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CARBON DIOXIDE NARCOSIS AND THE MANAGEMENT OF ADVANCED PULMONARY INSUFFICIENCY*

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THE DEVELOPMENT of drowsiness, confusion and coma in emphysematous patients receiving oxygen therapy has been recently reported.[1-4] The factor or factors responsible for this impairment of consciousness are not known, but there is considerable circumstantial evidence implicating the level of carbon dioxide in the blood. Comroe[4] observed the development of mental symptoms in nine of 66 patients with chronic anoxæmia given high concentrations of oxygen to breathe. All of these nine patients had emphysema; all had an arterial O_2 saturation of 90% or less, and all had a P_{CO_2} of more than 50 mm. of mercury. A considerable decrease in resting respiratory minute volume occurred in each case while on oxygen, as the P_{CO_2} rose and the arterial pH fell.

It appears to be generally accepted that high concentrations of oxygen will cause, in anoxæmic patients, a moderate to marked decrease in ventilation. This decrease in ventilation will in no way impair the saturation of hæmoglobin with oxygen, since the alveolar partial pressure of oxygen is considerably raised by the inhalation of this gas. However, in patients with pulmonary insufficiency, not only for oxygen intake but also for CO_2 elimination, the sudden restriction of alveolar ventilation results in further retention of CO_2 in the blood.

How ventilation is impaired by inhalation of high concentrations of oxygen in anoxæmic patients is not known. The theory has been advanced that the anoxæmia serves as a stimulus to the carotid body, which in turn reflexly controls ventilation through the respiratory centre in the medulla and pons. The respiratory centre is itself depressed by the high level of carbon dioxide in the blood. Carbon dioxide[5, 6] has been shown to be a depressant in high concentration, although a stimulant to respiration in low concentration. The administration of oxygen removes the stimulus to the carotid body, and a sudden decrease in ventilation results.

Whatever the mechanism for the decrease in ventilation with oxygen therapy in these patients, the result is a rise in CO_2 content and the development of mental symptoms, including the comatose state. It would appear from the studies reported by Comroe *et al.*[4] that actual hypoventilation need not develop in those patients who already show a pulmonary insufficiency with regard to the elimination of CO_2. Some of the patients reported by these workers were hyperventilating when first given oxygen and the sudden relative decrease in ventilation with consequent CO_2 retention was presumably enough to precipitate coma. It seems reasonable to assume that a similar state of mental impairment would appear, even in the presence of normal lung parenchyma, if an absolute hypoventilation developed from sudden severe respiratory centre depression. These patients would receive oxygen which would correct the consequent anoxia to a greater or lesser extent, but which would in fact tend to further impair the excretion of CO_2 by the lungs. Such cases have been reported following encephalitis lethargica,[7] poliomyelitis and narcotic poisoning,[8] and head injuries.[9] The sudden inhalation of pure oxygen with the respiratory centre depressed by anoxia has also been reported as a cause of coma in aviators.[10, 11]

The actual mechanism by which a sudden rise in CO_2 content is able to produce coma is, as yet, not clear. Carbon dioxide does cause cerebral vasodilation with increased blood flow,[12] and an increase in cerebrospinal fluid pressure. It would appear reasonable to assume that these changes are in some way related to the mental symptoms.

Over the past two years we have been particularly interested in emphysema and chronic cor pulmonale. A combined clinical and physiological study has been directed toward the determination of the degree of pulmonary insufficiency in patients with these conditions, and the response to the various therapeutic agents used in an attempt to correct the anoxia and cardiac failure. In the course of this study several patients were seen in confused or comatose states apparently brought on by sedation and/or oxygen therapy. The following case reports are cited as examples of this phenomenon occurring in advanced pulmonary insufficiency.

