and RV in pulmonary function testing. The centrilobular type of emphysema tends to affect more selectively the apices of upper and lower lobes; however, this patient did not exhibit characteristic signs or symptoms of emphysema and consequently, the existence of true emphysematous cysts (usually extremely thin walled) in this instance could be considered unlikely. The so-called post infection cyst (the "healed" residuum of nonspecific lung abscess), occasionally will develop an air-fluid level, either from a new infection to which it is susceptible or as a result of accumulation of blood within it following a hemoptysis. When the cyst is small, then sometimes called an "air space," it may become totally filled with clotted blood and consequently appear as a solid circumscribed lesion.9

However, since our patient clearly exhibited bronchiectatic changes in several bronchopulmonary segments, it is reasonable to conclude that the cyst formation in the apex of the left lower lobe was a result of bronchiectasis per se rather than intraparenchymal abscess "healing" as alluded to above. It is of interest that the pathologist can differentiate bronchiectatic cyst from post-infection (post-abscess) cyst by virtue of the fact that a small cavity, relined by epithelium (i.e., the residual of a true abscess), will have multiple communications with adjacent bronchi.6

In conclusion, this case illustrates the necessity for, and value of detailed radiologic investigation of patients with hemoptysis and concomitant, not readily explainable pulmonary densities. The patient with a hemoptysis who exhibits a pulmonary nodular lesion must not be assumed to have bleeding secondary to a malignant process. As demonstrated in the present case, an infected bronchiectatic cyst has the potential to fill completely with inflammatory debris before undergoing drainage, and hence, to transiently simulate a coin lesion. It becomes important, therefore, to urge that such a bronchiectasis be included in the differential diagnosis of entities predisposing to roentgenographic pulmonary nodule formation.

ACKNOWLEDGMENT: We wish to extend our appreciation to Dr. William J. Grace, Chairman, Department of Medicine, St. Vincent's Hospital and Medical Center of New York, for carefully reviewing the manuscript. We are also grateful to all the members of the Department of Radiology who cooperated so fully in studying the patient.

REFERENCES

Type I Truncus Arteriosus in an Adult*


A 24-year-old woman is the oldest patient in whom a diagnosis of true truncus arteriosus was made during life and confirmed by cineangiography. The patient has also had the longest known survival with a type I truncus arteriosus. Clinical findings were of interest.

The diagnosis of true persistent truncus arteriosus during life in an adult has not been reported previously. The patient reported here has had the longest reported survival with a type I truncus arteriosus. The clinical findings of a long, harsh systolic murmur and the chest roentgenogram in this cyanotic patient were suggestive of the diagnosis. Of further interest was the "slightly split" second heart sound.

CASE REPORT

This 24-year-old white woman has been cyanotic since birth. She had pneumonia three times as a young child. Strenuous exertion during childhood resulted in syncope. Cardiac catheterization at another institution, in 1958, was stated to show a ventricular septal defect with a bidirectional shunt. Two months after being placed on birth control pills, in 1969, she had thrombophlebitis in her left leg. Three grandmal seizures in 1970 were considered due to cerebral thrombosis. At the present time she is active and has shortness of breath only after climbing two flights of stairs.

Physical Examination

The patient was 58 inches tall, weighed 101 pounds and had moderate cyanosis and clubbing. Blood pressure was 130/70. A prominent A wave was visible in the jugular pulse. There was a right ventricular lift and along the left sternal border, a prominent systolic ejection click. The second heart sound was loud and by auscultation narrowly split (not a pure single sound). The splitting appeared to be due to vibrations of the "aortic" second sound on the phonocardiogram (Fig 1). A harsh grade 4/6 holosystolic murmur was heard best at the fourth left intercostal space. There was also a short grade 1/6 diastolic decrescendo murmur along the left sternal border. Hemoglobin was 18 gm percent and hematocrit 56 percent.

On chest roentgenogram (Fig 2) the right and left pulmonary arteries were higher than usual and there was right ventricular enlargement. Electrocardiogram and vectorcardiogram showed right axis deviation and right ventricular hypertrophy.

