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MANAGEMENT OF HYPOXIA FOLLOWING OPEN HEART SURGERY

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The most frequent causes of hypoxia following open heart surgery are retained secretions, and impaired pulmonary function due to fibrosis and pulmonary hypertension. The management of postoperative hypoxia in the past consisted of intratracheal aspiration, bronchoscopy, and intermittent positive pressure breathing. With the development of effective equipment for ventilatory assistance combined with an enlightened tracheotomy care program, a new and aggressive approach to postoperative airway management has evolved. Although the development of these concepts is largely the product of surgeons and cardiologists working in the field of open heart surgery, the benefits are being applied with increasing frequency to the management of postoperative general surgical patients as well as certain non-surgical patients with impaired ventilation.

RECOGNITION OF POSTOPERATIVE HYPOXIA

The postoperative patient who becomes hyperpneic, cyanotic and confused due to hypoxia presents an obvious need for urgent improvement in the oxygen transport system and the problem is easily recognized by all concerned. Lesser degrees of hypoxia, particularly in postoperative patients with other problems which must occupy the attention of the surgeon such as blood replacement, oliguria and cardiac arrhythmias, are apt to be overlooked. Hypoxia complicating these serious situations may then worsen and tip the balance toward a fatal outcome. Since it is possible to eliminate the burden of inadequate ventilation from the several difficulties preventing the patient’s recovery and allow attention to be focused on the remaining issues, it is important that even minor degrees of hypoxia be detected. Evaluation of hypoxia by observation of the patient’s skin and mucous membrane color is helpful but sometimes inadequate. An example of this deficiency may be observed in the operating room when the desaturated color of blood in the operative field indicates significant hypoxia to the surgeon and anesthesiologist at a time when the latter is unable to detect inadequate oxygenation by observations about the patient’s head. In order to improve our ability to recognize inadequate oxygen and carbon dioxide exchange in postoperative cardiac surgical patients, we have utilized an indwelling radial artery

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Flexible teflon needle in the radial artery which is used for postoperative blood gas analyses. Use of the hand is unrestricted. The incision in the antecubital fossa was used for a venous cutdown to monitor the central venous pressure.

teflon needle for serial blood gas sampling. The teflon needle with stylette* is inserted through a 1.5 cm. incision over the radial artery at the wrist during preparation of the patient for operation. The stylette is withdrawn and the needle attached to a stopcock (Figure 1). Ligation of the artery is unnecessary since the arterial wall seals the puncture site. When the needle is subsequently withdrawn, finger pressure over the incision is adequate for hemostasis. The needle may be used for monitoring arterial pressure or blood sampling during operation but is of greatest service in the postoperative period for blood gas analyses. Patency is assured postoperatively by injection of 1 cc. of Heparin solution into the needle every four hours. The cannula has now been used in 75 patients without complications. On a few occasions, it has been left in place for two weeks. Immobilization of the hand is not required because of the flexible teflon cannula. Blood gas and ph determinations were made using the Epsco polarograph with Beckman electrodes.

**Management of Postoperative Hypoxia**

Once the observation is made that the postoperative patient is inadequately oxygenated, the cause should be determined by clinical and laboratory observations. Three causes of hypoxia may be distinguished in postoperative open heart surgical patients — hypoventilation, reduced cardiac output and impaired alveolar-capillary

* Becton Dickinson, Rutherford, N. J.
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diffusion. Combinations of the three commonly co-exist; however, treatment is facilitated by identifying the most significant etiologic factor. A variety of pulmonary function and blood gas determinations may be performed to study these patients; however, arterial and venous oxygen and carbon dioxide levels, the pH and measurement of the tidal exchange have been adequate in our experience to identify the important causes. The indwelling arterial cannula facilitates the differential diagnosis by permitting serial sampling of the blood.

Hypoventilation: The simplest form of hypoxia to recognize and treat is that due to impaired ventilation. Examination of the patient reveals diminished chest wall excursion and measurement of the tidal volume through a tightly fitting mask or endotracheal tube indicates a reduced tidal exchange. Arterial and venous blood gas determinations in this situation demonstrate lowered oxygen values, depressed pH, and carbon dioxide retention (Figure 2). Administration of oxygen, clearing of the airway by tracheal aspiration or ventilatory assistance with a mask will promptly correct the abnormalities. Hypoventilation hypoxia in the cardiac surgical patient is readily managed by these measures; however, tracheotomy with ventilatory assistance may be required if the causes are not readily correctable, such as in certain

Figure 2
Chart of hypoventilation hypoxia.
Case 1. Hypoxia due to hypoventilation. Controlled ventilation through a tracheotomy was utilized in this severe myasthenia gravis patient following thymectomy. When artificial ventilation was discontinued on the second postoperative day, the arterial oxygen saturation fell to 80 per cent requiring reinstitution of ventilatory control. After two days of ventilatory assistance using the Bennett respirator, the patient was able to resume satisfactory breathing. Arterial $pO_2$ determinations have been converted to $O_2$ saturation for clarity.

non-cardiac surgical patients with massive chest trauma, refractory myasthenia gravis, aspiration pneumonitis or drug intoxication. The following case report concerns a non-cardiac surgical patient; however, the effects of hypoventilation and response to treatment are illustrative.

