RADIOLOGY

The Radiological Diagnosis of Cardiac Valvar Insufficiencies

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THE RADIOLOGICAL DIAGNOSIS of cardiac valvar insufficiency includes the description and identification of the insufficient valve or valves and their supportive structures, quantitation of the regurgitant flow, determination of the degree of myocardial functional deterioration that may be present, and detection of associated cardiac lesions.

Cardiac catheterization with selective angiography provides most, if not all, of this information. However, cardiac catheterization and angiography are not without risk. They are expensive, time consuming, and uncomfortable for the patient, and therefore are not employed until their need has been demonstrated by other, less elaborate diagnostic methods.

In the screening phase of the diagnostic workup of the patients with cardiac valvar insufficiency, such radiological tools as fluoroscopy with cineradiographic recording and, in particular, chest radiography are standard procedures. Although they may be very useful for specific diagnostic problems, the more recently added diagnostic techniques, such as isotope imaging and sonocardiography, have not as yet significantly reduced the need for conventional radiographic methods. With further methodological development, these tools may be expected to play a more significant role, particularly in follow-up examinations.

This communication is concerned with the conventional radiological methods of diagnosing cardiac valvar insufficiency: fluoroscopy, chest films, and selective angiography.

Methods

Fluoroscopy

Fluoroscopy may yield essential information regarding the presence of cardiac and valvar calcifications, specific cardiac chamber enlargement, and abnormal pulsations of the cardiac chambers and large vessels. In order to reduce the amount of radiation received by the patient and the attending physicians and to provide a permanent record, cineradiography is preferable to prolonged fluoroscopy.

Valvar calcifications are identified within the heart by the characteristic motion patterns for each valve. Since the motion of the individual cusps usually is severely restricted, the calcified aortic valve largely exhibits the motion of the valvar ring, i.e., downward in systole and upward in diastole. Mitral valvar calcification, on the other hand, shows dorsal excursions in systole and ventral movement in diastole, as the valvar ring motion is relatively small compared with the inherent motion of the leaflets.

For the specific diagnosis of valvar insufficiency, calcifications are of little value. They are common in stenosis and combined stenotic insufficiency, but rare in simple insufficiency.

Malfunction of valvar prostheses often requires angiography for diagnosis. However, fluoroscopy and plain cineradiography may assist in a preliminary effort to define the problem, because dehiscence of a prosthetic valve may cause a change in its motion pattern and position. The task is facilitated if an immediate postoperative baseline study is available for comparison. Attempts have been made to establish normal postoperative motion patterns for aortic and mitral valvar prostheses, but the practical value of such examinations is limited, as normal motion pattern may be seen in significant perivalvar insufficiency. Change in prosthetic valvar position may also be detected on serial chest films (fig. 1).

For the evaluation of selective chamber enlargement, fluoroscopy has little to offer beyond that which can be obtained from chest films. However, the dynamics of an enlarged left ventricle can often be assessed roughly. Large variations in the width of the ascending aorta may be seen in marked aortic valvar insufficiency, and pulsations of the main pulmonary artery are increased in marked pulmonic valvar incompetence, particularly when incompetence is combined with pulmonary hypertension.

Chest Radiography

Chest radiography offers a wealth of information about the functional and anatomic features of cardiac valvar insufficiencies, such as total heart volume, selective chamber enlargement, aortic and main pulmonary arterial dilatation, the condition of the lungs and their vessels, the large systemic veins, as well as cardiac and vascular calcifications. However, these cardiovascular parameters are often nonspecific. For example, left ventricular enlargement may indicate increased volume load, myocardial ischemia, or increased pressure load, as well as a combination of these factors. Furthermore, selective chamber enlargement may be difficult to demonstrate and virtually impossible to quantitate.

Because of the theoretical and practical difficulties involved, a specific diagnosis of cardiac valvar insufficiency cannot be made from chest films alone. However, when used in combination with other noninvasive methods, plain films often result in a correct diagnosis.

