Severe Hypoxemia Secondary to Pulmonary Embolization Treated Successfully with the Use of a CPAP (Continuous Positive Airway Pressure) Mask*

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We describe a patient who was admitted with acute onset of dyspnea and pleuritic chest pain. The patient was in acute hypoxic respiratory failure documented by arterial blood gas levels. The severe hypoxemia was refractory to 100 percent O2 administration. The cause of the patient's sudden deterioration was a pulmonary embolus documented by angiography. The patient was managed successfully with heparin therapy. A continuous positive airway pressure (CPAP) mask corrected the severe hypoxemia, which otherwise would have required a more invasive method of respiratory support.

It should be the goal of every physician to search for new techniques which reduce the cost of medical care and at the same time avoid the use of the more invasive techniques with their potential risks and added cost. The recent use of CPAP is a clear example of such new technique. The present literature is generous with examples of such situations in which the use of CPAP, applied by means of a face mask, avoided endotracheal intubation and mechanical ventilation both in children1 and in adults.2-4,7 Details of the actual mechanical setup,1-3 guidelines,4 and indications6 for the use of CPAP mask in adults with respiratory failure are also available. The purpose of this report is to illustrate yet another possible indication for this technique.

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CASE REPORT

In November 1975, a diagnosis of ovarian carcinoma, stage 3, was made in a 55-year-old woman. Treatment consisted of bilateral hysterectomy and bilateral salpingo-oophorectomy followed by adjuvant chemotherapy with methotrexate, cyclophosphamide and 5-fluorouracil for approximately 18 months. The patient was readmitted in September, 1977, with symptoms of pleuritic chest pain. She was in no obvious respiratory difficulty; however, chest x-ray films at the time revealed a left pleural effusion. Thoracocentesis and chest tube drainage revealed approximately 3,000 ml of serosanguineous fluid. Analysis of the pleural fluid documented the presence of adenocarcinoma, metastatic to the pleural space. This was followed by instillation of intrapleural tetracycline. The patient was discharged on Oct 3, 1977, in good condition.

She was readmitted ten days later because of reaccumulation of the left pleural effusion and again no respiratory distress was noted. Chest tube drainage was initiated, followed by intrapleural instillation of bleomine and chemotherapy with doxorubicin and vincristine. The patient was discharged in good condition five days later after removal of the chest tube.

She was readmitted the same afternoon with sudden onset of dyspnea and pleuritic chest pain, which awoke her from her nap. Physical examination revealed a 55-year-old cyanotic white woman in obvious respiratory distress. Vital signs were as follows: blood pressure 110/70 mm Hg, pulse rate 130/min and regular, respiration rate 30-40/min and shallow, and temperature 36.40°C (97.6°F). No jugular venous distention was present. Cardiovascular examination revealed no murmurs or gallops. Examination of the chest revealed
only the previously noted dullness to percussion at the base of the left hemithorax with absent breath sounds and absent vocal fremitus. Arterial blood gas levels with the patient breathing room air were as follows: PaO₂ 27 mm Hg; PaCO₂ 28 mm Hg; pH 7.50; and on 100 percent oxygen the PaO₂ rose to 80 mm Hg. An electrocardiogram showed sinus tachycardia. Chest x-ray film revealed opacification of the left hemidiaphragm by the previously documented pleural effusion. Pulmonary embolization was documented by angiography (Fig 1).

The patient was started on heparin therapy and on CPAP, with the use of a face mask (Bird mask No. 4343-5) on 5 cm H₂O pressure, and .60 Fio₂. Her condition improved dramatically, with the arterial blood gas levels on the above regimen showing marked improvement (Fig 2). By the fourth day of hospitalization, the patient no longer required the use of CPAP, and arterial blood gas measurements obtained on a Venturi mask at an Fio₂ of .28 were as follows: PaO₂ 75 mm Hg; PaCO₂ 30 mm Hg; and pH 7.47. The patient was discharged on sodium warfarin (Coumadin) therapy, and chemotherapy was reinstituted.

**Discussion**

The cause of arterial hypoxemia in pulmonary embolization often remains obscure. The decreased arterial Po₂ has been variously ascribed to different physiologic entities: a) diffusion impairment in areas with high blood flow and, therefore, reduced transit time; b) opening of potential pulmonary arteriovenous anastomoses as a consequence of the high pulmonary pressure; c) decrease in mean ventilation-perfusion ratio of the perfused, nonembolized area; d) right-to-left shunting of a fraction of the cardiac output secondary to atelectasis, pneumoconstriction, or pulmonary edema.

