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Jean Van de Kerckhof

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Recognition and Management of Hypoxia in Postoperative Open Heart Surgical Patients

Rodman E. Taber, M.D.* and Jean Van de Kerckhof, M.D.†

Hypoxemia is a frequent contributing factor to morbidity and mortality following open heart surgery. The most common causes of hypoxia in these postoperative patients are retained secretions, reduced cardiac output, and impaired pulmonary function due to fibrosis and pulmonary hypertension. Until relatively recently, the usual management of postoperative airway problems consisted of intratracheal aspiration, bronchoscopy and intermittent positive pressure breathing. With the development of effective equipment for ventilatory assistance and the adoption of an aggressive attitude toward control of the airway, an enlightened approach has been evolved toward the management of hypoxia in postoperative patients. Although the development of these concepts is largely the product of surgeons and cardiologists working in the field of open heart surgery,1,2,3 the benefits are being applied with increasing frequency to the management of postoperative general surgical patients4 as well as certain nonsurgical patients with hypoxia.

Recognition of Postoperative Hypoxia

The postoperative patient who becomes hyperpneic, cyanotic and confused on the basis of 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 are apt to be overlooked, particularly in postoperative patients with other problems which must occupy the attention of the surgeon such as blood replacement, oliguria and cardiac arrhythmias. Complicating these serious situations unrecognized

*From the Division of Thoracic Surgery, Henry Ford Hospital, Detroit, Mich.
†Present address: Diest, Belgium
A. The flexible teflon needle has been inserted into the radial artery and a stopcock attached. Serial blood gas and pH levels are then readily available. The wrist is not immobilized. The antecubital incision is at the site of a venous cut-down which was used to monitor central venous pressure.

B. Teflon needle with stylette.

Figure 1

A. The flexible teflon needle has been inserted into the radial artery and a stopcock attached. Serial blood gas and pH levels are then readily available. The wrist is not immobilized. The antecubital incision is at the site of a venous cut-down which was used to monitor central venous pressure.

B. Teflon needle with stylette.

hypoxia 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, allowing attention to be focused on other important issues, it is vital that even minor degrees of hypoxia be detected. Evaluation of hypoxia by observation of the respiratory rate and the patient's skin 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, but the latter is unable to detect cyanosis about the patient's head. In the same manner, clinically recognizable cyanosis is frequently absent in the postoperative patient with significant hypoxia. 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 teflon needle for repeated blood gas sampling. Suspicious clinical indications of hypoxia are thereby rapidly confirmed with objective measurements. The teflon needle with stylette* is inserted through a 1.5-cm skin 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 (Fig. 1). Ligation of the artery when the needle

*Becton Dickinson, Rutherford, N. J.
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is subsequently withdrawn is unnecessary since the arterial wall seals the puncture site. When the needle is removed, finger pressure for several minutes over the incision is adequate for hemostasis. The needle may be used for monitoring arterial pressure as well as blood sampling during operation but it is of greatest service in the postoperative period for blood gas analyses. Patency is assured postoperatively by the injection of 1 cc of dilute Heparin solution into the needle every two hours. The cannula has now been used in over 200 patients without serious complications. On a few occasions, it has been left in place for two weeks. We have had to remove the cannula because of ischemic signs in the hand on three occasions. Arterial spasm then disappeared and there was no subsequent tissue necrosis. The injection of 2 cc of one percent xylocaine through the teflon needle is helpful in relieving this type of arterial spasm when it occurs. Immobilization of the wrist is not required because of the flexible teflon, which is turned away from the wrist and taped to the forearm. Blood gas and pH determinations were made using the Epsco polarograph and Beckman electrode. An oximeter* is also kept in the Special Care Unit where these patients are cared for. It is available for use by resident personnel on a 24-hour basis to perform rapid oxygen saturation measurements.

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 types of hypoxia may be distinguished in postoperative open heart surgical patients; that due to hypoventilation, reduced cardiac output and impaired alveolar-capillary membrane diffusion. Combinations of the three commonly coexist; 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 pO₂ and pCO₂ levels in the arterial and venous blood, the pH and determination of the tidal exchange volume have been adequate in our experience to identify the important causes. The indwelling arterial needle facilitates the differential diagnosis by permitting serial sampling and evaluation of the results of treatment.