**CASE 1:** N. H., a 21 year old girl was incapacitated by refractory myasthenia gravis. Tracheotomy and controlled ventilation were required on November 13, 1963, following thymectomy which was performed through a midline sternum splitting incision. A radial artery teflon needle was used to monitor the blood gases over a period of 2½ weeks following surgery. Prostigmine, which had become ineffective, was discontinued while the patient was on controlled ventilation. A trial without ventilatory assistance on the second post-tracheotomy day resulted in a fall of the oxygen saturation to 80 per cent and necessitated re-institution of ventilatory assistance (Figure 3). She again became responsive to prostigmine and it was possible to wean her from the ventilator on the fourth postoperative day. Serial blood gas studies were especially helpful in evaluating the adequacy of oxygenation in this Negro patient.

**Low Cardiac Output Hypoxia:** The postoperative cardiac surgical patient who is hypoxic due to reduced cardiac output presents a picture of peripheral cyanosis and moderate hyperpnea in addition to hypotension. Blood gas studies reveal lowered arterial oxygen values and disproportionate venous hypoxia due to poor peripheral circulation with high oxygen extraction in the tissues. Carbon dioxide levels are
normal or reduced as a result of hyperventilation and the increasing acidosis due to poor peripheral circulation is reflected in the falling pH (Figure 4). With further progression in the acidosis, acetone may be detected on the patient’s breath and urinary output decreases. When the remedial causes of lowered cardiac output, such as inadequate blood replacement, arrhythmias, incomplete digitalization and pericardial tamponade have been eliminated, a variety of etiologic factors remain which are usually refractory to specific treatment. This group, for want of more specific definition, is referred to as cardiogenic in origin and may result from myocardial infarction due to coronary atherosclerosis or embolization of air or particulate matter to the coronary arteries. Myocardial hypoxic damage may also follow inadequate coronary perfusion during aortic valve surgery. Unrecognized valvular disease, incomplete relief of valve stenosis or insufficiency due to leakage around a valve prosthesis are other less common causes of impaired cardiac output.

Specific treatment of the cardiogenic type of postoperative low cardiac output must be indirect until prolonged postoperative extracorporeal pump assistance becomes practical. Isuprel is frequently helpful and is preferred to peripheral vasoconstrictors. Since the patient is not suffering from hypoventilation, controlled ventilation through a tracheotomy fails to improve oxygenation providing there is normal pulmonary function. Many of these patients, however, have pulmonary vascular damage and fibrosis which markedly decreases pulmonary efficiency. These patients will of course benefit from controlled ventilation. An additional factor favoring the use of ventilatory
assistance in patients with low cardiac output hypoxia is elimination of the considerable “cost of breathing” or muscular work of ventilation. Although the resting ventilatory effort in normal subjects may require but 1 to 2 per cent of the total oxygen requirements, patients who must hyperventilate because of impaired pulmonary efficiency may use up to 30 per cent of their already deficient oxygen supply for ventilatory work during this critical phase of recovery. Placing the patient on controlled ventilation removes this energy requirement which is then supplied by the ventilator. If controlled ventilation is of benefit, repetition of the blood gas and pH studies will indicate improvement. In addition to controlled ventilation, the acidotic patient with low cardiac output hypoxia must receive appropriate hydrogen ion binding medication, such as sodium bicarbonate solution or Tham. The following case presentation illustrates the clinical and laboratory findings in low cardiac output hypoxia.

CASE 2: G. S., a 47-year-old male office worker underwent open heart surgery for advanced mitral insufficiency on June 23, 1964. The preoperative pulmonary artery pressure was 104/40 mm Hg and the pulmonary diffusion capacity (DLco) was normal. The mitral valve was replaced with a Starr-Edwards ball valve prosthesis and tracheotomy was done following thoracotomy closure. The pump run was of 70 minutes duration and the patient awakened promptly on reaching the recovery room. Assisted ventilation was maintained continuously and the arterial oxygen which was sampled through a teflon needle remained fully saturated. The venous oxygen saturation however fell progressively during the succeeding three days indicating inadequate peripheral perfusion. There was increasing hypotension and metabolic acidosis. Isuprel improved the cardiac output which partially relieved the peripheral signs of hypoxia but death ensued on the third postoperative day (Figure 5). At necropsy, a large posterior myocardial infarction was demonstrated along with coronary atherosclerosis. The prosthetic mitral valve was properly implanted.

