TRICUSPID STENOSIS, WITH REPORT OF A CURE.

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Among the hundred and eighty-seven cases of tricuspid stenosis found in the literature in but ten cases was a correct diagnosis made clinically. In view of this fact it seems worth while to record such a case in which the clinical diagnosis was confirmed at autopsy.

Patient.—A housewife, aged 35.

History.—The patient's father died of heart disease. The patient had measles, mumps, pertussis, scarlatina, diphtheria twice, and smallpox, all in childhood; at 10 and 27 acute articular rheumatism, involving knees, ankles and wrists.

Onset of Present Illness.—The patient was first admitted to St. Luke's Hospital, April 20, 1905, giving history of shortness of breath, palpitation and observed irregularity of heart during the past four years. Three years before admission the abdomen began to swell and was first tapped one year later. Tapping was repeated four months, and again six weeks before entering the hospital. The only complaint throughout this period was shortness of breath "when the abdomen was swollen."

First Physical Examination (April, 1905).—Heart: Apex in fifth space, $4\frac{1}{2}$ inches to the left of mid-sternum. Right border $2\frac{1}{2}$ inches from mid-sternum in third space. Action regular. First apical sound loud; second pulmonary, not accentuated. Second aortic inaudible. At mitral area, and, transmitted to the left, a blowing systolic murmur. At aortic area a pronounced systolic thrill and a double murmur, a systolic, loud and harsh in quality, transmitted upward and heard in vessels of neck, also over greater part of anterior chest, and, posteriorly, between scapulæ; a blowing diastolic transmitted down left border of sternum.

Pulse: Small, regular, moderate force. Veins of neck distended; pulsation not remarked. Systolic arterial pressure 120 mm.

Chest: Small fluid accumulation at both bases.

Abdomen: Enormously distended, signs being those of large amount of fluid.

Extremities: Edematous, indurated, pigmented; superficial veins dilated.

Following aspiration of 592 ounces of fluid from peritoneal cavity, friction fremitus was noted over liver, edge of which was palpable three inches below costal border.

Remission.—Improvement was rapid; the patient left the hospital and was not heard from until over two years later. In August, 1907, she was readmitted complaining of edema, dyspnea and weakness.

Second Physical Examination (August, 1907).—Heart: Apex in fifth space, $4\frac{1}{2}$ inches to left of mid-sternum. Action irregular, intermittent, poor force. At apex a rumbling presystolic murmur and blowing systolic murmur, a loud first sound. At aortic area a double murmur, a systolic of rough, intense quality transmitted upward, heard in vessels of neck and a diastolic referred down sternum and also heard in second and third left interspaces. In fourth and fifth spaces to right of sternum a soft, blowing systolic of very different quality from

apical murmur of same phase, and a snapping, valvular second sound. Right border 2 inches from mid-sternum in third interspace. Epigastric pulsation marked. Veins of neck widely distended, but without visible pulsation.

Pulse: Irregular, small, of poor force; tension 105 mm.

Moderate effusion in right pleural space.

Abdomen: Circumference 62 inches, symmetrical, tense, showing distinct signs of fluid.

Extremities: Markedly edematous, excoriated, ulcerating.

Clinical Course.—Rest in bed, abdominal paracentesis, digitalis and morphin, as the only therapeutic measures, enabled the patient to sit in a chair after twelve days and she was soon walking about the ward. In the interval from August 26 to November 16, the abdomen was tapped eleven times and a total of 3,665 ounces of fluid removed. There was constant diffuse cyanosis of the entire body, but slight edema of the extremities after the first week, and little or no dyspnea, except when the abdomen became greatly distended with fluid. Leaving the hospital November, 1907, the patient continued in comfort until March, 1908, when ascites compelled her return. The cardiac signs were the same as previously, except that a diastolic murmur of quality thought to be different from that heard at the aortic area was noted at the ensiform.

Diagnosis.—Aortic stenosis and insufficiency with mitral insufficiency had been the most frequent diagnosis during the patient's stay in the hospital. The presence of a tricuspid stenosis suggested itself to me in 1907, and was affirmed positively and independently by Dr. Van Horne Norrie, on whose service the patient came to autopsy, and by whose courtesy I am able to publish this report.

