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Evaluation of symptoms and signs of organic mitral stenosis

John M. Kroyer
University of Nebraska Medical Center

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EVALUATION
OF SYMPTOMS AND SIGNS OF ORGANIC MITRAL STENOSIS

JOHN MONTGOMERY KROYER

.....Of all valvular lesions, stenosis of the mitral valve is the most protean in its manifestations, sometimes being so cleverly disguised that one scarcely suspects the wolf behind the apparent, at other times so outspoken and brazen that a veritable beginner might recognize its true nature at once. I am therefore convinced that of all valvular lesions, mitral stenosis is the most difficult to diagnose.....

Goodman, E.H. 1919

INTRODUCTION

As the previous quotation indicates, mitral stenosis is capable of exhibiting a galaxy of signs many of which are dependent upon the human ear for detection. Though we recognize the human ear as one of the most remarkable innovations of nature, we still must be aware that it is guilty of misinterpreting noise as to audibility, tone, frequency and intensity. With this statement granted, it is now safe to conjecture that mitral stenosis will continue to be misdiagnosed until such time that science is capable of analyzing auscultatory signs by methods far more precise than that afforded by the human ear.

The intent of this paper is to discuss the symptoms and signs seen in mitral stenosis with an attempt made to evaluate these findings partly by their popularity with students of cardiology and partly by the author's own conclusions. Perhaps a criterion can be established which will not be infallible but which will allow one to most safely diagnose mitral stenosis.

HISTORY

Clinical knowledge of the heart is of recent development, but its discovery as an organ of the body is ancient.

There is no mention made of a cardiac organ in stone age history, its discovery was made in Ancient Egypt (several thousand B.C.). But nothing was learned of the organ because it was considered to have something to do with the future of the individual and after death, it was never touched. However, recent dissection of bodies expertly mummified in that period has as yet not revealed any mitral stenosis.

Then there was Homer who in 1001 B.C. described the location and beating of the heart, and Aristotle who in 350B.C. gave an anatomic description of the animal heart. Galen in 160 A.D. made the first detailed description of the human heart but said nothing of the mitral valve and considered the heart not a muscular organ. So influential were Galen's writings that 1500 years elapsed before anyone opposed Galen. It was da Vinci in the sixteenth century who said the heart was muscle tissue. Vesalius in 1543 named the mitral valve because when opened, it resembled a bishop's miter.

John Mayow in 1699 was the first man to describe

the pathology of mitral stenosis by autopsy after treating a man several years for dyspnea, palpitation and syncope. Pitcairn in 1788 noted the relationship of rheumatic fever to mitral stenosis.

Corvisart in 1800 described a thrill over the mitral valve when stenosed.[2]

Laennec in 1826 was the first to really open the door to cardiac research when he developed the stethoscope. Nine years later, Williams definitely established a diastolic murmur as caused by mitral stenosis. Then Austin Flint came along and described a diastolic murmur in aortic regurgitation so the field again was completely confusing.[3]

But in 1862, more than 150 years after the first pathological description, Durosiez gave the first clinical description of mitral stenosis which was so accurate that his auscultatory findings are in accord with present day interpretation. However, he did not completely appreciate the mechanism of production of the heart sounds. He gave birth to the phrase "ffout-ta-ta-rou" as an imitation of the cardiac sounds in mitral stenosis. He said, "ffout was the presystolic murmur and first sound, ta-ta was the second sound reduplicated, and rou was turbelescence of blood flowing during the rapid

ventricular filling phase." Rouches in 1888 completed the picture by explaining that the first ta was the second heart sound but the second ta was actually the opening of the diseased mitral valve. Thus he created the term opening snap in mitral stenosis.[4] From that time on the outspoken case of mitral stenosis was easily diagnosed but there still remained the host of atypical cases some of which still remain a mystery.