Cardiac calcifications can rarely be located with precision on a plain chest film, as the motion patterns are essential components of the diagnostic process. Fluoroscopy with
cinerecording is a more effective method. Tomography may be helpful in demonstrating calcifications, but it rarely adds practical, significant information. The presence of aortic wall calcifications raises the possibility that the valvar insufficiency may be caused primarily by vascular changes rather than by defects of the valve per se. Aortic wall calcifications are common in the general population, however, and the value of the recognition of such calcifications is limited.

**Total Heart Volume.** Ventilation, posture, total blood volume, blood flow, and heart rate are among the physiological factors, irrelevant to the diagnosis of a specific cardiac lesion, which cause variations in total heart volume. The combined dimensional response of the cardiac chambers to these parameters is difficult to assess, and significant individual variations in cardiac chamber volumes occur.

To reduce irrelevant physiological changes, Larsson and Kjellberg have proposed that patients be examined in the recumbent position during shallow breathing. However, these authors also report that no significant volume difference was found between measurements taken in upright and recumbent positions until the heart rate exceeded 80 per minute. If a prolonged Valsalva maneuver is avoided, standard chest films form an adequate basis for clinically useful heart volume measurements. Amundsen measured total heart volume on such films in 755 patients with and without heart disease, using the formulae developed by Kahlstorf. He suggests that definitely enlarged hearts be separated from those not considered to be enlarged by a borderline zone which for males is 500 to 540 ml/m² and for females 450 to 490 ml/m².

Cardiac valvar insufficiencies as a group, like left-to-right shunt lesions, are characterized by volume overload. Typically, they cause greater cardiac enlargement than do obstructive lesions. Total heart volume determination is most valuable in monitoring patients with previously diagnosed heart disease. Used in this way, heart volume measurements do not depend upon variations between individuals, so that even small variations in volume in a single patient may be significant. Variations in heart volume correlate closely with the results of exercise testing and with clinically observed changes in the patient's cardiac status.

Hirshfeld et al. studied the relationship between the cardiothoracic index and survival rates in patients with valvar replacement for aortic valvar insufficiency and stenosis. They found no correlation between a single preoperative measurement of the cardiothoracic index and the postoperative course in patients with aortic valvar insufficiency. This may be explained by the fact that only a single preoperative measurement rather than serial measurements was made. Postoperative serial measurements showed good correlation between long survival and decreasing cardiothoracic index.

**Selective Chamber Enlargement.** Diagnosis of a specific cardiac valvar insufficiency depends on the recognition of selective chamber enlargement and appropriate dilation of the great vessels. Figure 2 shows the chambers which become enlarged when various valves are insufficient. The degree of enlargement depends on the amount of regurgitant flow as well as the duration of a lesion; for example, acute aortic or mitral valvar insufficiency may show only little cardiac enlargement initially when the increased left ventricular output is accomplished mainly by increased ejection fraction rather than by increased end-diastolic volume. The
Table 1. Effects of Chamber Enlargement Seen on Plain Chest Films

<table>
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<tr>
<th>Right atrium</th>
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<tbody>
<tr>
<td>1. Prominence of right cardiac border on PA radiography.</td>
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<tr>
<td>2. Prominence of right atrial appendage on LAO view.</td>
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<tr>
<td>3. Disproportionate enlargement of heart on PA as compared with lateral.</td>
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<table>
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<th>Right ventricle</th>
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<tr>
<td>1. Filling in of retrosternal space.</td>
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<tr>
<td>2. Upward displacement of cardiac apex.</td>
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<tr>
<td>3. In extreme cases, the right ventricle may form the left cardiac border.</td>
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<table>
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<th>Left atrium</th>
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<tr>
<td>1. Focal indentation on barium-filled esophagus.</td>
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<tr>
<td>2. Double density along right cardiac border on PA view.</td>
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<tr>
<td>3. Prominence of left atrial appendage on PA view.</td>
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<tr>
<td>4. Elevation of the left mainstem bronchus.</td>
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<td>5. Displacement of the descending aorta.</td>
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<td>6. In extreme cases, the left atrium may form the right cardiac border.</td>
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<th>Left ventricle</th>
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<tr>
<td>1. Prominence of left cardiac border on PA film.</td>
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<tr>
<td>2. Depression of cardiac apex.</td>
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<tr>
<td>3. Displacement of posterior inferior border of heart to a position behind the inferior vena cava at the diaphragm or 1.5 cm behind the inferior vena cava 2 cm above the diaphragm.</td>
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Plain film findings of specific and combined chamber enlargement have been previously described and are demonstrated in table 1.

