CLINICAL CONFERENCE
Editor: Edgar V. Allen, M.D.
Associate Editor: Raymond D. Pruitt, M.D.

Adult Fibroelastosis with Congenital Tricuspid Stenosis

Edited by Hans Popper, M.D., Daniel S. Kushner, M.D., and Benjamin Gasul, M.D.

This conference was presented at Cook County Hospital, Chicago, Illinois, on February 20, 1955, as one of the regular weekly clinical-pathologic exercises of the Hospital. The conferences are modeled after the Cabot prototype, the case being presented by the clinicians as an unknown for discussion. In order to preserve the spirit and spontaneity of the original conference, editing has been limited largely to making the material more suitable for publication, selection of pertinent photomicrographs for reproduction, and documentation of pathologic interpretations by appropriate references.

Clinical Data and Discussion

Dr. Benjamin Gasul: A 39-year-old American-born married white male clerk was admitted to Cook County Hospital in November 1954 with complaints of blueness, severe shortness of breath, marked weakness, abdominal distention, and incoherence for 1 day. He had been well until 1951, when he had the first episode leading to 4 admissions to another hospital, summarized as follows:

October 1951. The patient’s right foot suddenly became cold, blue, and pulseless. An embolectomy was performed but gangrene ensued and a supracondylar femoral amputation was done. Perhaps we should stop to analyze this very serious mode of onset of illness. This man, perfectly well for 36 years, suddenly developed a cold, blue, pulseless foot. This could mean arteriosclerosis with a local thrombus, a thromboangiitis obliterans, or a diabetic arteriosclerosis with gangrene; but we have no evidence for these. Deep venous phlebitis will not explain this cold, blue, pulseless foot.

What about the heart? We are given no information pertaining to the cardiac examination. What cardiac condition must be considered? The most common cause for embolization is a left-sided lesion such as rheumatic mitral valvular heart disease, which may be silent for 36 years. There could be a mural thrombus; there is no evidence of a coronary thrombosis nor of aortic valvular involvement. Therefore, the first thing to think of is a left-sided cardiac lesion, primarily mitral. Barring that, we have to consider a congenital noncyanotic type of cardiac malformation, the most common of which would be an atrial or ventricular septal defect. Even Ebstein’s malformation of the tricuspid valve is a possibility. Since 20 to 30 per cent have a patent foramen ovale, it could be associated with a paradoxical embolus. Amyloidosis and some form of fibroelastosis are also possibilities.

December 1951. The patient was readmitted with a femoral artery embolus, and a left lumbar sympathectomy was performed. He then did well with a prosthesis until August 1952, when he was readmitted for abdominal swelling of 3 months’ duration, ankle edema, orthopnea, and dyspnea, with improvement following treatment for congestive heart failure. And still nothing is said about the cardiac findings.

April 1953. The patient was readmitted with complaints of increasing edema, abdominal distention, severe dyspnea, orthopnea, nocturnal cough, and episodes of chills and sweats. Now, he had definite right and left heart failure.

From the Departments of Pathology, Medicine, and Pediatrics of the Cook County Hospital and Northwestern University Medical School, Chicago, Ill.
The chills and sweats are interesting but did they last a week or 2 or 3? If they lasted that long, the cause could be bacterial endocarditis or pericarditis. Was he treated? Did he get better because he had penicillin? Nothing is mentioned about this. Findings at that time included pulmonary rales and cardiac apex beat in the fifth intercostal space, 11 cm. from the sternum. If this is measured from the right sternal margin, it is normal; if from the mid-sternum, it is a little enlarged; and if from the left sternal border, it is definitely enlarged. A grade 3 systolic or grade 3 presystolic and a middiastolic murmur were heard at the apex. If he had a presystolic murmur at the apex or a middiastolic and grade 3 systolic, the chances are good that he had either mitral stenosis, with regurgitation on the basis of involvement of the mitral valve, or relative stenosis on the basis of enlargement of the left ventricle, or even a tricuspid lesion on the basis of right ventricular involvement.

Venous pressure at that time was 33 cm. of saline; circulation time was 19 sec. with ether and 30 sec. with Decholin, so both right and left sides of the heart were involved. Pericardiocentesis produced 150 ml. of brown turbid fluid, and abdominal paracentesis produced 3200 and 3500 ml. of ascitic fluid.