SYMPTOMS

One of the most constant factors in the mitral stenosis syndrome is a history of rheumatic endocarditis. Rarely stenosis of the mitral orifice may be due to atherosclerotic changes in the valve leaflets and very rarely this may be a congenital stenosis (about 2% of congenital hearts). These two factors may account for 2-3% of mitral stenosis whereas all the rest are due to rheumatic endocarditis. So if one cannot ascertain this infection in the history, one should give a guarded diagnosis of stenosis.[5,6]

The symptomatology of this affliction is not particularly significant. A stenotic patient was described as such during the era of medicine when the physician had only a scant supply of definite techniques and he

practiced medicine as an art which held inspection as its main tool. Obviously then, only the late cases of stenosis were recognized. The picture was something like this. The patient walking into the physician's office was rarely a large person, usually rather poorly developed and undernourished. His face seemed drawn and his eyes appeared sick and worried. There was a peculiar dusky, turgid appearance about his face and hands. Because you had a few steps leading to your office door, he was under labored respiration from his ascent. You walked over to greet him and when he offered his hand in welcome, you felt it to be cold though the day outside be warm and pleasant. Questioning the man as to symptoms and past illnesses revealed little except that he, as most other of the time had had many illnesses. When you began to feel of him, you found his pulse racing in the distance. Then you observed that his legs especially ankles were large though his body was small. Very often also his belly protruded and was firm not as if deposited with fat. At the end of the examination you may have concluded that he had ossification of the mitral valve or you may not have ventured any diagnosis. At any rate the physician was usually diagnosing cardiac decompensation unknowingly.[7,8]

SIGNS

Perhaps now we may discuss the numerous signs in mitral stenosis not in order of their elucidation during the patient's interview, but rather in order of their significance. These may be classified under two large groups, presumptive and confirmatory. In the discussion each sign will be given a differential diagnosis when warranted.

PRESUMPTIVE

The field of electrocardiography has offered some hints toward diagnosis, but are non-specific in nature and at best, when present, help to complete the typical picture but when absent, subtract nothing. Electrocardiography at best will show these things. Right axis deviation consistant with right ventricular hypertrophy is marked in late cases in which mitral regurgitation is minimal.[9,10,11,12] Secondly auricular fibrillation when seen before the age of forty is most often due to mitral stenosis in advanced form.[9,12] Thirdly the P-wave shows changes when the heart is not fibrillating. It may be wide indicating auricular dilitation, high denoting hypertrophy, or notched indicating disturbed conduction from ischemia or infarction.[10,11,12] None of these signs appear until stenosis is well developed and long standing.[13]

Radiology has recently offered additional help in mitral stenosis. Polevski[16] was apparently the first to use lateral x-rays to diagnose left auricular enlargement. He and others[12] have said that in silent stenosis, left auricular enlargement is pathognomonic of mitral stenosis. Since then, however, it has been found that left auricular enlargement is also seen in hypertension, thyrotoxicosis, congenital heart disease and patent ductus arteriosus, so this sign is hardly pathognomonic.[11] Now it is considered that left auricular enlargement, prominence of pulmonary conus, and calcified mitral valves in the absence of other disease is helpful in making one suspicious of mitral stenosis, but when absent does not rule it out.[10,14] Also there is a sign, cardiac enlargement, when absent is a factor in stenosis.[15] This will be discussed more fully later.

Auscultation---Herein lies most of the changes seen in mitral stenosis. The presumptive signs only will be discussed now.

The presystolic murmur was the first known auscultatory sign in mitral stenosis. Joseph Bertin 1824 was the first to describe the diastolic murmur and Fauvel, 1850 was the first to time the murmur as presystolic and as being loudest at the apex. In 1900 William

Broadbent said a presystolic murmur was pathognomonic of mitral stenosis. For many years, this was the concensus of opinion concerning mitral stenosis. But in 1925, Crummer said no murmur is characteristic of mitral stenosis. Finally Paul White recently said that if one must hear a presystolic murmur before diagnosing mitral stenosis, 50% of the cases would go undiagnosed.[2]

What is the cause of the presystolic murmur? Evans[17] using a Cambridge stethograph timed with the Cambridge electrocardiograph examined 31 cases with audible presystolic murmurs. He noted that on his graphic sound tracing 29 cases showed the murmur to begin before the S-wave of the EKG. Since he interpreted the S-wave as the beginning of the ventricular first sound, he said that the murmur must be of auricular origin. Nylin and Biorck[18] about the same time obtained a phonocardiogram timed by EKG of a woman with mitral stenosis and complete block. They noted that when the P-wave occurred in middiastole, .16 seonds later there occurred a low frequency murmur which was audible. When the P-wave occupied its normal space, preceding the QRS, then the typical presystolic murmur was heard. This along with other evidence[19] seems conclusive enough that the presystolic murmur is auricular in origin. The mechanism of production

of the murmur itself is apparently due to the passage of blood, propelled by the auricular systole, through the narrowed (actual or relative) mitral orifice.