Patients with aortic or pulmonic valvar insufficiency display an enlarged ascending aorta or pulmonary artery, respectively. However, because of extensive normal variations and difficulties in correctly establishing the width of these vessels by means of plain films, together with the many variations in the degree of dilatation caused by valvar and other diseases, the detection of large vessel dilatation in aortic and pulmonic valvar insufficiency is of small diagnostic significance. Turbulent blood flow which occurs in stenotic lesions is a more powerful stimulant to vascular dilatation than increased blood flow per se.

Redistribution of the pulmonic blood flow with greater flow and larger vessels in the upper parts of the lungs than in the lower parts is the result of increased pulmonic venous pressure. In mitral valvar insufficiency, redistribution of pulmonary blood flow is not a characteristic finding (although it occurs in severe, acute mitral valvar insufficiency and in patients with deteriorating myocardial function). Many patients with longstanding mitral valvar insufficiency and large left atria have normal left atrial pressures, and therefore no pulmonary vascular changes would be expected.* When the incompetence of the mitral or aortic valve has resulted in left heart failure, pulmonary venous hypertension and dilatation will occur.

Angiocardiography

The angiocardiographic examination provides direct demonstration of the incompetent valve, quantitation of the regurgitant flow, evaluation of the myocardial function of the involved ventricles, and demonstration of significant associated lesions, such as dissecting aortic aneurysm and coronary arterial obstruction.

The preferable radiographic recording technique is 35 mm biplane cineradiography with an exposure rate of 45 to 60 frames per second. Serial, full size, direct angiocardiography yields better image quality for the individual frame, but the advantage of movie demonstration of the behavior of the contrast medium, the valves and the other cardiac structures is so great that serial angiocardiography can no longer be considered an adequate tool for the diagnosis of cardiac valvar insufficiencies.

Measurement by angiocardiography requires rapid exposure rates in order to cover maximal systole and diastole and to assess effective diastolic filling time in comparing right and left ventricular stroke volumes.10 The full utilization of a biplane cineradiography system for quantitative functional and anatomic diagnosis requires that various distances, used in the calculations, be recorded at the time of the examination for each individual angiocardiographic run, as shown in figure 3. These measurements can be placed conveniently on the film storage box.

As manual measurements of anatomic structures with associated calculations are too time consuming to be practical, a data tablet and a computer, preferably of the dedicated desk type, are necessary adjuncts of a biplane cineradiographic system in the diagnosis of valvar insufficiencies.

Selective angiocardiography implies that the contrast medium needed for the visualization of the cardiac structures is injected selectively by means of a catheter into the cardiac chambers or vessels of interest. For the specific purpose of volume calculation of the left atrium and left ventricle, contrast medium may be injected semiselectively into the main pulmonary artery or the right ventricle, thus providing pictures of the leftsided chambers without ectopic beats.