Roentgenograms of the chest showed slight increase of the transverse diameter of the heart, without characteristic configuration, and exaggeration of bronchovascular markings. Fluoroscopy showed “mitral stenosis with aortic enlargement; no calcification in the wall of the heart.”

**Dr. William Meszaros:** A postero-anterior view of the chest was taken in 1951, at the time of his first illness. The heart is normal in size and shape. The lungs are clear. The chest is negative at this time (fig. 1A).

The second radiographic examination was performed in April 1953. The transverse cardiac diameter now appears to be increased. Measurements are not strictly comparable because the diaphragm is 1 interspace higher. However, the gross appearance indicates an increase in size of the cardiac shadow. There is some straightening of the upper left cardiac border. The lung fields remain clear.

**Fig. 1. A.** Roentgenogram of chest, October 1951. **B.** Roentgenogram of chest, May 1953.

The third radiographic examination was performed in May 1953, 4 days before surgery (fig. 1B). A film taken with increased penetration fails to show calcification of the pericardium or heart valves. An interesting finding is the straight left heart border, which may be seen in constrictive pericarditis.

The last chest film was taken 10 days after surgery, in 1953. There is a large left pleural effusion. The heart is displaced to the right. There are no other significant findings.
DR. GASUL: The electrocardiogram on May 15, 1953, (fig. 2) shows slight depression of S-T in lead I, marked depression in lead II, diphasic T, and normal QRS. There is no evidence of right bundle-branch block as one would expect in an atrial septal defect. From the standard leads the electrocardiogram is consistent with myocardial anoxia or pericarditis. V₁ and V₂ reveal no evidence of right ventricular hypertrophy. There is marked depression of the S-T segment. The inversion of the T waves is consistent with myocardial anoxia or pericarditis. There are inverted T waves in aVR and an elevated S-T segment, which may be a sign of anoxia. The other findings here are still consistent with pericarditis or with myocardial anoxia.

In November 1954, the tracing shows low amplitude QRS, which goes with pericarditis or shock from any cause. Again there is anoxia. The T waves are very low. There is no evidence of right ventricular hypertrophy. AVL shows a Q and S, which could occur in a vertically placed heart. This might be a sign of clockwise rotation of the heart, which could follow pulmonary embolism with marked anoxia. But that is not too much help. Certainly no one is justified in diagnosing mitral stenosis from the electrocardiogram. Nor would I want to commit myself on interpretation of the x-ray films, which are consistent with pericarditis or with almost anything else but not with an uncomplicated atrial septal defect, because there is no evidence of definite increase in pulmonary blood flow. I will be surprised if this is an atrial septal defect or an intraventricular septal defect. The electrocardiograms are definitely against either of these conditions and merely indicate a pericarditis with anoxia. There is no evidence of coronary thrombosis.

The cardiac catheterization was very interesting because it revealed the following: normal pulmonary arterial pressure; right atrial pressure of 17 mm. Hg (20 cm. of water) with an "M"-shaped curve. Right ventricular pressure tracing showed diastolic dip and plateau. Cardiac index was 1.22 L./M.²/min. at rest. What does this mean? If the pulmonary artery pressure was normal, we can assume the right ventricular pressure was normal too, because there is no evidence of pulmonary stenosis here. If pulmonary pressure was normal and so was the right ventricular pressure, that would definitely speak against a diagnosis of mitral stenosis, even with right heart failure, because even in the presence of tricuspid regurgitation there should be some elevation of right ventricular pressure. If this were a rheumatic mitral
stenosis, I would assume there was also a tricuspid stenosis and not regurgitation. But here again, the diagnostic sign of tricuspid stenosis is a high diastolic pressure in the right atrium, even higher than in the right ventricle. We have no information on that. A right atrial pressure of 17 mm Hg with "M" curve and plateau is characteristic of constrictive pericarditis or amyloidosis or left heart failure. But once again, before one can be sure it is not heart failure, one must see how high the plateau is; and that we do not know. In constrictive pericarditis, as the right ventricle expels the blood, it cannot distend and, therefore, even though the first curve is high, it goes down and quickly goes up again, but how high does it go? They used the term "M-shaped," which means 2 positive plateaus in the atrium of about the same height that do not go down to the base line because the atrium cannot empty itself. This could be a good sign of constrictive pericarditis or amyloidosis or very unusual fibroelastosis, which occurs even in adults and involves both sides of the heart. What about Ebstein's disease? This is a possibility. There appears to be some involvement of the tricuspid valve.