CASE 1

H.H., a 69-year-old man who had had bronchitis for 17 years and symptoms of emphysema for nine years,

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developed severe respiratory distress and congestive heart failure after prostatectomy. He was digitalized and given a mixture of 95% O_2 and 5% CO_2 by intranasal catheter. Although cyanosis was quickly relieved, seven hours later he was found to be comatose. The intranasal catheter was removed and within 25 minutes he was fully conscious. Two further episodes occurred with subsequent attempts to administer this mixture, and on each occasion rapid recovery of consciousness resulted on stopping the oxygen-carbon dioxide mixture.

Over the next few days his breathing became extremely shallow and he was confused and disorientated. Despite the usual therapeutic measures, including the intermittent administration of oxygen at a low rate of flow, his condition deteriorated and as a last resort pneumoperitoneum was established in the hope of improving his ventilation. The pneumoperitoneum was maintained with frequent refills over a period of three weeks and the patient showed a remarkable clinical improvement. He was allowed up in a chair and appeared to be making a very satisfactory recovery when he developed a paralytic ileus. The pneumoperitoneum was abandoned and frequent injections of demerol were required for pain. He again became confused and disorientated and died after being 18 hours in a comatose state. Post-mortem examination showed marked bilateral emphysema and cor pulmonale, the right ventricle being 6 mm. in thickness.

The laboratory data and clinical course of this patient during the initial phase of his illness are summarized in Fig. 1. Arterial blood was drawn on each occasion after oxygen therapy had been stopped for at least half an hour. Anoxæmia (normal arterial O_2 saturation = 95 - 100%) and hypercapnia (normal CO_2 content = 49 - 52 vols. %), with a normal pH, were detected on the initial analysis several days after the episodes of coma. The patient at that time was in a state of compensated respiratory acidosis. Studies of arterial blood showed gradually increasing oxygen saturation and decreasing CO_2 content. With the elimination of CO_2 a mild metabolic alkalosis developed. Pneumoperitoneum, which has been previously reported as an emergency procedure in this condition, may have contributed to the temporary improvement in pulmonary function.[13]

CASE 2

A.L., a 51-year-old man, was admitted because of severe respiratory distress, cyanosis and muscular twitching. A history of constant wheezing and dyspnœa dating back 20 years had necessitated two previous admissions to hospital for treatment of advanced emphysema and cardiac failure due to cor pulmonale. Several months before this present admission pulmonary function tests had shown a marked reduction in maximum breathing capacity and an increase in residual volume. No significant objective change had occurred on initiation of a pneumoperitoneum, despite some subjective improvement. One month before admission his arterial oxygen saturation was 61.9% and his CO_2 content 64 vols. %. Three phlebotomies totalling 1,500 c.c. were performed for the purpose of relieving headaches presumed due to secondary polycythæmia, after which his oxygen saturation rose to 67%. Congestive failure developed despite digitalis administration and a low sodium diet and he was admitted to hospital. After admission he was given oxygen by B.L.B. mask. He became comatose within two hours, and the oxygen was discontinued at this point.

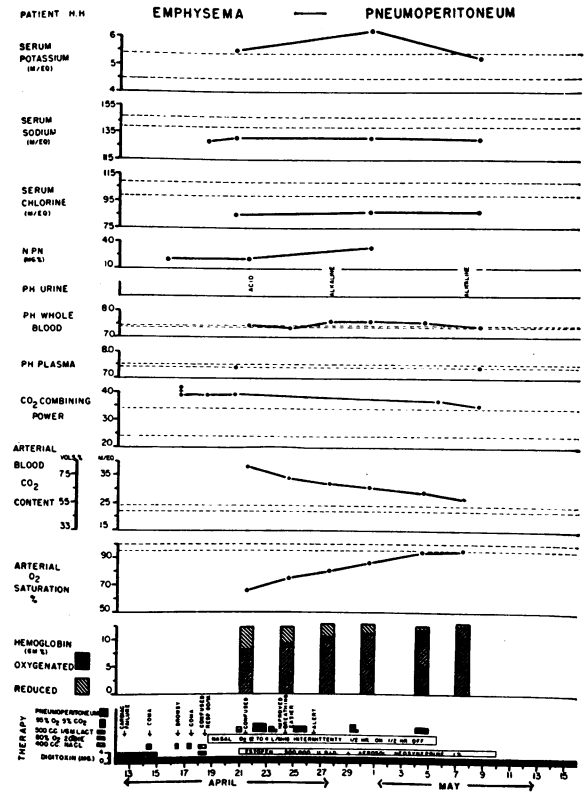

Fig. 1.—Laboratory and clinical data in Case 1.