To demonstrate mitral or aortic valvar insufficiencies, the catheter is usually introduced through a femoral or brachial artery, the tip being placed in the ascending aorta approximately 3 cm above the aortic valve or in the left ventricle, respectively. When the left ventricle is used, care must be taken to place the catheter tip at some distance from the...
mitral valvar ostium. Pulmonic valvar insufficiency may be demonstrated in a rare case from the arterial side if a patent ductus arteriosus is present. However, as a rule, rightsided cardiac valvar insufficiencies can only be demonstrated by angiocardiography after the insertion of the catheter from the venous side, with placement of the catheter tip in the right ventricle for tricuspid valvar study and in the main pulmonary artery for examination of the pulmonic valve. As a result, the pulmonic and tricuspid valves must be examined with the injection catheter located in the valvar ostium, potentially causing spurious valvar insufficiency. Accidental insufficiencies which are attributed to the presence of the catheter in the valvar ostium are often in reality caused by ectopic beats induced by the injection. When ectopic beats do not occur, the valve is capable of closing completely without regurgitation, even if the catheter is located in the valvar ostium, as long as the position is central and free in the ostium.11 Atroventricular valvar insufficiency caused by ectopic beats can usually be identified as such.

Normal closure of an atroventricular valve does require normal functioning of the pertinent atrium and the ventricle. Therefore, a regurgitation of contrast medium does not necessarily indicate an abnormal valve. Such regurgitation may result from functional abnormalities of the conduction system of the myocardium. So-called diastolic overflow may occur in patients with slow heart rate and enlarged hearts, often combined with reduced compliance of the ventricular myocardium. This phenomenon does not imply organic valvar lesions, although functional and organic lesions both may be present. It also has been shown that the competence of the aortic valve depends on a normally functioning left ventricle. Weak contractions may permit a regurgitation which is usually small and therefore of little hemodynamic significance.9, 12, 13 The analysis of these intricate and swift functional events obviously requires rapid exposure rate filmimg.

Measurements of Cardiac Chamber Volumes

Methods for angiocardiographic measurements of the volumes of the left atrium, the right ventricle, and the left ventricle have been developed with a validity that makes them useful tools for the functional evaluation of the heart in patients with cardiac valvar insufficiencies.14 17

Because of its regular shape and clear visibility in two projections after contrast medium injections in the main pulmonary artery, the left atrium early became the subject for quantitative efforts.18 As the volume variations of the left atrium account only in part for the blood transport through the atrium and because the absolute volume of the left atrium is determined by many other factors than the degree of mitral valvar insufficiency, measurements of left atrial volume are of little value in the quantitative assessment of mitral valvar insufficiency.

Ventricular volume variations, on the other hand, are directly related to the amount of regurgitant flow, since the gross ventricular output of the affected ventricle minus the effective forward flow constitutes the amount of regurgitant blood flow. The methods designed by Dodge19 and Arvidsson14 both assume that the shape of the ventricular cavity is ellipsoid; the volume is obtained by measurements on the films of key distances with a correction for magnification. There is a significant difference, however, inasmuch as the Arvidsson method presumes direct measurement of the transverse ventricular diameter (a procedure which is not always reproducible), while the Dodge method prescribes measurement of the longest diameter only, the transverse diameter being derived from a planimeter estimation of the ventricular surface area. As a result, an excellent correlation between true and estimated volumes is achieved by the Dodge method when applied to postmortem ventricular models, while the Arvidsson method, applied in the same manner without regression analysis, shows an overestimation of approximately 35%. Other methods based on the Simpson formula give equally good results (as does the Dodge method with the left ventricle) and, in addition, have the advantage of being applicable to the right ventricle.10, 20 22

The assessment of valvar regurgitation by indicator dilution techniques has been disappointing, methodologically, mainly because of the problem of inadequate mixing and unrepresentative sampling from the upstream chamber.23 Pressure recordings for the purpose of quantitating regurgitant flow have shown major inconsistencies.