Catheterization and Cineangiography

The systolic pressure in the right ventricle, pulmonary

*From the Department of Cardiology and the Clinical Investigation Center, U.S. Naval Hospital, San Diego, California. The opinions or assertions contained herein are those of the authors and are not to be construed as official or as reflecting the views of the Department of the Navy.

Reprint requests: Capt. Morgan, Naval Hospital, San Diego 92134

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artery (entered from the truncus) and aorta were all equal. Indicator dilution studies and oxygen saturation data were compatible with a bidirectional shunt at the high ventricular level and great vessel level. Injection of green dye into the proximal great vessel and sampling from the brachial artery showed an early dominant peak followed by a second hump five seconds later with a slow downslope. The brachial artery oxygen saturation was 85 percent.

Cineangiograms performed by injection of contrast material into the supraventricular great vessel (Fig 3) revealed a dilated single great vessel (truncus arteriosus); from the left side of the truncus there arose a main pulmonary artery which divided into normal appearing right and left pulmonary arteries. The truncus arteriosus continued into a normal left sided aortic arch. Slight regurgitation of contrast material from the truncus back into the ventricle was evident. A high ventricular septal defect was seen on ventricular cineangiograms.

**DISCUSSION**

In type I truncus arteriosus, a single arterial trunk from the heart gives rise to a main pulmonary artery and ascending aorta.\textsuperscript{1,2}

Truncus arteriosus is characterized by cyanosis (usually minimal) and dyspnea. Dyspnea is due to excessive blood flow to the lungs. Dyspnea is lessened after flow to the lungs is reduced by constriction of the pulmonary arteries; lack of prominent dyspnea during childhood implies that our patient had early development of pulmonary vascular disease.

In truncus arteriosus, there is usually a loud, harsh, blowing systolic murmur at the left sternal border due to the ventricular septal defect. As shown by the case presented here, the murmur in truncus arteriosus remains loud and long after development of pulmonary vascular obstructive changes. By contrast, the murmur is short and soft in a ventricular septal defect with Eisenmenger's complex.\textsuperscript{3}

**Figure 1.** Top: phonocardiogram at fourth left and second right intercostal spaces at 100 cps with carotid tracing and ECG. Bottom: phonocardiogram at fourth left and second left intercostal spaces at 100 cps with right ventricular apexcardiogram and ECG. Note the systolic ejection click, long systolic murmur beginning after the first sound and prolonged high-frequency vibration of the second heart sound.

**Figure 2.** Note high origin of the left pulmonary artery.
Type I Truncus Arteriosus in Adult

Figure 3. Cineangiogram from truncus. A (left)—posteroanterior view; B (right)—lateral view. Note the dilated single great vessel from which arises on the left a main pulmonary artery which divides into right and left main pulmonary arteries.

menger's syndrome. With balanced pressures there is minimal flow through the usual ventricular septal defect. With truncus arteriosus, however, both ventricles empty into the truncus; resistance does not change in the truncus with development of pulmonary vascular obstruction and marked flow continues across the ventricular septal defect to allow egress to the truncus which usually straddles one ventricle more than the other. The history of cyanosis since birth and the loud, harsh, long systolic murmur in our patient was not compatible with the previous diagnosis of a simple ventricular septal defect with balanced shunt.

In infants, there is often a mid-diastolic mitral murmur due to increased pulmonary blood flow, but after constriction of the pulmonary arteries this murmur is not present. There may also be a diastolic decrescendo murmur of truncus insufficiency.

A loud systolic ejection click is common in truncus arteriosus. The second heart sound is accentuated and with no pulmonary valve might be expected to be single. As noted in our patient, the second heart sound is not necessarily a pure, single heart sound, but can appear to be slightly split due to vibrations of the "aortic" second sound, as shown by phonocardiography. Perhaps the second sound will appear to be split more often in patients with four or more cusps.

The chest roentgenogram in truncus arteriosus usually reveals a large heart, about a 27 percent incidence of right aortic arch and, in the infant, increased pulmonary blood flow. Survival for a few years is usually due to development of pulmonary vascular obstruction and therefore, older patients will not have the markedly increased pulmonary vascularity found in infants. A clue to the diagnosis on chest roentgenogram is the high origin of the pulmonary arteries.