An arterial blood sample showed an oxygen saturation of 37%, a CO_2 content of 63.7 vols. % and a CO_2 combining power of 68 vols. %. He became fully conscious one hour after oxygen therapy had been stopped. Because of the severe degree of anoxæmia, it was felt necessary to readminister oxygen, which was given by intranasal catheter at a rate of 1 l./min. He became confused, remaining so for three days, after which he sank into a coma and died. The autopsy revealed marked bilateral emphysema and hypertrophy of the right ventricle.

On his final admission this patient, with his long history of dyspnœa, cyanosis, polycythæmia and recurrent episodes of congestive failure, pre-

TABLE I.

ARTERIAL BLOOD GASES ON THREE CONSECUTIVE DAYS BEFORE DEATH (CASE 2)

Date of hospitalization	*First day after admission*	*Second day after admission*	*Third day after admission*
Arterial O_2 content, vols. %	6.8		5.05
Arterial O_2 capacity, vols. %	18.5		16.5
Arterial O_2 saturation, % (normal 95% - 100%)	37.0%		30.6%
CO_2 content, vols. % (normal 49 - 52 vols. %)	63.7	66.0	72.7
CO_2 combining power, vols. % (normal 55 - 75 vols. %)	68.0	76.4	78.6
Mental status	Coma	Confusion	Coma and death

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sented the problem of a severe anoxæmia, requiring oxygen; this therapy precipitated coma. The CO_2 content increased steadily over a three-day period. A compensation for this CO_2 excess is indicated by the rise in CO_2 combining power. (See Table I.)

CASE 3

N.A., a 53-year-old male, was admitted in congestive failure, with rapid laboured respirations and marked cyanosis. Eight years previously a diagnosis of emphysema with secondary polycythæmia and cor pulmonale had been made. He had been in heart failure at that time, which had cleared on bed rest and digitalis. Four years previously he had again been admitted in congestive failure. Oxygen therapy had not been given on either of these two previous admissions. On the present admission he was placed in an oxygen tent and was given mercurial diuretics. The following morning he appeared drowsy and during the day, while still in the oxygen tent, he became deeply comatose. The tent was removed and within 20 minutes he was conscious, although still markedly cyanosed. He was then given oxygen by intranasal catheter at a flow rate of 3 l./min. which was gradually increased over a period of hours until he again went into a coma at a 10 l./min. flow. The oxygen was again stopped and within one hour he had recovered consciousness. Following this second episode of coma, oxygen therapy was given continuously at a rate of 3 l./min. He remained conscious for several days, but became confused and incontinent of fæces and urine; he expired on the eighth day after his original episode of coma.

Arterial blood was drawn just before death with the patient breathing room air. The arterial oxygen saturation was found to be 77.9%, the CO_2 content 82.7 vols. %, and the CO_2 combining power 87.8 vols. %. Autopsy showed an advanced emphysema with dilatation and hypertrophy of the right ventricle. There was marked pulmonary arteriosclerosis and chronic passive congestion of the liver and spleen.