Because of these difficulties and the prolonged catheterization often required for adequate sampling, radiological methods have been employed increasingly for the quantitation of regurgitant blood flow. The total stroke volume of the involved ventricle is measured by angiocardiography. By subtracting the effective forward stroke volume, the volume of regurgitating blood flow is obtained. The effective forward flow is usually measured by the Fick principle. Although a good correlation between angiocardiographic measurements and the Fick method has been demonstrated by many investigators, the comparison of data obtained in such different ways and at different times does create some uncertainty as to the validity of the results in individual patients. Practically simultaneous measurements of gross ventricular stroke volume and the effective forward flow can be obtained by angiocardiographic volume measurements of both the right and the left ventricles, as the stroke volume of the unaffected ventricle is equal to the effective forward flow of the involved ventricle. The feasibility of such comparative right and left ventricular stroke volume measurements by angiocardiography has been demonstrated.20

If two or more valves are incompetent, the regurgitant flow of each valve cannot be assessed by ventriculographic measurements. One must then rely on a direct estimate of the amount of contrast medium observed to regurgitate through the valve. Such estimates are quantitatively uncertain.

Ventricular volume measurements also provide a quantitative assessment of the ventricular myocardial function, because the ejection fraction can be calculated from the enddiastolic volume and the stroke volume, the latter being expressed as a fraction of the former. The normal value is 67%.

It has been shown24 that the ejection fraction is a reliable prognostic measurement when applied to valvar and ischemic heart disease.

Measurements of the Diameter of the Anulus of the Aortic Valve

Information about the width of the aortic valvar anulus before surgical intervention for aortic valvar replacement is
often requested by the surgeon so that the availability of a suitable valve prosthesis can be assured and a valve prosthesis of optimal size can be selected and prepared beforehand. Such measurements can be made from biplane angiographic records with great accuracy. The area of interest is located immediately below the lower attachment of the aortic valve. The width of this segment varies during the cardiac cycle, requiring a rapid exposure rate for maximal and minimal measurements.

**Specific Valvar Lesions**

**Aortic Valvar Insufficiency**

The secondary effects of aortic valvar regurgitation consisting of dilatation of the ascending aorta, enlargement of the left ventricle and to some degree the left atrium, form the basis for plain chest film diagnosis of this condition. Calcification of the aortic valve and the aortic wall may also be demonstrable on chest films. The degree of aortic dilatation may vary considerably. The factors involved include the degree of regurgitation, the duration of the condition, the presence of turbulent flow and, perhaps, the age of the patient when the aortic regurgitation began. As aortic stenosis and aortic wall changes also cause dilatation, such a finding is of limited diagnostic value.

Left ventricular enlargement and left atrial enlargement are nonspecific findings. However, when a diagnosis has been made, monitoring by serial plain films of the total heart size and, to the extent possible, specific chamber enlargement are helpful in the long term management of these patients.

**Angiocardiography.** The normal aortic valve is completely competent, e.g., no contrast medium can be seen to regurgitate from the aorta to the left ventricle. However, there is evidence that the closure of the aortic valve, like that of the mitral valve, may become less than perfect if the left ventricular myocardium is functionally insufficient. The effect on the closure of the aortic valve from such myocardial insufficiency is far less significant than on the closure of the mitral valve which is directly supported by myocardial structures.

Aortic valvar insufficiency is diagnosed angiocardiographically by the injection of contrast medium into the aorta approximately 3 cm above the aortic valve through a sidehole catheter so as to avoid displacement of the catheter tip during injection and also to avoid a jet stream of contrast medium directed toward the valve, since this may cause spurious regurgitation. There is no accurate alternative diagnostic method available for the demonstration of aortic valvar incompetence in general or its specific functional and anatomic features.

Congenital lesions of the aortic valve causing regurgitation usually take one of two shapes. They may consist of minor irregularities along the free edge of the cusp, frequently associated with asymmetrical development of the cusps and the sinuses of Valsalva, or the cusp may be fenestrated by a well defined defect. In the latter case, angiocardiography may show a clearcut, thin jet, often eccentrically directed. These small congenital defects are usually not of hemodynamic significance, but they may constitute an area vulnerable to bacterial attacks. Figure 4 shows contrast medium regurgitation from the ascending aorta to the left ventricle in a case of aortic valvar insufficiency of rheumatic etiology.