Cardiac operation was performed on May 16, 1953. We could speculate that the preoperative diagnosis was either mitral stenosis or constrictive pericarditis. They would not have operated for Ebstein's disease or amyloidosis. Venous pressure preoperatively was 33 cm. of saline; postoperatively, 27 cm. of saline. There was only slight improvement postoperatively; the patient required monthly abdominal paracentesis, weekly mercurial injections, and he was maintained on a low-salt diet, digitalis, and ammonium chloride. He was forced to stop work in November 1953, but was able to remain ambulatory at home. Since there was no improvement following operation, this probably was neither mitral stenosis nor constrictive pericarditis.

On the day prior to admission to Cook County Hospital, the patient noted sudden onset of increased abdominal distention, severe dyspnea, and marked weakness. The amputation stump became very red. On the day of admission he was noted to be very blue and became incoherent. Abdominal paracentesis was performed, and the patient was referred for hospitalization.

Systemic review revealed only nocturia, several times, for 2 years, but on the day prior to admission the patient seemed to pass no urine.

Past history revealed only appendectomy. Venereal disease was denied. There was no known rheumatic fever or chorea in childhood.

Family History. One older brother had died of a heart attack and 1 of cancer of the liver.

Social History. The patient drank only socially and smoked 10 to 20 cigarettes a day.

Physical examination revealed a well developed, moderately well nourished, apprehensive, young white man with labored respirations and marked cyanosis. His blood pressure was 82/58 mm. Hg, pulse 112 and regular, respirations 32/min. and temperature 101 F. His head, eyes, fundi, ears, nose, mouth, and pharynx were negative, apart from cyanosis. There were no petechiae. Neck veins were slightly distended. Dullness and decreased breath sounds were noted over the right lung base posteriorly; coarse rales were heard at the left posterior lung base. A well healed left thoracotomy scar was present. The cardiac apex beat was palpable in the left fifth intercostal space in the midelavicular line, which means it was not much enlarged. No thrills were felt, and no murmurs were heard. What happened to those murmurs? In the state of shock one may not hear murmurs.

A firm liver edge was palpable 3 fingerbreadths below the costal margin. The spleen was not felt. The abdomen was soft, and dullness was noted in both flanks. The paracentesis site was draining serous fluid. The femoral pulse was palpable on the left but not on the right; therefore, the thrombosis was high. It involved not only the right common femoral but the right common iliac. The right amputation stump was markedly erythematous, warm, and tender. The nailbeds were deeply cyanotic. There was a questionable left Babinski sign, but superficial neurologic examination was otherwise negative.

Laboratory data. Urinalysis: Specific gravity 1.015; acid; albumin 4 plus; no sugar, acetone;
sediment negative. Hemogram: Hemoglobin 88 per cent; leukocytes 21,250; platelets adequate; polymorphonuclear cells 40, band forms 53, lymphocytes 3, monocytes 4; marked toxicity; Doehle bodies; rouleaux and burl cells. Blood culture revealed no growth.

Electrocardiogram showed moderately low voltage and isoelectric to slightly inverted T waves in standard and limb leads.

**Hospital Course.** The patient was treated with morphine, mercurhydram, oxygen, penicillin (2.4 million units/day), streptomycin (1 Gm./day), digitalis, ammonium chloride, low-salt diet, and norepinephrine. Shock persisted although cyanosis subsided and sensorium cleared. On the second day, 350 ml. of concentrated urine was obtained by catheterization. On the third day, blood pressure remained at levels of 60-70/42-44, despite increasing amounts of norepinephrine. Urinary output was noted to be less than 50 ml./24 hours. Temperature remained elevated to 101 F. Increasing pulmonary rales and abdominal distention were noted. The patient was given 100 mg. of cortisone intramuscularly. His general condition remained poor, and he died 53 hours after admission, following a total duration of illness of 3 years.

There was nothing very unusual in the hospital course. The patient had a very low pressure and was in shock. Why? Because he developed pulmonary infarcts or embolism. Perhaps he even had a large thrombus in the atria, a ball-valve type of thrombus.

If this man did not have congenital heart disease, I would say primary amyloidosis would explain everything: intractable heart failure, cardiac murmurs, and the findings at catheterization. The only point that bothers me is, could the embolus be the first manifestation of amyloidosis? Possibly. The second diagnosis I would think of is constrictive pericarditis.

