidt and Sukumalchantra, Park, and Williams report difficulty in reducing patients PCO_2 with IPPB, our major problem is reducing the PCO_2 too rapidly. A rapid change from hypercapnia and respiratory acidosis to hypocapnia with respiratory alkalosis causes a syndrome characterized by tachypnia, hypotension, anxiety, confusion, tremors, asterixis, myoclonus, seizures, coma and fever. Thus, patients should be ventilated in such a way that the arterial pH never exceeds a normal value.

TRACHEOSTOMY

52. Nelson, Thomas G. and Warner F. Bowers: Tracheotomy-indications, advantages, techniques, complications, and results, analysis of 310 recent operations. J. A. M. A. 164:1530, 1957.

53. Ferris, B. G., Jr., and David S. Pollard: Effect of tracheostomy tubes on the resistance to breathing and pulmonary resistance in patients with polymyelitis. New Eng. J. Med. 263:1048, 1960.

54. Atkins, Joseph T.: Current utilization of tracheotomy as a therapeutic measure, a review of the literature and an analysis of 526 cases. Laryngoscope 70:1672, 1960.

55. Ted, John M.: Tracheostomy in the management of respiratory problems. New Eng. J. Med. 264:587, 1961.

56. Meade, James W.: Tracheotomy-its complications and their management. New Eng. J. Med. 265:519, 1961.

57. Froeb, Herman F. and Byong M. Kim: Tracheostomy and respiratory dead space in emphysema. J. Appl. Physiol. 19:92, 1964.

If continuous assisted or controlled respiration is necessary, an artificial airway must be provided, since the use of face mask applied

IPPB is unsatisfactory in these patients. Tracheostomy is the standard means of obtaining an artificial airway. However, this operative procedure should not be undertaken lightly. In the series collected here, of 1,949 tracheostomies there were 35 deaths (1.1%) due to the procedure itself. Rational estimates of the complication rate range between 13 and 35%. Such complications include wound bleeding early or late, pneumothorax, mediastonitis, subcutaneous emphysema, pneumonia, and a variety of mechanical problems with the tracheostomy tube. The problem of occlusion of the tracheostomy tube by mucous plugging, reported in most of these series, should not exist if adequate humidification is carried out. On the other hand, the complication that we frequently find troublesome in patients with chronic obstructive lung disease is not mentioned in any of these series. Specifically, having salvaged the patient from respiratory failure, the process of extubation may greatly prolong the patient's hospital stay or may reprecipitate respiratory failure. In elderly patients when the tracheostomy tube is removed the tracheastoma closes slowly; during the closure of the tracheal opening the patient's cough is ineffective, and he may not be given effective IPPB. The weaning process of gradually reducing the tracheal tube size to allow the trachea to close down around the progressively smaller tubes is usually satisfactory, but the patient's morbidity is greatly increased.

As indicated by Froeb and Kim, the performance of a tracheostomy to decrease the dead space in patients with emphysema is not rational. If the patient is to be carried on assisted ventilation, the reduction of dead space of 50 milliliters is insignificant. While breathing with his own respiratory muscles, the patient with obstructive airway disease and a tracheostomy does not increase his alveolar ventilation proportionately to the reduction in dead space ventilation. Rather, he simply decreases his total ventilation by the amount of reduction in dead space, and hence effective ventilation is not improved.

58. Oppenheimer, Peter and Francis B. Quinn, Jr.: Quick tracheotomy, incision at an easily identifiable, relatively safe site. Calif. Med. 104:51, 1966.

A cricothyrotomy is a rapid, relatively safe means of obtaining an artificial airway in an emergency. It is probably safer and faster than passing an endotracheal tube when the intubation is difficult or the physician is inexperienced.

ENDOTRACHEAL INTUBATION

59. Heller, Morris L. and T. Richard Watson, Jr.: Polarographic study of arterial oxygenation during apnea in man. New. Eng. J. Med. 264:326, 1961.

60. Lu, Abraham T., V. Tamura and Dick H. Koobs: The pathology of laryngotracheal complications, lesions of the larynx and trachea after intubation anesthesia. Arch. Otolaryngol. 74:323, 1961.

61. McDonald, I. H. and Jay G. Stocks: Prolonged nasotracheal intubation, a review of its development in a paediatrics hospital. Brit. J. Anaesth. 37:161, 1965.

62. Allen, T. H. and I. M. Steven: Prolonged endotracheal intubation in infants and children. Brit. J. Anaesth. 37: 566, 1965.

63. Gold, Martin I. and John M. Atwood: Respiratory obstruction. Anesth. 26:577, 1965.

64. Birkhan, H. J. and M. Heifetz: "Uninflatable" inflatable cuffs. Anesth. 26:578, 1965.

65. Smotrilla, Margaret M., Eugene L. Nagel and Frank Moya: Failure of inflatable cuff resulting in foreign bodies in the trachea. Anesth. 27:512, 1966.