Figure 5
Case 2. Hypoxia due to low cardiac output. Following prosthetic substitution of the mitral valve and tracheotomy, this patient developed increasing hypotension and cyanosis. Although the arterial oxygen remained saturated, the venous oxygen content and pH progressively fell, indicating inadequate cardiac output. Controlled rather than assisted ventilation would now be preferred.
Hypoxia Due to Impaired Alveolar-Capillary Diffusion: This form of postoperative hypoxia is primarily seen in patients who have required prolonged cardiopulmonary by-pass for repair of advanced and complicated intracardiac lesions. It occurs with greatest frequency in our experience in patients who have a pre-existing reduction in pulmonary function due to long-standing pulmonary hypertension. Patients undergoing open mitral valve surgery account for many of the patients in this group. This form of impaired alveolar-capillary diffusion increases in severity during the first 36 to 48 hours postoperatively. It is frequently complicated by hypoventilation since many of these patients have impaired pulmonary function.

The patient is markedly cyanotic and hyperpneic but has a satisfactory blood pressure. Blood gas determinations show arterial and venous hypoxia with little or no change in the pH levels (Figure 6). Since carbon dioxide diffuses across the capillary membrane with 20 times more facility than oxygen, there is usually no impairment of carbon dioxide transfer. The presence of a normal cardiac output prevents metabolic acidosis and lowered pH. The minute respiratory volume is normal or above indicating that hypoventilation is not a causative factor in this type of hypoxia. Patients with impaired alveolar-capillary diffusion improve with oxygen administration. If arterial sampling continues to show hypoxia after instituting oxygen therapy by mask, then tracheotomy and controlled ventilation should be employed, particularly if there is a hypoventilation component due to reduced pulmonary function.

Figure 6
Chart of impaired alveolar-capillary membrane diffusion hypoxia.
The cause or causes of this uncommon but perplexing postoperative complication of open heart surgery are under investigation in several centers. Although few pertinent clinical observations have been made, there is increasing experimental evidence to indicate that a reduction in alveolar surface active material — surfactant — is involved. This material which has been identified as a lipoprotein is responsible for preventing spontaneous alveolar collapse during respiration. The clinical and experimental pathologic picture is remarkably consistent and shows patchy intra-

![Figure 7]

Case 3. a. Chest x-ray on day of operation which consisted of mitral valve replacement and aortic valve drilling. A patchy infiltrate is present.

b. Forty-eight hours postoperatively there is a marked increase in the pulmonary infiltrate.

c. At three months postperfusion, the chest x-ray shows clearing of the pulmonary infiltrates. The mitral valve prosthesis may be seen.
alveolar hemorrhage and fibrin deposition with granulocytic infiltration of the alveolar septa. The lesions are seen with all types of pump oxygenator equipment but are related to the length of bypass and therefore in part to the degree of blood trauma.

Patients with impaired alveolar-capillary diffusion may need ventilatory assistance for two to three weeks before pulmonary function improves sufficiently to permit adequate respiration. The patient in the following case report continued to show a lowered pulmonary diffusion capacity (DL_{co2}) for several months postoperatively.

CASE 3: A. E., a 60-year-old housewife underwent prosthetic ball-valve replacement of the mitral valve and aortic valve debridement using the air turbine drill on December 10, 1963. The preoperative pulmonary artery pressure was 90/50. By-pass was of 105 minutes duration. An indwelling radial artery teflon needle was used to repeatedly sample blood gases. After awakening from anesthesia in the recovery room and while receiving oxygen by nasal catheter, the arterial oxygen saturation fell to 80 per cent. The carbon dioxide level and pH were normal. Tidal exchange was 800 cc's with a rate of 30/min. Hypoxia was considered to be due to a diffusion disturbance across the alveolar-capillary membrane combined with poor pulmonary function resulting from pulmonary hypertension. Tracheotomy and assisted ventilation were instituted and the blood oxygen saturation promptly returned to normal. The chest x-ray demonstrated marked patchy infiltration at 48 hours postoperatively (Figure 7). On the fifth and 12th postperfusion days, the patient was allowed to breathe spontaneously through the tracheotomy for 15 minutes. On both occasions, the arterial oxygen saturation again fell to 80 per cent. By the 15th postperfusion day, the patient was able to maintain normal arterial oxygen values with spontaneous respiration. The carbon monoxide diffusion capacity (DL_{co2}) at this time was 8.9 cc/mm. Hg, which was less than 50 per cent of the normal preoperative value of 22 cc/mm. Hg. Eventually, the DL_{co2} returned to normal by the ninth postoperative month and the patient made an excellent recovery (Figure 8).
Tracheotomy and Artificial Ventilation

