

REPORT OF A CASE OF TRICUSPID STENOSIS ASSOCIATED  
WITH MITRAL STENOSIS AND AORTIC STENOSIS.

By THOMAS G. ASHTON, M.D.,

CHIEF OF THE OUT-PATIENT MEDICAL DEPARTMENT OF THE JEFFERSON MEDICAL COLLEGE  
HOSPITAL, DEMONSTRATOR OF CLINICAL MEDICINE AT THE JEFFERSON MEDICAL COLLEGE  
AND VISITING PHYSICIAN TO ST. MARY'S HOSPITAL, PHILADELPHIA,

AND

ALONZO H. STEWART, M.D.,

INSTRUCTOR OF CLINICAL MICROSCOPY AT THE JEFFERSON MEDICAL COLLEGE AND CLINICAL  
ASSISTANT AT THE OUT-PATIENT MEDICAL DEPARTMENT OF THE JEFFERSON MEDICAL  
COLLEGE HOSPITAL.

THE occurrence of tricuspid stenosis, while not so very rare, is yet sufficiently uncommon to lead us to believe that a report of the following case, in view of its many interesting features, will not prove un instructive.

The history is as follows:

W. N., aged nineteen years, a joiner by occupation. Both parents are living and in good health, and in the family of neither is there a history of rheumatism.

The patient appeared to be healthy at birth. When six years of age he had measles, his childhood being otherwise uneventful until his fifteenth year, when he suffered from an attack of influenza. With the latter affection he was ill two weeks, but, while unable to attend to his usual work during that period, was not confined to bed. The chief symptoms of the influenza were referred to the head, bone pains being of very moderate degree. There is no personal history of scarlet fever, nor had the patient at any time suffered from what might be termed a frank attack of rheumatism, although at various times vague pains, somewhat rheumatic in character, had been complained of.

For the six years preceding his death the patient's occupation had been that of a joiner, and therefore one that entailed no very laborious exercise. Throughout his entire childhood he had lived a very quiet and retired life, and never indulged in the active pursuits of his playmates, although there never appeared to be, as his mother stated, any physical incapability. When first he accepted employment, however, in the year 1888, it was remarked that he could not walk to and from the factory, a distance of some thirteen or fourteen blocks, without suffering great fatigue. No especial cardiac symptoms showed themselves, however, until about four years preceding his death when, after a hard day's work, he would frequently complain of precordial distress and cardiac palpitation associated with easily induced shortness of breath. Upon such occasions the violence of the cardiac impulse had been noticed, and it was of sufficient strength to make itself evident through the several layers of the patient's clothing. At that period, however, his color was good, and there existed no further evidence of ruptured compensation.

The patient first came under observation in the early part of March, 1894, at which time he presented himself for examination as an appli-

cant for life insurance. So far as rational symptoms were concerned, he then appeared to be in the heat of health, though the physical examination at once revealed the diseased condition of the heart, the diagnosis at that time being mitral stenosis and mitral incompetency. As such his case is included in a report of a series of cases of chronic valvular disease of the heart read before the College of Physicians of Philadelphia, by Dr. T. G. Ashton, at its meeting held June 6, 1894, and subsequently published in *The Medical News*.

Toward the end of March, 1894, the patient began to show signs of cardiac failure. Upon exertion, especially upon bending over, his face, which usually had a good color, would become decidedly blue, and even the slightest exertion was sufficient to induce shortness of breath.

The patient's habits had always been abstemious; he had never used alcohol or tobacco and, as previously stated, had never taken any interest in athletic exercises.

On June 23, 1894, he first made his appearance at the clinic of the out-patient medical department at the Jefferson Medical College Hospital. The notes of the physical examination made at that time are as follows: Marked cyanosis of the patient's lips, cheeks and hands; dyspnoea evident; puffiness of under eyelids and marked pretibial oedema; the radial pulses were imperceptible, and it was impossible, therefore, to obtain a sphygmographic tracing.

Inspection of the præcordium revealed a greatly extended impulse which was plainly perceived as far as the sixth interspace and one and one-half inches to the left of the nipple; there was marked pulsation in the epigastric region, which also extended well to the right of the sternum.