**Dr. Hans Papper:** Can we show you the operative findings now?

**Dr. Gasul:** Yes. The report informs us that the lungs appeared normal; there was no pleural effusion; 200 ml. of clear fluid was removed. The heart was rotated and the right ventricle was very muscular. Systolic contractions were normal. Systolic thrills were present over the pulmonary artery and the posterior surface of the heart. The pulmonary pressure was very low, but that can be very misleading because there does not have to be a stenosis. The pulmonary artery pressure was normal previously, so that perhaps he had a pulmonary stenosis with embolism from a congenital heart; but there is no evidence of that on the electrocardiogram or on the x-ray films or from the heart sounds. I would expect the pulmonary second sound to be absent or diminished. The left atrium was enlarged but neither atrium appeared to be under increased pressure. There was no evidence of mitral stenosis. The right ventricle appeared abnormally formed. There was a long pulmonary infundibular stenosis. The pulmonary valve could not be reached because of thickening of the ventricular wall. By means of force the infundibular stenosis was incised.

Now that changes things but the information is very misleading. I cannot see how anyone can have infundibular stenosis without high pressure in the right ventricle, and with an electrocardiogram of this kind, unless there are associated lesions decreasing the pressure in the right ventricle. Ebstein's disease is still a possibility; it could have gone on for 36 years, with paradoxical embolism having occurred through a patent foramen ovale. I believe that there are definite evidences of involvement of the tricuspid valve by a downward displacement and malformation of the valve.

**Dr. A. B. Rimmerman:** All the way through you have spoken of amyloidosis. Was that in anticipation? You have no evidence for it.

**Dr. Gasul:** Intractable heart failure and the findings at catheterization suggest amyloidosis. The only thing that disturbs me is the embolus as the first manifestation.

**Dr. L. Feldman:** I wish I had not heard the operative report. I thought at first that there could be a patent foramen ovale without right heart strain, but it would be unlikely to have a paradoxical embolus in such a condition. However, he could have had Cocari's disease, which is a polyserositis, and also a paradoxical embolus occurring during a transient increase in right atrial pressure.

**Dr. Jacob W. Fischer:** I think the electro-
cardiogram is suggestive of right ventricular hypertrophy. The T waves in leads II and III are inverted and asymmetric. The S-T sectors in these leads are depressed. While this pattern is not pathognomonic of right ventricular hypertrophy, it is, however, suggestive. Of course, if the patient was taking digitalis, the S-T and T changes may in part be attributed to this drug.

Dr. Gasul: I also thought of Concato's disease but that term does not mean anything. What is Concato's disease? It is not a disease. It is a polyserositis that can be due to a number of things. I considered paradoxical embolus but there has to be a cause for the increased pressure in the right atrium. I do not believe that there is any evidence of either atrial or ventricular septal defects. A congenital involvement of the tricuspid valve appears very probable.

Pathologic Observations

Dr. Popper: At necropsy the body was edematous. The iliac artery was occluded by a thrombus extending to the amputation stump. The arterial wall showed moderate atherosclerosis similar to that in the aorta. The kidney was severely congested as evidenced by proteinuria and hyperemia of the vessels at the corticomedullary junction. The gastrointestinal tract also was congested. The veins in the gastric cardia and in the esophagus showed varicoceles dilatation. The scrosa of the gastrointestinal tract was diffusely thickened, as a result of chronic fibroplastic inflammation, in keeping with ascites (500 ml.). Considerable ascitic fluid was also found at necropsy. The spleen was very large (630 Gm.). Its thick capsule was hyalinized. Histologically, there was evidence of portal hypertension and recent reactive hyperplasia.

A section of the liver biopsy specimen, obtained at operation 1½ years before death, showed moderate passive congestion, the architecture being rather well preserved. The liver at necropsy was not significantly enlarged (1600 Gm.). Its capsule also was very thick, gray-white, and hyalinized, resembling a sugar-icing, as is seen in chronic ascites. The lobular architecture on the cut surface was obscured by a severe chronic passive congestion. The wall of the central and sublobular veins was very thick, due to fibrosis, and newly formed elastic membranes were noted, all caused by increased venous pressure. The central lobular zones showed either severe congestion and necrosis or fibrosis, and connective tissue septa extended bridgelike from one central field to the next, reversing the lobular architecture. These are the changes referred to as cardiac fibrosis of the liver. In many lobules, however, the septa extended from the central to the portal canals. The lobular architecture was destroyed and regenerative nodules formed; the picture of a true cirrhosis was thus produced. This is one of the rare specimens of true cardiac cirrhosis that we have observed. It develops in only 2 instances: chronic pericarditis and tricuspid incompetence.