66. Kohli, M. S. and R. S. Manku: Reinforced endotracheal tube-diversion of air from cuff balloon producing obstruction. Anesth. 27:513, 1966.

67. Striker, Theodore W., Sylvan Stoll and John J. Downes: Prolonged nasotracheal intubation in infants and children. Arch. Otolaryng. 85:106, 1967.

68. Weg, John G.: Prolonged endotracheal intubation in respiratory failure. Arch. Intern. Med. 120:679, 1967.

It has become apparent in recent years that endotracheal tubes may be left down for longer periods than the 48 hour maximum that was considered possible in the recent past. Endotracheal tubes, when compared to tracheostomies, have the advantage of decreased morbidity and increased ease of extubation. Although at other institutions they are used for much longer periods of time, at Parkland we are using them for artificial airways for 7 to 10 days. If it is apparent that the patient will need an artificial airway for a longer period of time, a tracheostomy is done as soon as the need for a prolonged artificial airway is recognized. As indicated in the various references, the tubes are not without danger, and are somewhat more tricky to use than a tracheostomy. Two specific complications deserve comment. First, it is mandatory that the tube be stabilized so that it cannot slip up and

down the airway endangering the vocal cords. Secondthe tube must not be passed so far that the right mainstem bronchus is intubated, since atelectasis of the right upper lobe and the entire left lung results.

Heller and Watson have shown that the PO_2 of a normal person may fall from a normal value while breathing room air of 100 mm Hg to 50 mm Hg after only one minute of apnea. The fall in oxygen tension and saturation may be even more precipitious in patients with pulmonary disease. On the other hand, if a patient with normal lungs is ventilated with 100% oxygen for 3 minutes causing the arterial PO_2 to approach 400 mm Hg, after 4 minutes of apnea the PO_2 is still 150 mm Hg. This study indicates the critical importance of oxygenating a patient well before attempting endotracheal intubation. Most patients, no matter how severely ill, can be adequately oxygenated by means of an oral airway to hold the tongue out of the way plus assisted ventilation. Thus, prolonged periods (2 minutes) of unsuccessful intubation need not be tolerated; when the intubation is not proceeding smoothly the procedure should be stopped and the patient re-oxygenated before a second attempt is made.

Tracheal intubation can be carried out either through the nose or the mouth. Nasotracheal intubation has the advantages of being better tolerated by the patient and having better support for securing the tube so that it will not slip. It has the disadvantages of requiring a smaller tube and making tracheal suction more difficult. Orotracheal intubation has the advantages of being easier to perform and allowing better tracheal suctioning. It has the disadvantages of greater discomfort to the patient and greater liklihood of slipping in the airway. It should be noted that these tubes are remarkably well tolerated in a fully awake patient. Indeed, we have had several patients able to eat by mouth while being ventilated through a nasotracheal tube.

69. Deming, Margery Z. and Steffen R. Oech: Steroid and antihistaminic therapy for post intubation subglottic edema in infants and children. Anesth. 22:933, 1961.

Most patients will be hoarse for several days following extubation. A few patients will develop an upper airway stridor indicating subglottic edema following extubation; this is potentially serious, since it can progress to upper airways obstruction. At the first sign of stridor, if the patient is immediately given an injection of some glucocorticoid, the edema usually regresses very promptly.

RESPIRATORY STIMULANTS

70. Westlake, E. K. and E. J. M. Campbell: Effects of aminophylline, Nikethamide, and sodium salicylate in respiratory failure. Brit. Med. J. 1:274, 1959.

71. Tyler, John M.: The effects of progesterone on the respiration of patients with emphysema and hypercapnia. J. Clin. Invest. 39:34, 1960.

72. Silipo, Samuel, Clem Hagedorn, Ira N. Rosenstein and George L. Baum: Experiences with Ethamivan, a new respiratory stimulant and analeptic agent. J. A. M. A. 177:378, 1961.

73. Miller, William F., Richard Archer, Harold F. Taylor and William F. Ossenfort: Severe respiratory depression, role of a respiratory stimulant, Ethamivan, in the treatment. J. A. M.A. 180;905, 1962.

74. Rodman, Theodore, Joseph F. Fennelly, Albert J. Kraft and Henry P. Close: The effect of Ethamivan on alveolar ventilation in patients with chronic lung disease. New Eng. J. Med. 267:1279, 1962.

75. Said, Sami and C. M. Vanerjee: Effects of a newer respiratory stimulant (vanillic diethylamide) in respiratory acidosis due to obstructive pulmonary emphysema or obesity. Am. J. Med. 33:845, 1962.

76. Galdston, Morton and Martin D. Myles: The use of aminophylline in respiratory depression and carbon dioxide retention induced by oxygen inhalation in patients with pulmonary emphysema. Am. J. Med. 33:852, 1962.