Autopsy.—The patient died April 30, 1908. The autopsy, by Dr. Zinsser, showed:

Heart: Apex in seventh space, $4\frac{1}{2}$ inches to left of mid-sternum. Right border $\frac{3}{4}$ inch to right of right sternal margin. Pericardial sac totally obliterated by very firm adhesive pericarditis. Right auricle and ventricle much dilated, walls flabby and thin. Tricuspid valve segments much thickened, adherent at angles, forming a firm ring about auriculoventricular opening, which admitted with difficulty tips of two fingers, and forming distinct stenosis. Pulmonary valves normal. Left ventricle greatly hypertrophied showing interstitial myocarditis. Aortic valve adherent; marked thickening of valve segments with resultant button hole stenosis. Mitral valve with funnel stenosis admitting one finger. No fresh vegetations on endocardium.

Left pleural cavity: Large amount of fluid. Left lung thickened, atelectatic, compressed to one-fifth normal volume.

Right lung: With adhesive pleurisy of long standing.

Abdomen: Peritoneum universally thickened, in areas showing fibrinous exudate.

Liver: Weight, 2,890 gm., marked perihepatitis (Zuckergussleber), chronic congestion, cirrhosis of pronounced degree.

Spleen: Very large, marked perisplenitis.

Kidneys: Large, soft, congested.

To become convinced that tricuspid stenosis is not a rare lesion one has to review the observations of the many who have described the condition since Duroziez, in 1868, first brought it convincingly to notice. The subjective and objective evidences of the condition have been in the course of time formulated until a fairly definite clinical picture may be drawn. This has been well done by J. B. Herrick.

The case reported well illustrates the important features of the condition: A woman having a history of acute articular rheumatism or chorea, prominent among whose symptoms are persistent cyanosis of mucous membranes and skin, marked tendency to edema or general anasarca and with relatively little dyspnea; the signs of a mitral stenosis without accentuation of the second pulmonic sound, often, in addition, an aortic lesion, an enlarged right ventricle and auricle, distended veins of the neck, frequently without pulsation. These comprise the most constant diagnostic factors. To them may be added Mackenzie's observation that seven of these cases proved at autopsy showed a liver pulse of the auricular type. The murmur is very inconstant and more often absent than present. In fact, very few of the cases carefully observed and recorded have shown the presystolic murmur to the right of the sternum so insisted on by many. As a positive sign such a murmur is confirmatory, but negatively has no weight in the diagnosis. All auscultatory signs are so often obscured by the presence of lesion of the mitral and perhaps aortic valves that very great weight can not safely be placed on them.

The remarkable comfort of these patients in the presence of cyanosis and, frequently, edema or general anasarca, has been very often noted. This is doubtless due to the effect of the narrowed right auriculoventricular orifice lessening the engorgement of the pulmonary circulation and the consequent dyspnea. This same factor, by its tendency to equalize pressure on either side of the pulmonary valve, furnishes a satisfactory explanation for the frequent absence of accentuation of the second pulmonic sound in the presence of the almost universally accompanying stenosis of the mitral valve.

In Steele's collection of reports of eighty-six cases a chronic obliterating pericarditis was present in seven. Broadbent, Kassen Beck, Cochs and Chadbourne also record one each. These, together with the case here reported, make a total of twelve showing this condition.

Duroziez in 1868, Leudet in 1888 and J. B. Herrick in 1897 have summarized the cases of tricuspid stenosis, the last-named tabulating one hundred and fifty-four. In reviewing subsequent literature I have found reports, complete or incomplete, of thirty-three additional cases. These include eight collected by Satterthwaite in 1902.

SUMMARY OF REPORTED CASES TO DATE.

Sex.—		Age.—	
Male	38	10 to 20 years	16
Female	133	20 to 30 years	59
Sex not known	16	30 to 40 years	38
		40 to 50 years	28
		50 to 60 years	10
Previous History.—		60 to 70 years	6
Rheumatism	61	Not known	30
Doubtful rheumatism or chorea	11		
No rheumatism	33		
Not known	82	Total cases	187
Association of Valvular Lesions.—			
Tricuspid alone			14
Tricuspid and mitral			102
Tricuspid and aortic			64
Tricuspid and aortic and pulmonary			1
Tricuspid and endocardium of left auricle			1
Tricuspid mitral and pulmonary			2
Total cases			187
Cases showing adherent pericardium			12

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