CASE 4

A.C., a 68-year-old man, was admitted for investigation of myxœdema. He had been admitted to hospital the previous year in cardiac failure, with emphysema, polycythæmia and cor pulmonale. During the interval he had remained moderately dyspnœic and cyanosed. During the first six days of hospitalization he underwent investigation for hypothyroidism. He appeared quite rational during this time. On the morning of the seventh day he fell out of bed and fractured the neck of his right femur. He was noted to be confused and very cyanotic, with extremely shallow respirations. The blood pressure was 108/70 mm. Hg as compared to an admission reading of 130/80. Fifty mg. of demerol was given for relief of pain, and within one hour he became comatose. Because of the extreme cyanosis, oxygen was given by B.L.B. mask at 8 l./min. flow. Blood pressure rose to 150/80 mm. Hg and cyanosis disappeared, but coma persisted. Oxygen was temporarily discontinued and an arterial puncture was done. The oxygen saturation was 55% and the CO_2 content 72.8 vols. %. He was put in a respirator with the hope of improving his ventilation and blowing off the carbon dioxide. Arterial oxygen saturation, as determined with an ear-piece oximeter (Waters-Conley)*, was 52% before and rose to 76% while in the respirator. It was found impossible to synchronize the respirator with the patient's own respiratory movements, and even with oxygen flowing at 10 l./min. by nasal catheter the blood did not become fully saturated. He was in the machine for 26 hours, and was removed from time to time to determine the character of his own respiratory movements. He was comatose throughout this entire period. Arterial blood taken two hours before death showed a CO_2 content of 75.2 vols. %. Terminally, frothy, blood-tinged fluid exuded from his nostrils and mouth.

At autopsy there was found to be a diffuse emphysema with dilatation and hypertrophy of the right ventricle, which was 10 mm. in thickness. The lungs were large, heavy, œdematous and hyperæmic. The thyroid gland was replaced by a Riedel's struma.

Coma in this patient was associated with marked hypoventilation which appeared after an accident. The small dose of demerol may have been a factor in precipitating coma, since the patient with advanced emphysema and myxœdema would presumably have been very sensitive to narcotics. A Drinker type respirator was used in an attempt to improve ventilation. Pulmonary œdema is an unusual finding in cor pulmonale but the giving of 4.5 litres of 5% glucose over the 24 hours before death may have contributed to this condition.

CASE 5

W.K., a 54-year-old man with emphysema and chronic cor pulmonale, was admitted in congestive heart failure. He gave a history of an episode of cardiac failure six months previously, successfully treated with a low salt diet, digitalis and rest. Arterial O_2 saturation as measured by oximeter three days before admission was 86%. Treatment consisted of oxygen administration by nasal catheter at a flow of 1 l./min., antibiotics and a low sodium diet. He showed gradual improvement over the first ten days in hospital. On the night of the tenth day, he was given chloral hydrate grains xx for insomnia. Early the next morning he was in a semi-comatose condition and very cyanotic. Oxygen was given by B.L.B. mask. Twenty minutes later he was in a deep coma. The mask was removed and oxygen was continued by intranasal catheter at 2 l./min. The arterial oxygen saturation with the oxygen running at this rate was 92% and the CO_2 content 79.9 vols. %. The patient remained in coma for approximately three hours. Twitching, particularly of the face and limbs, was then noted. Over the next hour he was completely irrational, shouting and attempting to get out of bed. He became rational, only to pass once more into a comatose state which lasted approximately five minutes. With return of consciousness twitching was again noted. During all this time oxygen administration was continuous by intranasal catheter at a flow of 1½ l./min., and the arterial O_2 saturation never rose above 94%. No further episodes of coma occurred and oxygen therapy was stopped 48 hours later, when his arterial oxygen saturation, while breathing room air, became stabilized at 90%.

This patient appeared to be progressing satisfactorily on intranasal oxygen, but went into a prolonged episode of coma following sedation and oxygen administration by B.L.B. mask. His CO_2 content rose to a very high level (see Table

*Waters-Conley Company, Rochester, Minn., U.S.A.

TABLE II.

Day of hospitalization	*11th day coma and confusion*	*12th day rational*	*16th day rational*	*25th day rational*
Serum K. (normal 4.3-5.4 mEq.)	5.75	4.75	5.45	
Serum Na. (normal 139-148 mEq.)	121.0	135.0	129.0	142.0
Serum Cl. (normal 99-107 mEq.)	68.9	73.0	85.2	94.2
CO_2 combining power (normal 55-75 vols. %)	85.0	80.5		
CO_2 content (normal 49-52 vols. %)	79.9	79.5	68.5	59.6
Art. O_2 saturation (normal 95-100%)	86.0	77.0	91.7	90.5

Changes in electrolytes, carbon dioxide and arteria blood oxygen coincident with clinical improvement (Case 5)

II) and this hypercapnia was accompanied by a marked hypochloræmia. Except for severe exertional dyspnœa he has remained well for the last 9 months since discharge from hospital. His arterial O_2 saturation continues in the range of 90%.