There was right pleural effusion (800 ml.) and obliterating left pleural adhesions. Acute pulmonary congestion was not accompanied by evidence of prolonged pulmonary hypertension. Emboli were present in the right pulmonary artery.

The heart weighed 400 Gm. There were extensive, but loose, pericardial adhesions. The right atrium was very large and dilated. Its septum was intact and the foramen ovale closed. Fairly recent thrombi in organization completely filled the right atrium. The tricuspid valve appeared stenosed, measured 9 cm. in diameter and failed to admit 2 fingers (fig. 3A). When the value was opened, the cusps, however, were not significantly altered, and the chordae tendineae were delicate (fig. 3B). No muscular portion of the right ventricle was noted above the tricuspid stenosis. This excludes Ebstein’s malformation, which consists of a pulling down or falling down of the tricuspid valve and a division of the right ventricular cavity rather than a separation of right atrium from right ventricle.1 The tricuspid ring was thickened and scarred (fig. 3B). The stenosis was thus produced by changes in the tricuspid valve ring. Histologically, the connective tissue of the valve ring was a loose, in places almost an embryonal connective tissue, indicating the congenital character of the tricuspid stenosis. An elastic layer separated
Fig. 3. A. Dilated right atrium with closed foramen ovale (F), organized thrombi in right appendix (T), and fishmouth stenosis of tricuspid orifice (O). B. Tricuspid orifice exposed by incision of the markedly fibrosed ring. The cusps and chordae tendineae are normal. C. Section through fibrosed myocardium of atrophic inflow tract of right ventricle, with markedly thickened endocardium, above, and pericardial fibrous adhesions, below. (3X, Mallory aniline blue stain.) D. Border between endocardium and myocardium in atrophic inflow tract of right ventricle, indicated by reduplicated elastic membrane. Sparser elastic membranes and vessels in thickened endocardium. (150X, orcein stain.)
Fig. 4. A. Atrophic inflow tract of right ventricle forms a small appendix (between arrows I₁ and I₂) with thickened endocardium and fibrosed myocardium. Tricuspid ostium leads directly into widened pulmonary conus (C). The supraventricular crista (below arrow C) lies between conus and ostium. The pulmonary valve is normal. B. Periartrial fibrosis and cellular infiltration in myocardium of left ventricle (fibrosing Aschoff body). (135X, hematoxylin and eosin stain.) C. Subacute bacterial endocarditis with fibrinoid degeneration and leukocytic infiltration of the valve. (135X, periodic acid-Schiff reaction.) D. Mild, rheumatic fibroplastic deformity of mitral valve, and vegetative endocarditis of posterior leaflet of mitral valve. Mural thrombi at apex.
this connective tissue from the myocardium and seems to represent the original endocardium (fig. 3D). Moreover, in the connective tissue, elastic membranes arranged like a collapsed sac suggested a fetal endothelial-lined canal, possibly representing a rudimentary chamber. It appears, thus, that in this instance the endothelial cushion that normally separates right atrium and ventricle and gives rise to the tricuspid valve is disorganized. This results in excessive connective tissue with irregular formation of elastic membranes, narrowing of the ring, and tricuspid stenosis. The lesion may be included in the group of fibroelastosis. The tricuspid ostium led into a small blind sac, measuring 1 by 4 cm. and lined by thickened endocardium, surrounded by a very thick, fibrosed muscular wall (fig. 3C). This blind sac represents the hypoplastic inflow tract, or sinus, of the right ventricle. Apparently this area was incised during operation, and this may have contributed to the fibrosis. However, the endocardium here also showed excessive numbers of elastic membranes that doubtless are independent of the operation. The wall of the right ventricle showed diffuse myocardial fibrosis (fig. 3C). The tip of the blind sac contained mural thrombi in organization.

