rhythm was restored after five days of therapy. Propranolol also has been used successfully to treat SVT in a fetus with Wolff-Parkinson-White syndrome by Teuscher et al. Hypoglycemia and bradycardia have been observed by Habib and McCarthy in four neonates with good Apgar scores whose mothers had received propranolol during pregnancy. Cottrill et al have also reported marked bradycardia and hypoglycemia in a neonate whose mother had received 160 mg of propranolol daily. These authors have considered propranolol therapy during pregnancy as a risk factor for the neonate. However, Rubin, on the basis of current available information on β-blockers in pregnancy, has cast doubts on the adverse effects of propranolol on the fetus. In conclusion, this case confirms the usefulness of ultrasound examination in the diagnosis of fetal cardiac failure and the effectiveness and safety of transplacental digoxin therapy for SVT.

REFERENCES

Pulmonary Arteriovenous Fistula Showing a Fall in Shunt Fraction During Exercise*

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A 23-year-old man with pulmonary arteriovenous fistulas of the right middle lobe is described. During the incremental exercise test, the shunt fraction dropped from 19 percent to 12 percent as the cardiac output increased. We discuss the mechanism of this fall in shunt fraction in this patient during exercise.

Exertional dyspnea is the most common symptom in patients with a pulmonary arteriovenous (AV) fistula, and the arterial oxygen tension has been reported to decrease during exercise in about half the patients. The patient described herein showed a fall in shunt fraction during the incremental exercise test. His arterial oxygen tension first increased and then decreased. The purpose of this report is to describe the behavior of the fistulas and the other pulmonary capillaries of this patient in response to increased cardiac output during exercise.

CASE REPORT

A 23-year-old man was referred to our hospital for evaluation of occasional mild dyspnea while sleeping. He had never felt dyspnea on exertion. There was no history of pulmonary disease or congenital malformations in the patient’s family, and he had no history of smoking.

The patient was a well-developed young man with normal vital signs and no cyanosis or digital clubbing. There were no hemangiomata or telangiectasias. The heart sound was normal and auscultation of the lungs revealed no rales or bruits.

The hemoglobin level was 17.9. The ECG findings were normal. The chest x-ray film showed two rounded densities in the right lung. Pulmonary angiographic studies (Fig I) demonstrated two pulmonary AV fistulas originating from the vessels of the right middle lobe. The pulmonary function tests were normal.

He underwent the incremental exercise test on a cycle ergometer (Mijnhardt Medical Instrument, Model FEMS) one week before and four months after surgery. Minute ventilation (Ve), oxygen consumption (Vo2), and carbon dioxide production (VCO2) were measured by an on-line microcomputer combined with a hot-wire respiratory flow meter, a zirconia solid electrolyte oxygen analyzer, and an infrared carbon dioxide analyzer (Minato Medical Science, System RM-200). The radial artery was cannulated, and a Swan-Ganz catheter was inserted for arterial and mixed venous blood sampling. The cardiac output and right-to-left shunt fraction were calculated according to the following formulas:

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Pulmonary Arteriovenous Fistula

I. Pulmonary angiogram showing two arteriovenous fistulas originating from the vessels of the right middle lobe.

Cardiac output = \( \frac{V_{O_2}}{(CaO_2 - C\bar{V}O_2)} \)

Shunt fraction = \( \frac{(Cc'O_2 - CaO_2)}{(Cc'O_2 - C\bar{V}O_2)} \)

where \( CaO_2 \) = arterial oxygen content; \( C\bar{V}O_2 \) = mixed venous oxygen content; and \( Cc'O_2 \) = end-capillary oxygen content. The end-capillary oxygen tension was regarded to be identical with the alveolar oxygen tension. All studies were performed with the patient breathing room air. The results of the tests are shown in Figures 2 and 3. In the preoperative evaluation, the intracardiac and pulmonary arterial pressures were normal. At rest in the sitting position, the arterial oxygen tension (PaO_2) was 73.1 mm Hg, the cardiac output was 6.1 L/min, and the shunt fraction was 19 percent. During the incremental exercise test in the sitting position, the shunt remained almost constant as the cardiac output increased, so the shunt fraction dropped inversely to 12 percent. The PaO_2 increased to 79.9 mm Hg at 20 W and then decreased to 70.7 mm Hg at 50 W. The test stopped at this workload because the patient complained of skipped heart beat and precordial discomfort.

On Jan 24, 1983, he underwent right middle lobectomy. His postoperative course was uneventful.

Four months after surgery, he was admitted for reevaluation. Pulmonary angiography demonstrated no residual fistulas. He underwent the exercise test again in the same manner. At rest, the PaO_2 was 102.4 mm Hg, the cardiac output was 4.3 L/min, and the shunt fraction was 2.8 percent. During exercise test, the shunt fraction did not exceed 4 percent. The preoperative and postoperative values of \( V_{E} \), \( V_{O_2} \) and \( V_{CO_2} \) were almost the same.

II. Changes in cardiac output (CO) and shunt relating to body position and work rate.

FIGURE 2. Changes in cardiac output (CO) and shunt relating to body position and work rate.