CASE 6

J.B., a 60-year-old man, was admitted in congestive heart failure. He gave a history of having had pneumonia and empyema at the age of 18. He had had increasing dyspnœa for the past three years. On examination it was noted that there was little movement of the right chest and air entry was poor. The left lung appeared normal both clinically and radiologically, but on the right side there was massive thickening and calcification of the pleura. Pulmonary function studies failed to reveal any evidence of emphysema. Anoxæmia and hypercapnia were present. The patient was polycythæmic and an E.C.G. revealed right ventricular hypertrophy. It was felt that this patient's pulmonary insufficiency resulted from poor ventilation, in the presence of good circulation, in the right lung. He had been admitted five months earlier in congestive failure, which readily responded to treatment. Anoxæmia and hypercapnia had been detected on several arterial blood examinations before admission. Arterial O_2 saturation, determined weeks before admission, had been 81.5% and CO_2 content 64.4 vols. %.

This patient was never actually found in coma but was completely disorientated while receiving oxygen by intranasal catheter at a low rate of flow. He recovered despite the continuation of O_2 therapy which was considered necessary in view of the degree of anoxæmia. He had advanced pulmonary insufficiency associated with unilateral lung disease. We have seen a similar clinical picture in patients with thoracoplasty. The mechanism by which anoxæmia and hypercapnia develop under these conditions requires further study.

CASE 7

H.V., a 40-year-old-man, was admitted with cardiac failure and cyanosis. He had a history of left-sided pneumonia and empyema at the age of three years. Surgical drainage with the removal of several ribs had resulted in a marked scoliosis. During his teens he had been active in competitive sports, but had developed a gradually increasing dyspnœa and repeated respiratory infections during the two years before admission. Increasing cough and sputum, with a low-grade fever, had been present for one week. For the first twelve hours after admission the patient was not given oxygen. He was fully conscious during this time. Because of severe cyanosis, oxygen was then started by intranasal catheter at a flow of one to two l./min. After a period of eight hours he became confused and, following a period of twelve hours of marked confusion, he became comatose. Since cyanosis persisted, oxygen flow was increased to 3 l./min. Arterial oxygen saturation at this rate of flow was 86% and the CO_2 content was 85 vols. %. When oxygen therapy was temporarily stopped, the patient developed extreme cyanosis and the arterial oxygen saturation by oximeter fell rapidly to 45%, at which time oxygen therapy was reinstituted. The ventilation was found to be 2.6 l./min., an extremely low figure and actually not enough to do more than wash out the dead space air. An unsuccessful attempt was made to improve the patient's ventilation by electrophrenic stimulation. Over the next two days he was in and out of coma. He died on the fourth day after admission.

Autopsy showed a fibrotic collapsed left lung, bronchopneumonia, and diffuse emphysema of the right lung. The right ventricle was dilated and hypertrophied.

With severe anoxia and hypoventilation this patient showed a marked retention of carbon dioxide. He remained in a comatose state with oxygen flow regulated to keep his arterial oxygen saturation around 90%, rather than at full saturation. This was done on the assumption that the chemoreceptors are stimulated by this degree of anoxæmia, whereas complete saturation of arterial blood is considered to suppress this reflex centre for ventilation.