Upon palpation the cardiac impulse and apex beat were found to possess but little force. A feeble thrill was detected above the apex beat of the heart, the time of occurrence of which was presystolic; no thrill existed over the tricuspid area.

Auscultation at the apex revealed a systolic murmur, soft and of moderate intensity, which was plainly propagated into the left axilla. Above the apex, and at a point corresponding to the thrill above mentioned, was heard a murmur, presystolic in time, harsh in character, and terminating abruptly with what appeared to be the first sound of the heart. At a point below this, upon a level with the apex of the heart but about one and a half inches to the right, it was possible to hear both the systolic and the presystolic murmurs. To the right of the sternum, toward its lower end, was heard a murmur of systolic time and differing in its characteristics from that heard at the apex. There existed no visible pulsation in the veins of the neck, nor was it possible to detect hepatic pulsation.

At the aortic cartilage no murmur was heard save that supposed to have been transmitted from the apex, which, as the great vessels of the neck were approached, disappeared altogether. The pulmonary sounds were clear.

The clinical diagnosis was combined mitral stenosis and incompetency associated with tricuspid incompetency, the latter supposed to have been a relative defect depending upon primary mitral disease.

The specific gravity of the urine was 1017; its reaction acid; it contained no sugar; albumin, however, existed in large amount, but no casts were found.

The patient's condition became so serious that he was unable to return

to the hospital, and subsequent treatment was conducted at his home; nevertheless, his symptoms became progressively worse. Toward the end anasarca of the lower extremities became marked, and effusions took place into the abdominal, pleural, and pericardial cavities, while throughout both cyanosis and dyspnea were extreme.

On July 28, 1894, the patient died.

The autopsy notes read as follows: The body that of a young man, about nineteen years of age. Post-mortem rigidity marked; slight discoloration of the dependant portions of the body; general anasarca, most marked in the lower extremities; abdomen distended; the skin generally extremely white and glistening, due to distention.

*Thorax:* Upon opening the thoracic cavity the lungs were hidden from view by a greatly distended pericardium. Both pleural cavities contained clear serous fluid; that of the left side three pints, that of the right side about two quarts.

FIG. 1.



Showing narrowing of the tricuspid orifice and intimate adhesion of the three cusps of the valve. Observed from the auricular aspect.

The lungs were partially collapsed and lying in the upper part of the thoracic cavity along the spinal column. The left lung showed considerable edema and some hypostatic congestion at its base. The right lung was deeply congested, and at its base was a moderate sized area of consolidation, a piece taken from which sinking when placed in water. Neither lung showed any evidence of a recent or old tuberculous process.

The pericardium, upon being opened, was found to be filled with a blood-stained serum, the measured quantity being fifteen ounces. In several places the parietal layer appeared to be adherent to the right auricle.

The heart's right chambers were found to be distended with soft blood-clots, the left side being completely empty. The apex of the right ventricle extended about one inch below that of the left ventricle. Upon the external surface of the right auricle could be seen four small openings, each about 5 mm. in diameter, two being at the upper portion and two near the right auriculo-ventricular boundary. Corresponding points were noted upon the parietal pericardium, denoting that the latter at these places had been adherent to the auricular pericardium.

Upon opening the right auricle it was found to be filled with dark, soft clots; its cavity was much enlarged and its walls thickened. The auriculo-ventricular orifice was much narrowed and would permit the passage of the middle finger only with some difficulty. As observed

FIG. 2.



Showing narrowing of the mitral orifice; the light shading about the orifice and upon the auricular wall denoting calcareous plates. Observed from the auricular aspect.

from the auricular aspect the three cusps of the valve were found to be firmly adherent to each other and very much thickened, resulting in a stenosis of the tricuspid orifice of the button-hole variety. The valve, though thickened, was smooth and free from any calcareous deposits. The cavity of the right ventricle was much enlarged and its walls increased to about 1 cm. in thickness. The pulmonary valves were normal.