Tracheotomy is always performed in the operating room with adequate lighting, exposure, personnel and equipment, whereas, emergency control of the airway is obtained by insertion of an endotracheal tube if the need arises outside the operating room area. A cuffed No. 8 tracheotomy cannula is used in adults and shortened tubes of small size for infants and children. Tracheotomy suction is performed as a sterile maneuver both in the operating room and in the postoperative period. A new, sterile, plastic aspiration catheter is used for each suctioning procedure (Figure 9). Catheters are cleansed, resterilized by ethylene oxide and reused. Personnel caring for the tracheotomy wear a surgical mask and handle the aspiration end of the catheter with sterile forceps. All tracheotomy patients are cared for in the Special Care Unit for postoperative patients. Only through the development of this strict program has it been possible to avoid the introduction of extraneous infection into the tracheobronchial tree.

A nebulizing device for humidification is used at all times, either through a tracheotomy shield or through the ventilator. Three to five cc's of sterile saline are injected into the tracheotomy tube for irrigation before each suctioning procedure. Nebulizing devices and connecting tubing are gas sterilized between patient usage. It has been necessary to use intratracheal instillations of proteolytic enzymes only on rare occasions with this program of tracheotomy care.

The Bennett, Bird and Air Shields ventilators have been used in the management of these patients. The Engstrom respirator allows sophisticated adjustments which are not available with these instruments, however the less complicated devices have proven entirely adequate and permit time and attention to be focused on other equally pressing problems. Involved computations of the required ventilatory volumes for normal patients such as those recommended for the Engstrom respirator are of little value when dealing with impaired pulmonary function. The best test of adequate ventilation is production of visible chest cage expansion accompanied by improvement in the blood gas analyses. Although total ventilatory control may be desirable to eliminate the energy requirements of the "cost of breathing" in patients with low cardiac output hypoxia, ventilatory assistance is used most frequently since observation of the patient's spontaneous ventilatory rate is helpful in assessing the adequacy of ventilation. The need for controlled rather than assisted ventilation is indicated when serial blood gas studies demonstrate impaired oxygen or carbon dioxide exchange while the patient is receiving ventilatory assistance. Ventilation pressures above 20 centimeters of water are avoided because of the deleterious effect on right heart filling. The ventilating equipment and connecting tubing are gas sterilized between patient usage and every three days while in continuous use on the same patient.

Summary

The recognition of postoperative hypoxia in open-heart surgical patients is facilitated by an indwelling radial artery teflon needle and the performance of serial blood gas studies. Three types of postoperative hypoxia may be distinguished by clinical observation, serial blood gas studies and measurement of the tidal exchange.
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1. Hypoxia due to hypoventilation from retained secretions or drug depression is recognized by reduced ventilatory volume, acidosis, carbon dioxide retention and lowered oxygen saturation. Oxygen administration and tracheal aspiration are usually sufficient to correct the hypoxia.

Figure 9
Sterile aspiration technique used for tracheotomy care in the Special Care Unit. A mask is worn and the sterile aspiration catheter is handled with sterile forceps. A new catheter is used for each aspiration.
2. Hypoxia due to low cardiac output in the postoperative cardiac surgical patient may have a variety of causes. It is characterized by hypotension, normal or lowered CO₂ levels due to hyperventilation, depressed pH from metabolic acidosis and normal ventilatory volumes. The venous oxygen saturation is markedly depressed. Tracheotomy and ventilatory control are frequently of value in these patients because of co-existing impairment of pulmonary function due to pulmonary hypertension and to eliminate the considerable ‘‘cost of breathing’’ work in the hyperventilating, hypoxic patient.

3. Hypoxia due to impaired alveolar-capillary diffusion occurs in the presence of normal ventilation volumes, CO₂ and pH levels but with marked arterial desaturation. Tracheotomy and use of ventilatory assistance will improve oxygenation when hypoxia is due to a diffusion disturbance. Several weeks may be required before pulmonary function improves sufficiently to permit spontaneous breathing. The cause of this diffusion disturbance is not known but apparently is related to impaired function of the surface active material in the alveolus.

A tracheotomy care program is presented which utilizes a sterile aspiration technique that is carried out in a Special Care Unit. Constant humidification by nebulization and installation of sterile saline at the time of tracheal aspiration are mandatory.

Relatively simple ventilatory equipment is preferred. Ventilatory assistance is used more frequently than control. All equipment which contacts expired air is gas sterilized between patient usage and after three days of use by each patient.

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