DISCUSSION

The patient with severe pulmonary insufficiency presents a very complex therapeutic problem. Most commonly he suffers from emphysema and is admitted to hospital with a respiratory infection which has precipitated further anoxia in an already anoxic patient. This acute-on-chronic anoxia causes constriction of the pulmonary arterioles, a rise in pulmonary artery pressure and failure of the right heart. Motley *et al.*[14] have demonstrated that acute anoxia in man, produced by breathing 10% oxygen, causes a rise in the pulmonary artery pressure. Conversely, Harvey *et al.*[15] have shown a fall in pulmonary artery pressure, coincident with improvement in arterial oxygen saturation in emphysematous patients. It would therefore appear reasonable to give these patients oxygen, not only

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as a means of relieving congestive failure, but also to correct the severe degree of anoxia which in itself may cause death. However, as can be seen by the cases cited above, oxygen therapy may produce confusion or coma in such patients. The patient with anoxia and hypercapnia is very often found to have a decreased minute ventilation. The reason for this reduction in ventilation so often seen in advanced emphysema is not known, but it may possibly be due to the very high CO_2 content or the blood bicarbonate level. Carbon dioxide has been shown by Nielsen[5] to increase pulmonary ventilation proportionally with increasing concentrations of the inspired gas until a mixture of 9% CO_2 is used, following which higher concentrations lead to depression of respiration. Whatever the mechanism may be for the poor ventilation in this type of patient, there is no doubt that further depression of ventilation occurs on breathing oxygen, and under these circumstances coma may develop.

A sudden decrease in ventilation in the patient with marked pulmonary insufficiency may induce severe respiratory acidosis due to an uncompensated retention of carbon dioxide. If kidney function is adequate, compensation will be established by the excretion of chloride ions and the selective retention of basic ions. Sedatives and hypnotics will depress the respiratory centre and increase the tendency to hypoventilation. These drugs may in themselves precipitate coma, and in Cases 4 and 5 this may have been a factor.

Of primary importance in the prevention of cardiac failure, coma and respiratory acidosis is the close surveillance of the patient. Congestive failure will not develop in the absence of anoxæmia, and coma does not occur unless hypercapnia is also present. A patient with cyanosis is an obvious candidate for these complications, but even without cyanosis they can occur, since cyanosis is not recognised clinically until the arterial O_2 saturation has fallen to 85%,[16] and consequently lesser grades of anoxæmia cannot be ascertained on clinical grounds.

The electrocardiogram is of considerable aid in determining the presence of cor pulmonale. In all of the cases cited above, electrocardiographic changes characteristic of chronic cor pulmonale were found. In our pulmonary clinic we have adopted the policy of establishing the degree of pulmonary insufficiency in all patients with emphysema. A routine work-up consists of a history and physical examination, a chest radiograph, electrocardiogram and lung volume studies, including residual air and a determination of arterial oxygen saturation with an earpiece oximeter. If any degree of unsaturation of arterial blood is present with the patient at rest, an arterial puncture is done and analyses for O_2 content and capacity as well as CO_2 content are made. Patients showing both hypercapnia and anoxæmia are carefully watched and oximeter readings are taken from time to time in order to detect any sudden worsening in their pulmonary function.

Because of the progressive pathological changes seen in emphysema, it is to be expected that a stage will ultimately be reached when the remaining functioning lung tissue is incapable of sustaining life. However, it is our experience, as well as that of others,[17-19] that acute exacerbations of anoxia, usually precipitated by respiratory infections, are responsible for the majority of deaths in these patients. The respiratory infection need not take the form of a frank bronchopneumonia or lobar pneumonia, but is more often manifested by a slight increase in cough, with the expectoration becoming purulent and yellow or green in colour. There may be little or no change in the x-ray picture. A low-grade fever and a mild leukocytosis are usually found. Arterial oxygen saturation shows a further decrease. Early signs of congestive failure may be present. Immediate treatment is indicated at this stage, and in most cases is best carried out in hospital.