The left auricle contained a small quantity of fluid blood, but was not distended. Through the auricle the mitral opening was found to consist of a mere slit, the edges of which were in firm and close approximation, so that even the tip of the little finger could not be inserted into the opening. The valvular ring upon its auricular surface was rough and hard, having many sharp calcareous projections which extended in several places upon the inner surface of the auricular wall. The slit

constituting the mitral opening, being so short and the edges so firmly approximated, could have permitted but a very small quantity of blood to pass through.

The left ventricle was found completely empty; its cavity was greatly reduced in size, and its walls of great thickness. The aortic valve, by the water-test, showed no incompetency. The most extreme grade of aortic stenosis existed; the semilunar leaflets were firmly adherent to one another, resulting in the formation of a funnel-shaped deformity, the small end of which projected into the aorta some distance, and possessed an opening that would no more than admit the tip of a slate-pencil. The aortic orifice also showed some slight degree of atheroma.

FIG. 3.



Showing narrowing of the aortic orifice; the leaflets of the valve adherent together, resulting in a funnel-shaped projection, the narrow end of which points toward the aorta. Observed from the aortic aspect.

The abdomen was found to contain a large quantity of ascitic fluid. The intestines appeared to be normal. The other organs presented the conditions that might be expected to attend on advanced case of cardiac disease in which the right side of the heart had become involved. The liver was enlarged, congested, its lobules distinct, and its tissue upon section dense. The spleen also was enlarged and of increased firmness. The kidneys were somewhat increased in size and congested, the cortices being increased in width; their capsules stripped readily. The remaining abdominal organs were normal.

The accompanying illustrations, made by Dr. Stewart, well represent the various valvular lesions, and add materially to the value of the report.

Especially to Bedford Fenwick, and subsequently to Leudet, are we indebted for whatever of accuracy pertains to our present knowledge concerning tricuspid stenosis. In 1881, 1882, and 1883 Fenwick collected and analyzed before the London Pathological Society a series of seventy cases presenting this lesion, and in his Paris Thesis of 1888 Leudet has collected together, including the cases previously collated by Fenwick, one hundred and fourteen cases, with the results of the autopsies. The results of Leudet's observations as regards the influence

# 182 ASHTON, STEWART: CASE OF TRICUSPID STENOSIS.

of sex, age, antecedent causal history and the association, or non-association, of other valvular lesions, we have summarized as follows:

Sex.	No.	Age at death.	Previous history.	Associated lesions.
Female	86	2 from 60-64 years.	11 out of 60 cases had antecedent rheumatism; in 54 cases no history was mentioned.	{ Tricuspid and mitral . . . 78 " mitral, and aortic . . . 21 " " and pulmonary . . . 1 " and pulmonary . . . 3 " alone . . . 11 114
		4 " 50-60 "		
		14 " 40-50 "		
		24 " 30-40 "		
		33 " 20-30 "		
		3 " 10-20 "		
		6 ages not mentioned		
Male	22	2 from 60-64 years.		
		2 " 50-60 "		
		3 " 40-50 "		
		3 " 30-40 "		
		6 " 20-30 "		
		5 " 10-20 "		
		1 age not mentioned		
Not mentioned	6			

Since the publication of Leudet's Thesis we have found recorded in literature sixteen instances of tricuspid stenosis; but for three of these cases we have been unable to obtain more than the references. An analysis of the remaining thirteen cases and our own, however, offers some interesting comparisons with the results obtained from the above summary of Leudet's cases. Thus, we find these cases offer the following points:

Observer.	Sex.	Age.	Previous history.	Associated lesions.
Shattuck . . . .	F.	43	No rheumatism, but alcoholic.	Tricuspid, mitral, aortic, and pulmonary disease.
Chaplin . . . .	F.	18	No rheumatism, but scarlet fever.	Tricuspid and mitral stenosis.
Chaplin . . . .	F.	Not given.	Rheumatism.	Tricuspid and mitral stenosis.
Vanhan . . . .	F.	26	No rheumatism.	Tricuspid and mitral stenosis and aortic vegetations.
Vanhan . . . .	F.	50	No rheumatism.	Tricuspid and mitral stenosis and aortic vegetations.
Phillip . . . .	M.	46	No rheumatism.	Simple tricuspid stenosis.
Gibson . . . .	F.	31	Rheumatism.	Tricuspid and mitral stenosis.
Gibson . . . .	F.	21	No rheumatism.	" " " "
Balfour . . . .	M.	14	" " " "	" " " "
Thacher . . . .	F.	29	" " " "	Tricuspid, mitral, and aortic stenosis.
Grawitz . . . .	M.	24	Not mentioned.	" " " " " "
Hay . . . .	F.	33	" " " "	" " " " " "
Drummond . . . .	F.	40	" " " "	" " " " " "
Ashton and Stewart . . . .	M.	19	Doubtful rheumat.	" " " " " "

The above may be summarized as follows:

Sex.	No.	Age at death.	Previous history.	Associated lesions.
F.	10	2 from 41-50 years.	Only 3 out of 11 cases had antecedent rheumatism; in 3 cases no history was mentioned.	Tricuspid and mitral . . . . . 5
		3 " 31-40 "		" mitral, and aortic . . . . . 7
		3 " 21-30 "		" " aortic, and pulmonary . . . . . 1
		1 " 11-20 "		" alone . . . . . 1
M.	4	1 not mentioned. 46, 24, 19, 14 years.		14

From these tables it will be at once noticed that the preponderance of tricuspid stenosis in the female is striking. Thus, in 108 of the 114 cases collected by Leudet in which the sex of the individual is mentioned, the lesion occurred in 86 females and only 22 males. Adding to these figures the 14 cases collected by ourselves, we find that 96 females were the victims of this lesion, and 26 males, a proportion in favor of the former of nearly 79 per cent. This difference in the liability of the sexes has been remarked by most observers, and Bedford Fenwick states its relative occurrence to be higher than even the above figures would show, claiming that it occurs seven or eight times more frequently in women than in men. The cause for the relatively greater predisposition of the female sex is held by Fenwick to be that the work of women is less laborious, and that in consequence the action of the heart is less powerful. As a result, back-pressure is slighter, and therefore the valve disturbance is less than in men. In men laborious occupations, greater compensatory hypertrophy, and more powerful cardiac action prevent the inflamed edges of the valves from adhering together.

If this explanation be the correct one, it would apply to the case which is the subject of this paper, inasmuch as this patient had always been nverse to nctive exercise and had never followed any occupation entailing excessive exertion and, therefore, heart-strain. For these reasons the back-pressure may have been slight and the valve disturbance of only limited degree, thus permitting of the ndbering together of the inflamed edges of the valves. This point if followed up might show that in the majority of the instances of the male sex presenting the lesion of tricuspid stenosis there exists a bistory of comparative freedom from much muscular strain, and that, therefore, they could in reality be classed with the female sex so far as their predisposition to tricuspid stenosis is concerned. If this should prove to be the fact, it would go far toward confirming the position assumed by Fenwick.

In reference to the age at which death occurs in individuals the subjects of tricuspid stenosis, Leudet states that the decade during which death takes places most frequently is between the ages of twenty and thirty years, and it will be noticed that the cases that we have collated confirm this statement. It will be seen, therefore, that the existence of tricuspid stenosis is unfavorable to a long life. Nevertheless, that it is not absolutely incompatible with a moderate degree of longevity is seen from the fact that in 33 of the 122 cases of which the ages are recorded, death did not happen until after the fortieth year. It cannot be controverted, however, that in tricuspid stenosis, as stated by Leudet in his conclusions, the prognosis is more fatal than in any other form of valvular defect. From the very nature of the affection this cannot but be the case, as with tricuspid stenosis there exists as a primary condition that which occurs only in the last stages of other valvular lesions;

namely, dilatation of the right anricle and consequent venous engorgement. Thus, from the onset in this disease there is a transference of the blood pressure from the arterial to the venous system, and with it engorgement and subsequent structural changes in the viscera.