Presystolic murmurs occur in conditions other than mitral stenosis per se.[3]

(Austin Flint murmur. This is a well accepted presystolic murmur associated with aortic regurgitation causing a relative mitral stenosis.

(2. A presystolic murmur is sometimes seen with adherent pericardium. This is probably also on the basis of relative stenosis.

(3. A presystolic murmur is sometimes seen with great left ventricular dilitation per se.

(4. A presystolic murmur may be transitory simply in an over active heart.[20]

If one can satisfactorily rule out the above four conditions, then a presystolic murmur in a patient with rheumatic fever history is good presumptive evidence for mitral stenosis.

But there are many cases of mitral stenosis that do not have a presystolic murmur. It is well known that a majority of cases of mitral stenosis eventually develop auricular fibrillation.[2,5,12,20] The onset of auricular fibrillation denotes the cessation of auricular

contraction; hence the cessation of presystolic murmur but the continuance of the mitral stenosis, so this sign is not an infallible guide.

Another occasional sign in stenosis is a thrill over the mitral valve. This was first described by Corvisart in 1800.[2] This is not considered an important sign now because of its frequent absence and its difficulty in timing when present.

Another sign which appeared early in the evolution of knowledge concerning mitral stenosis was the diastolic murmur first described by Hope in 1832.[2] Later he rejected this murmur and it was forgotten until in 1888 Graham Steele firmly established the basis for this murmur.[3] In mitral stenosis, less blood than normal gets through the mitral orifice. This results in a decreased stroke volume which leads to small and rapid pulse. At the same time there is an excess of blood in the left auricle with resultant backup of pressure into the pulmonary tree. This condition is termed pulmonary hypertension.[5] Because of the increased pressure in the pulmonary artery, its semilunar valves following systole snap back with greater force than the aortic valves. The result is called accentuated pulmonary second sound. The high pressure sometimes dilates the pulmonary

arterial walls sufficiently to render its semilunar valves incompetent. This results in an early diastolic murmur most marked over the pulmonary area. This murmur was so named the Graham Steele murmur in 1888.[2,20] The murmur and accentuated pulmonary second sound have since come to be considered occasional signs in mitral stenosis.[10,13,21]

An accentuated first sound at the apex is considered a sign of variable importance among different authors. References are[1,11,13,16,22,23,24] This sign is very frequently present but is considered as only presumptive evidence because of its difficulty in recognition as accentuated over normal. The first sound has a wide range of normal variability concerning intensity and there is no fine line of separation between normal and accentuated. Therefore for accuracy of diagnosis one must rely on other evidence. Doch suggests that the first sound is entirely valvular in origin and that if when the ventricle contracts, the mitral leaflets are relaxed, then the resultant snap of the leaflets to the closed position produces a loud sound. Whereas if the mitral leaflets are partially closed and tension is on the chordae tendinae when the ventricle contracts, then the resultant sound is soft. Using this theory,

Keith[22] explains that in mitral stenosis the intra-auricular pressure is prolonged because the impeded blood is still flowing into the ventricle and the mitral leaflets are flattened against the ventricular wall, the chordae tendinae under absolute minimal tension. The resultant sound is accentuated over the normal because in the latter, the mitral leaflets are partially closed. This theory for mitral stenosis holds for P-R intervals up to .23 second.

The most important of all presumptive signs in mitral stenosis is the Opening Snap. Perhaps the reason for not considering this as a confirmatory sign is didactic, but it does occur with slightly less frequency than the confirmatory sign to be mentioned. Fauvel 1843 was probably the first to hear an opening snap but he unknowingly called it the reduplication of the second sound.[3] Durosiez in 1862 was the first to accurately describe this sound, but he too thought it to be a reduplication of the second sound. Rouches in 1888 possibly at the suggestion of Potain ventured that this sound was the snapping open of the diseased mitral valve and was actually a third heart sound. Thus he created the term opening snap of mitral stenosis, which held to this day.[4] Orias and Menendez by phonocardiography

timed with venous pulse found the opening snap to occur just before the summit of the V-wave of the pulse and thus concluded this must be the opening of the atrio-ventricular valves.[4]

Wolferth and Margolies[25] have given the best discertation of opening snap of any to my knowledge; therefore the following discussion is largely from their article. The opening snap is audible in the majority of cases of mitral stenosis. It is always short and high pitched, usually being quite loud. This resembles a reduplicated second sound, but differs from it by being heard best always near the apex whereas the split second sound is always loudest at the base of the heart. Third heart sounds are readily distinguishable from the opening snap because they are always low frequency, low intensity sounds heard always at the apex.