77. Canter, Hall G.: Comparative study of 3 respiratory stimulants in chronic obstructive emphysema. Am. Rev. Resp. Dis. 87:830, 1963.

78. Canter, Hall G. and Peter C. Luchsinger: The treatment of respiratory failure without mechanical assistance. Am. J. Med. Sc. 248:206, 1964.

79. Fraimow, William, Paul Diamond and Richard T. Cathcart: Ventilatory response of patients with pulmonary emphysema to doxapram hydrochloride. Am. J. Med. Sc. 249:150, 1965.

Respiratory stimulants may be used in the management of some patients with respiratory depression to obviate the necessity of an artificial airway and continuous assisted ventilation, and in other patients respiratory stimulants may serve a useful adjunct to patients on continuous assisted ventilation. Although a variety of stimulants have been used in the past, Ethamivan and Doxapram are probably the most useful. When given by continuous infusion these drugs may stimulate the patient to a higher level of consciousness and hence make practical patient management more easy. In addition, they may keep a patient breathing despite the administration of oxygen in sufficient amounts to prevent hypoxia.

The poor response to Ethamivan found by Rodman, et al, is likely due to the very small dose of drug that he administered (50 mgms single

injection). As reported by Cherniack, if the drugs are administered in too large a dose, the generalized muscular activity that ensues causes such a large increase in CO_2 production that the increase in ventilation is offset, and the PCO_2 may not fall or may actually rise. However, this has not been a very great problem in the local experience. It should be remembered that the drug plays only a part in total patient care, and the aim of its use is not necessarily to cause the patient to hyperventilate and blow off CO_2. Rather it is used to prevent further hypoventilation while administering oxygen, to keep the respiratory center active enough so that the patient will trigger the IPPB machine, or to promote an increased level of consciousness to make patient management more easy.

The continuous intravenous infusion of either drug must be tritrated to maintain a respiratory response without generalized side effects such as itching, sneezing, or generalized muscular irritability. Miller, et al, found that the usual continuous intravenous dose of Ethamivan was 0.05 to 0.15 mgms/kilo/min. Fraimow, et al, have used Doxapram at a rate of 2.5 mgms/min.

PERIODIC HYPERINFLATION

80. Mead, Jere and Clarence Collier: Relation of volume history of lungs to respiratory mechanics in anesthetized dogs. J. Appl. Physiol. 14:669, 1959.

81. Ferris, Benjamin G., Jr. and David S. Pollard: Effect of deep and quiet breathing on pulmonary compliance in man. J. Clin. Invest. 39:143, 1960.

82. Bendixen, H. H., J. Hedley-Whyte, B. Chir and M.B. Laver: Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation, a concept of atalectasis. New Eng. J. Med. 269:991, 1963.

83. Farhi, Leon E.: Atmospheric nitrogen and its role in modern medicine. J. A. M. A. 188:984, 1964.

It has been clearly demonstrated that the lungs of normal men must be periodically hyperinflated if progressive, patchy atelectasis is to be avoided; this process is even more imperative in patients with lung disease. The effects of lack of inflation include a progressive stiffening of the lung (decreased compliance) due to a progressive loss of lung volume, and progressive hypoxia due to right to left shunting through the areas of atelectasis. If the patient with respiratory failure is awake, this can be accomplished with periodic IPPB administration at high enough inflation pressures to cause deep breathing. If the patient is apneic, and hence on controlled ventilation, the pressures of the machine should periodically be

increased to cause deep breathing. In either case, the only assurance that the IPPB is causing deep breathing is to measure the tidal volumes during the IPPB treatment.

STEROIDS

84. Cullen, James H. and William U. Reidt: A study of the respiratory effects of prednisone in diffuse airway obstruction. Am. Rev. Resp. Dis. 82:508, 1960.

85. Beerel, Frederick, Hershel Jick and John M. Tyler: A controlled study of the effect of prednisone on airflow obstruction in severe pulmonary emphysema. New Eng. J. Med. 268:226, 1963.

86. Morgan, W. K. C. and E. Rusche: A controlled trial of the effect of steroids in obstructive airway disease. Ann. Int. Med. 61:248, 1964.

The beneficial effect of steroids in patients with asthma is well known. In the very small number of patients treated in controlled series, such beneficial effects do not accrue to patients with clear cut chronic bronchitis or emphysema unless there is some asthmatic component to their disease. This can best be determined by a history of the onset of their disease with periods of intermittent airways obstruction with wheezing.

COMMON PITFALLS

87. Hunter, Charles C.,Jr.: Errors in management of patients dying of chronic obstructive lung disease. J. A. M. A. 199:488, 1967.

The most important errors in management of patients with severe degrees of respiratory failure frequently relate to the faulty assessment of the severity of the disease. Errors in management include the use of sedatives, the high incidence of digitalis intoxication and faulty oxygen therapy.