From the evidence deduced by Leudet it will be seen that the origin of tricuspid stenosis may be either congenital or acquired. Fenwick, however, asserts that the disease is always acquired, probably never congenital, and that in half the cases there is an antecedent history of acute or subacute rheumatism. So, also, Sansom writes: "I consider that it is without doubt the result of an endocarditis of the rheumatic form, causing adhesion and thickening of the curtains of the tricuspid valve and adjacent structures; it may be initiated in intra-uterine life, but is more commonly a development of the endocarditis associated with rheumatism that affects the mitral valve concurrently." Leudet further concludes that tricuspid stenosis in the fœtus is more often due to malformation than to endocarditis, but when endocarditis does occur in intra-uterine life the tricuspid valve is more frequently attacked than the other valves, because during foetal existence the greater amount of work is placed upon the tricuspid valve.

Whether acquired or congenital, however, there can be no question that the most frequent cause of tricuspid stenosis is an endocarditis of rheumatic origin. In Fenwick's statistics an antecedent history of rheumatism is found to exist in exactly 50 per cent. of the cases. This proportion is even higher in Leudet's cases, 41 out of 60 cases having had antecedent rheumatism, the previous history of the remaining 54 cases not being mentioned. At the lowest estimate, however, rheumatism existed as a morbid cause in over one-third of these cases, and it is fair to assume that in a large proportion of the cases in which the observers failed to mention the antecedent history, rheumatism probably played its usual part. In the 14 cases that we have collected rheumatism had occurred in but 3 of the 10 cases of which the previous history is recorded; and in our own case it is very doubtful whether the somewhat vague "rheumatic pains" from which the patient from time to time suffered could be ascribed to rheumatism. In this case, as will be observed in the clinical notes, no positive history of rheumatism could be obtained as having occurred either to himself or to any member of his family. He had been singularly free from infectious diseases, and but for the mild attack of measles from which he suffered when six years of age, and which is probably the least likely of all the acute affections to attack the endocardium, he presents a history of no disease that might be considered as having caused the extensive endocardial involvement. The possibility must not be overlooked, however, that the so-called rheumatic pains above referred to might in reality have been an evidence of rheumatic infection latent so far as the articular synovial



membranes were concerned, but disastrous in its effects upon the endocardium.

The extraordinary latency in the symptoms of this case is remarkable and bears a striking resemblance to the case reported by Shattuck. The only possible cause in Shattuck's case was an attack of scarlet fever at eleven years of age, the patient not dying until the age of forty-three. Throughout this period a remarkable tolerance was exhibited to the extensive lesions that existed, and which, in reality, involved every valve in the heart; to the slow development and the gradual extension of these lesions Shattuck ascribes the tolerance acquired.

Although in our own case no positive evidence of cardiac disability occurred until six years prior to the patient's death, it is quite clear from his history that the endocarditis must have existed from a much earlier period. The very fact that from the patient's earliest childhood he had been indisposed to join in the amusements of his playmates, and had always been inclined to remain in quiet and retirement, is significant that there existed some ill-defined self-consciousness upon the part of the patient that he was incapacitated from taking part in the active exercises of others of his own age. When, however, upon being subjected, six years prior to his death, to an occupation not entailing any very laborious exertion, it at once became evident that a serious cardiac disability existed. In this connection it is interesting to call attention again to the fact that the patient presented himself for examination as an applicant for life insurance in March, 1894, and, as already stated, he believed himself to be at the time in good health, not suspecting the serious condition of his heart which the physical examination revealed. He next came under observation during the latter part of June, 1894, when he presented himself for treatment at the Jefferson Hospital. His symptoms then were those of ruptured compensation, and the physical examination revealed, in addition to the murmurs of mitral stenosis and mitral incompetency, a well-marked systolic murmur having its greatest intensity near to the sternal junction of the sixth right costal cartilage. The latter murmur was believed to be dependent upon a relative stretching of the tricuspid orifice, and was held to be secondary to the disease at the mitral orifice; the existence of tricuspid stenosis was not suspected.