The pathway of egress of blood from the right ventricle remained a problem. The clinical assumption was made that an infundibular stenosis was present. Therefore, the pulmonary artery was opened; it appeared normal, as were the pulmonary cusps. The infundibular portion of the right ventricle, over which a metal clip indicated a surgical incision, was surprisingly dilated rather than stenosed. This infundibular area had a normal smooth wall. It extended to the supraventricular crista, which normally separates the smooth outflow tract from the inflow tract normally lined by trabeculae carneae. In this instance, the crista was close to the tricuspid ostium and except for the blind sac, hardly any inflow tract existed (fig. 4A). We deal, therefore, with a hypoplasia of the right inflow tract, with fibroelastosis. The blood flowed through the stenotic tricuspid valve directly into the infundibular portion. This is compatible with the physical findings as well as the catheterization data.

The left atrium was markedly dilated. The left ventricle was hypertrophic and dilated. Its wall was fibrosed, and the thickened endocardium of the apex was covered by organizing mural thrombi. Microscopic examination of the myocardium revealed diffuse myofibrosis in addition to periarterial scars derived from fibrosing Aschoff nodules (fig. 4B). The rheumatic disease of the left ventricle was confirmed by valvular changes. In contrast to the changes at the tricuspid ostium, where the valve was free but the ring altered, the valve cusps themselves were the seat of disease. Thick irregular adhesions deformed the commissures of the aortic valve. Histologically, the cusps appeared fibrosed and vascularized. This fibroblastic deformity was probably not marked enough to produce murmurs or circulatory disturbance. The chordae tendineae of the mitral valve were thickened and matted together. The mitral valve leaflets were scarred on the free edge at the line of closure, producing a rheumatic mitral stenosis. On the atrial surface of the posterior leaflet there was an irregular ulcerated area more than 1 cm. in diameter (fig. 4D). It was in part covered by irregular vegetations, some of which were calcified. Histologically, fibrinoid degeneration of the valvular tissue produced irregular vegetations, and infiltration by histocytes, lymphocytes, and especially segmented leukocytes, confirmed the diagnosis of a subacute bacterial endocarditis (fig. 4C).

**Clinical Pathologic Correlation**

*Dr. Popper:* This patient had a congenital tricuspid stenosis caused by a disorganization of the cushion separating atrium and ventricle. In addition, he had a hypoplasia and deformity of the pulmonary sinus. Both are an expression of fibroelastosis, which is now considered to be a disturbance of the organization of the endocardial lining in prenatal life. This congenital lesion accounted for the demonstrated high right atrial pressure associated with low pressure in the right ventricle and pulmonary artery but probably caused little functional impairment. A rheumatic lesion in the left heart with mild fibroblastic deformity of aortic and mitral valve and myo-
fibrosis led to mural thrombi in the left ventricle and embolism into the right leg, the first clinical sign, 3 years before death. Now the minimal functional disturbance attributable to the congenital lesion in the right heart was aggravated by the rheumatic lesion. Cyanosis and dyspnea appeared, the venous pressure and the circulation time increased, and edema and ascites developed. The murmurs heard may have been caused by both right and left-sided valvular lesions. The operation 1½ years before death did not improve the condition but possibly increased the cardiac fibrosis, especially of the right ventricular wall, and caused pericardial adhesions. Both led to increased hepatic congestion and, as a comparison with the liver biopsy demonstrated, only now true cardiac cirrhosis developed with the characteristic hyaline perihepatitis and perisplenitis. Terminally, a subacute bacterial endocarditis developed in the mitral valve that was damaged by the preceding rheumatic valvulitis. It was reflected in the leukocytosis, fever, and splenomegaly, and was the ultimate cause of death.

**Final Pathologic Diagnoses**

Fibroelastosis with congenital tricuspid stenosis; hypoplasia of right ventricular inflow tract; myocardial fibrosis and mural thrombosis; rheumatic myocarditis; aortic and mitral valvulitis with mitral stenosis; chronic passive congestion of viscera; cardiac cirrhosis; and terminal subacute bacterial endocarditis.

**REFERENCES**


Thus history teaches us that any division of the science and the art of medicine is necessarily harmful to practice. The physician of today, better realizing the limitations of bacteriological and other technical aids, is experiencing the need of returning to the patient's bedside, from which medicine should never have separated itself.—Arturo Castiglioni, 1874.
Adult Fibroelastosis with Congenital Tricuspid Stenosis
EDGAR V. ALLEN, RAYMOND D. PRUITT, HANS POPPER, DANIEL S.
KUSHNER and BENJAMIN GASUL

Circulation. 1956;14:412-421
doi: 10.1161/01.CIR.14.3.412
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1956 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/14/3/412.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/