On admission to hospital a sputum culture is done. It is probably best to start the patient immediately on a broad spectrum antibiotic which may, if necessary, be changed at a later date when results of culture and antibiotic sensitivity tests are received. A bronchodilator, preferably by aerosol inhalation, should be given to improve air entry and promote drainage. Oxygen therapy can be omitted if there is only a moderate degree of hypoxæmia, but in most cases we felt that it was inadvisable to withhold oxygen, especially in the presence of cardiac failure. We have followed the recommendations of Barach[1, 3] and given oxygen by nasal catheter at low rates of flow. With the use of the earpiece oximeter at the bedside, we have adjusted the flow rate to produce an arterial oxygen saturation in the vicinity of 90%. This has been done on the assumption that ventilation may not be depressed by oxygen therapy if the anoxic stimulus to the chemoreceptors is not completely removed. In

severe respiratory depression, such as was found in the cases reported above, attempts have been made to improve ventilation by various mechanical devices. We have had as yet little or no success with a Drinker type respirator and an electrophrenic stimulator. We have not used the method of "exsufflation" recently described by Barach *et al.*[21]

The treatment of the patient with cor pulmonale in failure secondary to emphysema is not infrequently described as being identical to that of cardiac failure from valvular or myocardial disease. This point of view, in our opinion, is incorrect and is due to the similar clinical picture of venous engorgement, hepatomegaly, peripheral œdema and ascites. However, underlying physiological disturbances are quite different and require different measures for their correction. Anoxæmia is a late sequel in congestive failure due to acquired, non-pulmonary disease, but is of primary importance in cor pulmonale with failure and should be corrected with oxygen therapy. Polycythæmia and hypervolæmia, which are usually considered to be "compensatory mechanisms" in the anoxic emphysematous patient, almost certainly contribute to the eventual congestive failure. Cournand found polycythæmia to be present in 18 of 19 emphysematous patients in failure. By use of phlebotomies we have raised the arterial oxygen saturation[20] in five patients with secondary polycythæmia.

The importance of antibiotics in chronic cor pulmonale in failure has already been stressed. These three therapeutic measures, i.e. administration of oxygen and antibiotics and phlebotomies, plus the all-important bed rest, are usually enough to restore compensation in cor pulmonale. Digitalis, which is of such value in other forms of heart disease, is of secondary importance in cor pulmonale, but is usually given. Diuretics are usually prescribed for patients with cor pulmonale in failure. On purely theoretical grounds we question the advisability of drastic curtailment or excessive elimination of sodium, which is necessary for formation of bicarbonate to compensate for carbon dioxide retention. Similarly ammonium chloride may precipitate acidosis, or accentuate an already present acidotic state.

SUMMARY

1. The clinical picture of coma occurring in patients with advanced pulmonary insufficiency on excessive oxygen therapy has been described.

2. Patients with advanced emphysema, anoxæmia and hypercapnia may be readily precipitated into coma on excessive oxygen therapy. Anoxæmia and hypercapnia were present in a series of seven patients who became comatose following oxygen therapy and/or sedation.

3. Close surveillance of the patient with advanced emphysema is important.

4. Prompt treatment of pulmonary infection is required in these patients, or death will occur from pulmonary insufficiency or cardiac failure.

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TUBERCULOSIS IN INFANCY

Now that so much has been done to cut down the high mortality rate from tuberculosis, the diagnostic guards seem to be slipping. For instance, a recent survey (Kendig, E. L. Jr.: *Postgrad. Med.*, 16: 217, 1954) conducted among some 1,500 pædiatricians reveals that no more than 55% are using the tuberculin test routinely; and of those who do, no more than 21% use it on patients over three years old.

Since tuberculosis of infancy and early childhood cannot be satisfactorily diagnosed by physical examination and x-rays, the physician must employ the tuberculin test, either patch or intradermal, routinely, for early diagnosis. The test should be used between the ages of 12 and 18 months, and on all new patients more than a few months old. For routine case finding the patch test is quite enough. After thorough cleansing of the skin with acetone or ether, the patch may be applied, and should remain *in situ* for 48 hours. The test is read 48 hours after the patch has been removed. An area of redness and induration 0.5 cm. in diameter at the site of either or both of the peripheral squares is a positive reaction. Any suspicious reaction should be checked with the Mantoux. All children with known contacts should be carefully watched and studied.