The electrocardiogram in infancy may show left ventricular hypertrophy and/or right ventricular hypertrophy, but in the older patient with pulmonary vascular obstruction only right ventricular hypertrophy would be expected.

Although several patients with type II and III truncus arteriosus have survived to the third decade or longer (with diagnosis by autopsy) the longest known previous survivor with type I truncus arteriosus was 13 years. Most patients with type I truncus arteriosus die in infancy; Fontana and Edwards could find only two patients with type I truncus arteriosus who survived more than a year. Early development of pulmonary vascular obstruction was the probable reason for long survival in our patient.

The best hope for increased salvage in truncus arteriosus is complete correction as advocated by McGoon and colleagues. Early banding of the pulmonary arteries in some patients may prevent irreversible pulmonary vascular disease. Our case points out that a rare patient will survive to adulthood without benefit of surgery.
Augmented Collateral Circulation in The systemic collateral circulation to the lungs is Continuous Murmur as a Sequel of Suppurative Lung Disease: Report of

Three adult men with bronchiectasis presented with continuous thrill and murmur over the chest and wide pulse pressure. One had visible collaterals in the chest wall and neck. These signs are the result of augmented pulmonary collateral circulation, demonstrated by aortography and pulmonary angiography. Such a clinical presentation has not been reported, in suppurative lung diseases. Reversal of blood flow in the main pulmonary artery seen in two patients is rare.

The systemic collateral circulation to the lungs is increased in suppurative lung diseases. Three patients with bronchiectasis, who presented with a continuous thrill and murmur and wide pulse pressure due to augmented collateral circulation, are reported. One patient had, in addition, visible collateral vessels in the chest wall and neck. We are not aware of similar case reports in the literature.

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Continuous Murmur as a Sequel of Augmented Collateral Circulation in Suppurative Lung Disease: Report of Three Cases*

Solomon Victor, M.S.; C. Lakshmikanthan, M.D., F.C.C.P.; Gowri Shankar, M.S.; P. G. Parameswaran, M.S., M.Ch.; A. Sreenivasan, M.D.; and C. S. Sadasivan, M.S.

Three adult men with bronchiectasis presented with continuous thrill and murmur over the chest wall and neck. These signs are the result of augmented pulmonary collateral circulation, demonstrated by aortography and pulmonary angiography. Such a clinical presentation has not been reported, in suppurative lung diseases. Reversal of blood flow in the main pulmonary artery seen in two patients is rare.

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CASE REPORTS

CASE 1

A 35-year-old man was hospitalized for cough with profuse, foul-smelling expectoration of two years' duration. He had had small bouts of hemoptysis for one year. He had clubbing of fingers and toes. Pulse was collapsing. There were pulsating tortuous vessels in the right supraclavicular region and right interscapular region. Mediastinum was shifted to the right. Medium rales were heard all over the right hemithorax. A continuous thrill and murmurs were found in the right supraclavicular and infraclavicular regions. Heart sounds were normal. Investigations revealed the following: blood pressure 140/80 mm Hg; sputum—negative for tubercle bacilli; *Neisseria cattarhalis* and nonhemolytic Strep tococcus were grown. Roentgenogram of the chest revealed irregular opacity over the right lung zones. Bronchogram showed cystic bronchiectasis in the right lung and left upper lobe. Aortogram showed branches from tortuous and dilated internal mammary, lateral thoracic, intercostal and other vessels, entering the right lung (Fig 1). The contrast medium, after opacifying the lung, filled the right pulmonary artery in a retrograde fashion and proceeded toward the left hilum. Pulmonary angiography showed that all the contrast entered the left pulmonary artery confirming reversal of blood flow in the patent right pulmonary artery (Fig 2). Surgery was not undertaken in view of bilateral bronchiectasis.

CASE 2

This 45-year-old man had cough with profuse foul-smelling expectoration and exertional dyspnea of five months' duration. He had had small bouts of hemoptysis, as well as clubbing of fingers and toes. Pulse was collapsing. Bronchial breath sounds and rales were heard over the right supraclavicular, infraclavicular and suprascapular regions. A continuous thrill and murmur were present in the right supraclavicular and infraclavicular regions. Investigations revealed blood pressure 128/60 mm Hg; sputum—negative for tu-