A study of the various lesions found in association with tricuspid stenosis reveals the fact that mitral stenosis is the most frequently associated lesion, and occurs in the great majority of the cases. Both Fenwick and Sansom assert, however, that tricuspid stenosis is never met with except in association with mitral stenosis; and, indeed, in all of Fenwick's cases this lesion of the mitral orifice did in reality exist. Nevertheless, in Leudet's collection fourteen cases are cited in which no lesion of the mitral valve coexisted; in eleven of these the tricuspid

valve alone was involved, and in three the tricuspid and pulmonary. So, also, in the fourteen cases that we have collected it will be seen that tricuspid stenosis unassociated with other lesions existed in one case. The above statistics of Leudet include the much-quoted instances of simple tricuspid stenosis reported by Duroziez and Torres Homem. The occurrence of tricuspid stenosis, however, not associated with mitral stenosis must be considered as a pathological curiosity. So striking is this fact that Leudet looks upon the disease at the mitral orifice as being the primary cause, in many instances, of the tricuspid lesion.

Aortic stenosis in association with tricuspid stenosis is stated by Sansom to occur in one-fourth of the cases. A study of the 128 cases shows that this lesion coexisted in twenty-nine of them, being a proportion of nearly 23 per cent., so that the statement of Sansom is about correct. It will further be noticed that no instance is noted in which the aortic disease is not also associated with mitral disease; in other words, tricuspid stenosis is never associated with aortic disease unless there also exists mitral stenosis, a point to which, I believe, attention has not until now been directed.

In such a case as the one the basis of this paper the question naturally arises, which valve system was the first to become diseased? From the drawings it will be noticed that the endocarditis is far more advanced in the left than in the right heart. Although this might be expected to be the case when we take into consideration the fact that the left heart is called upon to do the greater amount of work, and, therefore, would show the greater extent of lesion, a closer study of the lesions will show the existence of a simple adhesive endocarditis in the right heart, while in the left heart, especially upon the mitral valve and about the mitral orifice, even extending a considerable distance upon the inner wall of the auricle, will be seen numerous atheromatous and hard calcareous masses—a condition that is really remarkable considering the age of the patient. This is shown in the drawing illustrative of the mitral valve by the white shadings about the mitral orifice and upon the wall of the auricle. It seems to us, therefore, that the disease began first in the left heart, and that it later extended to the right heart, where it developed more gradually and insidiously as the result of the same cause which at first attacked the left heart, and which no doubt subsequently became repeatedly operative.

The high grade of aortic stenosis is well demonstrated by the drawing illustrating the condition of that valve. Notwithstanding this extreme stenosis its existence was not diagnosed during life. This failure of diagnosis, as well as the absence of the radial pulse during life, require explanation. The cause of the latter phenomenon becomes evident if we reflect that because of the extreme mitral narrowing the quantity of blood passing into the left ventricle was necessarily very small, and that

the obstruction offered to the passage of this small quantity of blood into the aorta by the gently-narrowed aortic orifice was sufficient to obliterate completely the limited impulse that would have been imparted to the blood-column had aortic stenosis not existed. The same physical conditions, too, explain the failure to diagnose the existence of aortic stenosis; the small amount of blood in the left ventricle, and the difficulty with which it must have been forced through the narrowed aortic orifice, must have resulted in the production of a murmur possessed of such a low degree of intensity as not to admit of its perception.

In conclusion we will say that the diagnosis of tricuspid stenosis during life is rarely made, and in this connection the statement is made by Shattuck that in the eighty-nine cases upon which he bases his article, and which include the seventy cases brought forward by Fenwick, a diagnosis was made during life in less than half a dozen instances. This writer concludes that: "Whether a presystolic tricuspid souffle can be heard or not, tricuspid stenosis can be pretty safely diagnosed if the patient is a female with rheumatic history, has mitral stenosis, perhaps also aortic disease, and presents the evidences of prolonged or recurrent venous stasis of greater or less degree."

The only particulars in which our case answered to this description were in the doubtful antecedent history of rheumatism and the existence of prolonged and marked cyanosis, a cyanosis which in its degree and persistency was remarkable; and in the light thrown upon the case by subsequent events we cannot but think that this symptom is of extreme significance as pointing to tricuspid stenosis, for in no other cardiac lesion is it found in so marked a degree, so prolonged in duration, and so uninfluenced by treatment.

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