DISCUSSION

The clinical triad of cyanosis, exertional dyspnea, and digital clubbing is a frequent finding in patients with a pulmonary AV fistula. The severity of the clinical symptoms, however, depends on the degree of shunt—10 to 56 percent of these patients have been reported to have no complaints. Some papers have described the influence of exercise on patients with a pulmonary AV fistula, but no case has reported a fall in shunt fraction during exercise. Slutter-Eringa et al performed ergometric studies on 20 patients with a pulmonary AV fistula, and reported that eight of them showed a decrease in arterial oxygen saturation (SaO_2) of more than 5 percent and/or a decrease in PaO_2 of more than 10 mm Hg. Harrow et al reported a case of pulmonary AV fistula associated with chronic obstructive pulmonary disease (COPD) and ischemic heart disease. In that case, the exercise induced even greater hypoxemia and shunting (49 to 43 mm Hg and 37 to 39 percent, respectively). By et al described a 7-year-old boy and the results of exercise tests before and after surgery, but they did not calculate the shunt.

III. Changes in shunt fraction relating to body position and work rate.

FIGURE 3. Changes in shunt fraction relating to body position and work rate.
fraction.

In our patient, the shunt fraction dropped during exercise as the cardiac output increased. Studies of pulmonary function tests and cardiac catheterization of this patient were normal. The theoretic mechanism of this fall of shunt fraction is as follows. In the normal person, pulmonary vascular resistance is lowered in response to increased cardiac output by the mechanism of recruitment and distention of the pulmonary capillaries.\textsuperscript{9} We considered that pulmonary vascular resistance, except for the fistulas, dropped during exercise, but the resistance of the fistulas did not change, so that the increased blood flow circulated only through the normal pulmonary capillaries. This would explain why the results differed from those of Harrow et al\textsuperscript{10}, who had a larger fistula and combined with COPD.

The PaO\textsubscript{2} increased at 20 W but decreased at 50 W despite the reduction in shunt fraction. At that time, the alveolar oxygen tension decreased from 110.6 mm Hg to 102.6 mm Hg, and the mixed venous oxygen tension decreased from 40.0 mm Hg to 31.0 mm Hg. We consider that the decrease of the PaO\textsubscript{2} at higher work load resulted from these two facts. We cannot definitely explain the reason why the patient complained of mild dyspnea while asleep. The shunt fraction may have increased in the left-side-up recumbent position, but we regret not having measured it at such a position.

The results in our patient show the value of the study of shunt fraction and cardiopulmonary dynamics during exercise to clarify the mechanism of adaptation to exercise in a patient with a pulmonary AV fistula.

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Chronic Mountain Sickness at an Elevation of 2,000 Meters*

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A resident living at Lake Tahoe, Calif., at an elevation of 2,000 meters, had fatigue, edema, and erythrocythemia. Hematocrit was 63 percent, and arterial blood gas values revealed hypoxemia and respiratory acidosis. Results of pulmonary function tests, sleep study, and thyroid function all were normal. Erythrocytosis, cor pulmonale, and respiratory acidosis resolved after the patient moved to sea level. This patient suffered from chronic mountain sickness. Her symptoms resolved with relief of hypoxia.

Chronic mountain sickness (Monge’s disease) is commonly described in Leadville, Col., and Cero de Pasco, Peru.\textsuperscript{1,2} Lake Tahoe, Calif., with an altitude of only 2,000 m, has not previously been considered an outpatient of human adaptation to high altitude. I report a case of chronic mountain sickness in a 67-year-old woman living at Lake Tahoe. Her hypercapnia and erythrocytosis resolved when she moved to sea level.

CASE REPORT

A 67-year-old woman was admitted to Letterman Army Medical Center for evaluation of fatigue and erythrocytosis. The patient was a nonsmoker residing at Lake Tahoe, Calif., at an elevation of 2,000 m (6,500 ft). She had chronic medical problems of obesity, hypertension, and adult onset diabetes mellitus for which she took hydrochlorothiazide and insulin.

Six months prior to admission, she was seen elsewhere for gradual onset of fatigue, dyspnea on exertion, and a weight gain of 15.9 kg (35 lb). Two months later, she noted pedal edema for which she was treated with triamterene (Dyrenium) with modest improvement. One week prior to admission to Letterman, her blood pressure was 160/95 mm Hg, and her hematocrit reading was 63 percent. The chest was clear, and a cardiovascular examination result was normal. She had pitting edema to both knees. She was given furosemide (Lasix), 80 mg/day for four days, and referred to this hospital.

On admission to Letterman Army Medical Center, a physical examination revealed no change except resolution of the pedal edema. Chest x-ray films showed the heart size at the upper limit of normal. Complete blood cell count showed a hemoglobin of 19.1 g/dL, hematocrit of 60.2 percent, and normal indices, platelets, and leukocytes. Arterial blood gases showed a pH of 7.44, P\textsubscript{CO\textsubscript{2}} of 49 mm Hg, and P\textsubscript{O\textsubscript{2}} of 60 mm Hg on room air. The serum sodium level was 142 mEq/L, potassium, 5.4 mEq/L, chloride, 100 mEq/L, and CO\textsubscript{2}, 32 mEq/L. Serum creatinine and BUN levels were normal, and blood glucose level was 304 mg/dL. Thyroid function tests were normal, and thyroid-stimulating hormone was normal at 4 IU/mL. The ECG was normal.

Sleep study results were normal with no sleep apnea. Pulmonary function tests showed poor effort on spirometric study. The FEV\textsubscript{1} was 1.45 L (80 percent), FVC was 1.52 L (59 percent), and exhalation time was less than two seconds. Total lung capacity by the neon dilution single-breath method was 3.23 L (76 percent), and D\textsubscript{50} was 169 percent. Voluntary hyperventilation decreased the P\textsubscript{CO\textsubscript{2}} from 45.8 to 30.5 mm Hg. Echocardiography showed the right ventricle size to be at the upper limit of normal, and a multigated uptake scan

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