The lack of correlation between the mean pulmonary artery pressure and the percentage of the cardiac output shunted weighs against the presence of arteriovenous anastomoses as the cause of arterial hypoxemia. The decrease in mean ventilation-perfusion ratio of the perfused, nonembolized areas has been demonstrated only in experimental animals with experimentally induced pulmonary embolization. Wilson et al demonstrated the persistence of shunting for several weeks after the pulmonary embolus, making pulmonary edema an unlikely cause. Furthermore, the authors failed to document a significant obstructive ventilatory defect in patients with pulmonary embolization, raising the question whether bronchoconstriction was a major contributing cause of the hypoxemia being observed. The high incidence of discoid atelectasis observed on chest x-ray films after pulmonary embolus and the presence of roentgenographic evidence suggesting microatelectasis, loss of volume and elevated hemidiaphragms suggested to Wilson et al that atelectasis was the major cause of the arterial hypoxemia seen in pulmonary thromboembolism.

In order for the shuntlike effect to appear, perfusion would have to be preserved or restored in the areas of atelectasis. Wessler et al demonstrated persistence of blood flow after releasing a large thrombus from a segment of the inferior vena cava by injecting radioactive microspheres and later measuring the radioactivity. Pulmonary emboli that only partially occlude flow may cause alterations in surface-active properties enough to cause atelectasis. It is possible that spontaneous fibrinolysis with clearing of some emboli occurs faster than restoration of surface-active properties of the alveoli in the embolized regions.

The use of CPAP in hypoxemic respiratory failure secondary to pulmonary embolization seems a logical supplement to the treatment of this condition, if, indeed, the arterial hypoxemia is due to venous admixture secondary to low V/Q ratios in areas of unstable alveoli which would lead to a loss of functional residual capacity. In matching ventilation to perfusion in disease states, the method of delivering ventilation becomes important. With CPAP, functional residual capacity increases, and areas that are perfused but poorly ventilated become reexpanded. Another advantage is that patients on whom CPAP therapy is used and mechanical ventilation plus PEEP (positive-end-expiratory pressure) is avoided, have a smaller impairment of venous return and
a smaller reduction in cardiac output.5,7

In our patient, we successfully reversed the profound hypoxemia with the use of a CPAP mask. We documented in the first three days of hospitalization a progressive decrement in \( F_{\text{O}_2} \) requirement, with a marked improvement in arterial oxygen tension along with a trend to normalization of the a/A ratio (Fig 2).

Of all the proposed etiologies for the arterial hypoxemia in pulmonary embolization, our patient's response to CPAP therapy supports the concept of venous admixture occurring possibly secondary to microatelectasis. We further suggest the use of CPAP mask as an adjunctive modality of therapy in the treatment of hypoxemic respiratory failure secondary to pulmonary embolization, provided the necessary guidelines and criteria for the selection of these patients are met.

REFERENCES

18 Fleischer P, Hampton AO, Castleman B: Linear shadows in the lung (interlobar pleuritis, atelectasis and healed infarction). Am J Roentgenol 46: 610-618, 1941

Left Ventricular Outflow Obstruction Produced by a Pedunculated Fibroma in a Newborn*

Clinical, Angiographic, Echocardiographic and Surgical Observations

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An unusual case of a one-day-old infant with significant left ventricular outflow obstruction produced by a pedunculated fibroma is described. The clinical findings were indistinguishable from severe valvar or subvalvar aortic stenosis. The tumor was difficult to detect by echocardiography. It produced an echo-free widening of the left ventricular outflow tract. Left ventricular cineangiography clearly demonstrated a mobile mass beneath the aortic valve.

Fibromas of the left ventricle are rare cardiac tumors, which are usually located within the anterior wall and/or septum of the left ventricle.1,2 It has been suggested that a fibroma could create inflow or outflow obstruction of either ventricle,3,4 but the actual occurrence of obstruction is rare.4,5 Subaortic obstruction appeared to be present (at autopsy) in three cases,1,3,4 but hemodynamic or angiographic proof of obstruction has not been described.

The present case documents several unusual or previously undescribed features of a ventricular fibroma: (1) attachment of the tumor to the septum by a peduncle, in contrast to the usual intramural location; (2) its angiographic appearance; (3) its difficult echocardiographic

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