Hypoventilation: The easiest form of hypoxia to recognize and treat is that due to impaired ventilation. Examination of the patient reveals diminished chest wall excursion. Measurement of the tidal volume with a ventilometer through either a tightly fitting mask or an endotracheal tube indicates a reduced tidal exchange. Arterial and venous blood gas levels in this situation demonstrate lowered oxygen values, depressed pH and carbon dioxide retention (Fig. 2). Administration of oxygen, clearing of the airway by tracheal aspiration, or ventilatory assistance with a mask will usually correct the abnormalities. However prolonged (up to 24 hours) intratracheal intubation or tracheotomy may be required. Vigorous measures are particularly needed if the causative factors are not readily reversible. Hypoventilation due to postanesthetic depression is best managed by prolonged endotracheal intubation and

*American Optical Company
assisted ventilation. The following case report concerns a noncardiac surgical patient. However the signs of hypoventilation hypoxia and response to treatment are illustrative.

CASE 1: N. H., a 21-year-old girl was incapacitated by refractory myasthenia gravis. Tracheostomy 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% and necessitated reinstitution of ventilatory assistance (Fig. 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 impaired cardiac output on a primary myocardial basis presents a
CASE 1. Hypoxia from hypoventilation. It was necessary to use controlled ventilation through a tracheotomy following thymectomy in this patient with severe myasthenia gravis. An attempt was made to discontinue artificial ventilation on the second postoperative day but the arterial oxygen saturation fell to 80%, requiring reinstitution of ventilatory control. After two more days of artificial ventilation, the patient was able to resume spontaneous breathing. Arterial pO₂ determinations have been converted to O₂ saturation for clarity.

picture of mottled peripheral cyanosis and other signs of poor tissue perfusion such as anuria, and an obtunded sensorium. There is 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 (Fig. 4). With further progression in the acidosis, acetone may be detected on the patient’s breath. 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 usually referred to as cardiogenic since impaired myocardial contraction is present. Myocardial infarction on the basis of coronary artery occlusive disease is a frequent source of reduced cardiac output in older patients undergoing open heart surgery. Diffusely scattered microinfarctions due to embolization of air or particulate to the coronary arteries during cardiopulmonary bypass are another source of myocardial injury and impaired function. Inadequate coronary artery perfusion...
Typical findings in a patient with hypoxia due to impaired cardiac output. The pO₂ and pH are depressed as in the previous example but the pCO₂ is normal or lowered.

during aortic valve surgery may also lead to hypoxic myocardial damage. Incomplete relief of valvular stenosis or insufficiency due to leakage around a valve prosthesis are other causes of impaired cardiac output but these are not primarily myocardial in origin.

Specific treatment of the cardiogenic type of postoperative low cardiac output is usually impossible. Prolonged postoperative extracorporeal pump assistance may be helpful in some of these cases in the future. Isoproterenol (Isuprel) is frequently helpful in improving both cardiac output and peripheral tissue perfusion. Isoproterenol with its inotropic and peripheral vasodilating effects is preferred to vasoconstrictors. Since the patient is not suffering from hypoventilation, controlled ventilation through a tracheotomy usually fails to improve oxygenation, providing there is no pulmonary fibrosis or endarteritis. Many of these patients, however, have impaired pulmonary function due to the effects of prolonged pulmonary hypertension, and they will 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 one or two percent of the total oxygen requirements, patients who must hypertventilate because of impaired pulmonary efficiency may use up to 30% of their already deficient oxygen supply.
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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 mechanical ventilator. If controlled ventilation is of benefit, repetition of the blood gas and pH studies will indicate improvement. In addition to controlled ventilation, the acidic 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 104/60 mm Hg and the pulmonary diffusion capacity (DLaw) 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

![Graph](image)

**Figure 5**

CASE 2. Hypoxia from impaired cardiac output. Following mitral valve replacement with a ball valve and elective tracheotomy, the patient developed increasing hypotension and cyanosis. Although the arterial blood remained saturated, the venous oxygen saturation and pH fell progressively over a period of three days, indicating poor tissue perfusion. An anterior myocardial infarction was identified as the source of impaired cardiac output at necropsy.
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acidosis. Isuprel improved the cardiac output which partially relieved the peripheral signs of hypoxia but death ensued on the third postoperative day (Fig. 5). At necropsy, a large posterior myocardial infarction was demonstrated along with coronary atherosclerosis. The prosthetic mitral valve was properly implanted.

_Hypoxia Due to Impaired Alveolar-Capillary Membrane Diffusion:_ This uncommon form of postoperative hypoxia is primarily seen in patients who have required prolonged cardiopulmonary bypass for repair of advanced and complicated intracardiac lesions. In our experience, it occurs with greatest frequency 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 in this group. The signs of impaired alveolar-capillary membrane diffusion typically increase in severity during the first 36 to 48 hours postoperatively. This source of hypoxia is often accompanied by hypoventilation since many of these patients have impaired pulmonary function.