Time intervals of the three sounds measured from the onset of the second sound.

Reduplicated second sound	.06 sec. or less
Opening snap	.06-.12 seconds
Third heart sound	.12 sec. or more

So far as is known, this opening snap when heard is caused only by mitral stenosis. The disadvantage of this sign is that it is usually absent in early cases when

the mitral leaflets are as yet too mobile and absent in late cases when the mitral leaflets are too immobile.

CONFIRMATORY

I believe there is only one sign worthy of being classified as confirmatory evidence of mitral stenosis. In the absence of this sign, one should give only a presumptive diagnosis. This all-important landmark to be discussed is the mid-diastolic murmur.

From the interpretation of old writings, apparently Durosiez 1862 was the first to describe a mid-diastolic murmur. He said it followed immediately the reduplicated second sound and was indicative of turbelescence of blood during the rapid ventricular filling phase.[4] From that time until now, his description has remained classic and unchanged. Paul White[20] in his latest book has given an excellent description of the murmur. He states it to be rough, rumbling, low-pitched and faint in character. The onset is with the opening snap of the mitral valve, and is restricted in audibility to a small area over the apex showing no transmission. The murmur is heard best with a bell stethoscope and with the patient in complete supination.

Many writers[26,27,28,29,30,31] have in part discounted the value of mid-diastolic murmurs on the basis that

they occur many times when mitral stenosis was found non-existant by post-mortem. Robinow and Harper[32] have offered an excellent differential diagnosis for the mid-diastolic murmur. They found it to exist in the following conditions:

(1 Organic mitral stenosis

(2 Austin-Flint murmur

(3 Adhesive pericarditis

(4 Chronic severe anemia

(5 Early rheumatic endocarditis in young people

(6 Acute nephritis with hypertension.

With the exception of (1, the murmur can be explained on the basis of relative stenosis. The cause of the relative stenosis in (2 has been discussed and all the rest are produced on the basis of left ventricular dilitation. Whether the murmur in these conditions is produced by relative stenosis per se or whether a dilated ventricle gives rise to eddy currents which vibrate the chordae tendinae cannot be answered at present.

Two other groups of writers[15,33] appreciated the existance of a functional mitral stenosis and gathered a series of cases in which a mid-diastolic murmur was present. They diagnosed organic mitral stenosis only in those cases in which there was no left ventricular

dilitation either by percussion or x-ray and found by autopsy, their error approached zero.

The one disadvantage of relying so heavily on the elicitation of a mid-diastolic murmur is that it is often missed by stethoscope because this murmur is usually just above the threshold of audibility. This can be overcome by the use of a phonocardiograph which is capable of accurately registering the inaudible as well as the audible components of cardiac sounds. Evans[17] examined 72 cases of mitral stenosis and found the mid-diastolic murmur clinically audible in 51 cases but visualized by phonocardiography in every case! Johnston[13] at Ann Arbor also found the greatest degree of accuracy in eliciting these murmurs by graphic sound registration.

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SUMMARY AND CONCLUSION

In a given case of suspected mitral stenosis after obtaining a history of rheumatic fever, one should place the signs elicited in the following category:

PRESUMPTIVE

(1 Electrocardiography

(2 Radiology

(3 Palpation

(4 Auscultation

a Presystolic murmur

b Graham Steele murmur

c Accentuated pulmonary second sound

d Accentuated first sound

e Opening snap

COMFIRMATORY

(1 Mid-diastolic murmur

Cabot has said recently that the average physician can diagnose mitral stenosis correctly only 50% of the time. However, perhaps if the physician would use the above criterion in either presuming or confirming the diagnosis and freely accept the aid of phonocardiography, he would find his accuracy in diagnosis will exceeding the 50% quota.

John M. Kroyer

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