In this syndrome, 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 (Fig. 6). Since carbon dioxide diffuses

![Impaired Alveolar-Capillary Membrane Diffusion Hypoxia](image)

**Figure 6**

Typical findings in a patient with hypoxia due to impaired alveolar-capillary membrane diffusion. The pCO₂ and pH are normal while the pO₂ is depressed.
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across the capillary membrane with 20 times more facility than oxygen, there is usually no impairment of carbon dioxide transfer. The presence of a near normal cardiac output prevents metabolic acidosis and a lowered pH. The minute respiratory volume is normal or above indicating that hypoventilation is not a causative factor if the hypoxia is caused solely by a diffusion disturbance. 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.

The cause or causes of this uncommon and 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-alveolar hemorrhage and fibrin deposition (hyaline membrane) with granulocytic infiltration of the alveolar septa. These findings are similar to the oxygen toxicity type of damage to the lungs produced by prolonged inhalation of oxygen concentrations greater than 50%. The lesions are seen following use of 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 unassisted respiration. The patient in the following case report continued to show a lowered pulmonary diffusion capacity (DLcO2) 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. Bypass was of 105 minutes duration. An indwelling radial artery teflon needle was used to sample blood gases repeatedly. After awakening from anesthesia in the recovery room and while receiving oxygen by nasal catheter, the patient showed arterial oxygen saturation fallen to 80%. The carbon dioxide level and pH were normal. Tidal exchange was 800 cc’s with a rate of 30 per minute. 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 (Fig. 7). On the 5th 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%. By the 15th post-perfusion day, the patient was able to maintain normal arterial oxygen values with
A.) Chest x-ray on day of operation which consisted of mitral valve replacement and aortic valve drilling. A patchy infiltrate is present and the blood gas studies indicated an alveolar capillary block.

B.) Forty-eight hours later there is a marked increase in the bilateral pulmonary infiltrate.

C.) Three months after operation there is clearing of the pulmonary changes. The pulmonary diffusion capacity (DL_{CO}) returned to normal nine months postoperatively.

Figure 7
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spontaneous respiration. The carbon monoxide diffusion capacity (DLco) at this time was 8.9 cc/mm Hg which was less than 50% of the normal preoperative value of 22 cc/mm Hg. By the ninth postoperative month the DLco had returned to normal and the patient made an excellent recovery (Fig. 7).

Tracheotomy and Artificial Ventilation

Tracheotomy should always be performed in the operating room with adequate lighting, exposure, equipment and assisting personnel. Emergency control of the airway in the patient's room is best obtained by insertion of an endotracheal tube. A cuffed no. 8 Morsch tracheotomy cannula is usually used in adult patients and shortened tubes in the smaller sizes 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 (Fig. 9). Catheters are cleansed, resterilized with ethylene oxide and reused. Personnel caring for the tracheotomy wear a surgical mask and handle the aspiration end of the catheter with sterile forceps. The 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.

CASE 3. Hypoxia due to impaired alveolar-capillary membrane diffusion. The patient demonstrated arterial desaturation following open mitral valve replacement and aortic valve drilling. Tracheotomy and ventilatory assistance corrected the hypoxia but could not be discontinued without recurrence of the arterial desaturation (5th and 12th days) until the 15th postoperative day. The carbon monoxide diffusion capacity (DLco) was markedly reduced postoperatively indicating impaired alveolar-capillary membrane diffusion. It returned to normal by the ninth postoperative month.
Sterile aspiration technique used for tracheotomy care in the Special Care Unit. A mask is worn and the aspirating end of the catheter is handled with sterile forceps. A new, sterile catheter is used for each aspiration.

Prolonged intratracheal intubation with an endotracheal tube during the postoperative period has enjoyed recent popularity. We have frequently used an endotracheal tube in this manner during the first postoperative night but prefer to perform a tracheotomy if intubation is required beyond 24 hours.

A nebulizing device for humidification is used at all times, either through a tracheotomy shield or through the ventilator. Before each suctioning procedure, three to five cc’s of sterile saline are injected into the tracheotomy tube for irrigation. Nebulizing devices and connecting tubing are gas-sterilized between patient usage and after every 24 hours of use. It has been necessary to use intratracheal instillations of proteolytic enzymes only on rare occasions with this program of tracheotomy care. Frequent culture of tracheal secretions is performed. If clinical infection develops, isolation procedures are instituted.

The Bennett, Bird and Air-Shields ventilators have been used in the management of these patients. These instruments leave much to be desired but represent significant
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steps in the development of adequate ventilatory equipment. Although the Engstrom respirator, a volume controlled device, allows sophisticated adjustments which are not available with these instruments, the less complicated devices have proven reasonably effective, 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. This is because 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 cm of water are avoided if at all possible because of the deleterious effect on right heart filling.

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.

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.

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 coexisting 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.
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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 24 hours of use by each patient.

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