Calcific and noncalcific proliferative aortic valve disease on a congenital or an acquired basis often produces combined aortic stenosis and insufficiency. The thickened valve often shows no inherent motion, but follows the motion of the valvar ring.

The interrelationship between aortic valvar insufficiency and ventricular septal defect is well documented. Valvar incompetence is, therefore, often progressive, and the recognition of even small lesions is of practical significance. The subaortic region of the ventricular septum, weakened by the ventricular septal defect, does not properly support the valve, thereby supposedly making it incompetent. Logically, a ventricular septal defect, in order to reduce the support of the aortic valve, should be located high in the ventricular septum, close to the valvar ring. This is not always the case.

Membranous subaortic stenosis also is associated with aortic valve insufficiency. In patients suffering from this condition, a membrane located immediately below the aortic valve causes varying degrees of outflow obstruction. In practically all cases, one will find some degree of aortic valve incompetence as well. It is pedagogically useful to explain the insufficiency as a result of interference by the stenotic membrane with the diastolic closure of the valve. However, minor anatomic changes of the valves are often seen, sometimes making the valve itself incompetent. Figure 5 shows a cine of a subvalvar diaphragm which was associated with aortic valvar insufficiency. Surgical relief of the subvalvar stenosis often creates additional incompetence of the valve. The presence of aortic valvar insufficiency in a patient with a systolic pressure gradient in the valvar region should alert the diagnostician to the possible presence of a subvalvar membrane.

The typical feature of aortic valvar insufficiency resulting
from bacterial endocarditis is destruction of one or more cusps by the infectious process, with no significant thickening nor calcification of the valve and with normal valvar motility. The acute onset and often serious functional impairment of the patient's condition lead to early detection and therefore secondary changes, such as aortic dilatation and left ventricular enlargement, are often absent. Within weeks or months, left ventricular enlargement will develop if the patient survives.

Dehiscence may result in abnormal position of an aortic valvar prosthesis with paravalvar leak and abnormal motions of the prosthesis which can be seen on fluoroscopy with cineradiographic recording without contrast medium injection. However, the dislocation and change in motion pattern are not always great enough to permit diagnosis, and the degree of valvar insufficiency is not proportionate to the dislocation when present in detectable degree. Therefore, cardiac catheterization with selective contrast medium injection in the ascending aorta is indicated when prosthetic valvar dehiscence is suspected. Figure 6 shows tissue erosion in the vicinity of a prosthetic aortic valve with significant regurgitation caused by bacterial endocarditis. The movement of the ball in a ball-cage valve often can be demonstrated cineradiographically, with or without contrast medium visualization, permitting evaluation of a ball variability. Radiopaque impregnation of the ball facilitates this type of examination.

As the aortic valve is suspended in the most proximal part of the ascending aorta rather than in the muscular ostium of the ventricle, aortic wall disease is a frequent cause of aortic valvar insufficiency. Lesions of the aortic wall may cause aortic valvar insufficiency by dilatation of the aortic valvar ring, making the normal cusps too small to cover the ostium, or by direct functional or structural involvement of the aortic valve. It is usually impossible to tell with certainty, even after angiocardiography whether the valve is incompetent because of aortic dilatation or because of damage to the valvar cusps, or both. However, marked dilatation of the proximal ascending aorta and lack of demonstrable valvar changes indicate vascular rather than valvar involvement.

Dissecting aneurysms, syphilis, atherosclerotic, mycotic, and congenital aneurysms may produce valvar insufficiency. Dissecting aortic aneurysms may cause aortic regurgitation by aortic valvar dilatation or by direct interference with valvar closure. However, regurgitation does not occur consistently, even when the dissection proceeds to and involves the aortic valvar ring. Figure 7 shows regurgitation in a patient with dissecting aortic aneurysm involving the aortic valvar ring. The angiographic differentiation between dissecting aortic aneurysm on the one hand and valvar lesions on the other is not always easy. At surgery, an unexpected valvar lesion now and then turns out to be in fact a dissecting aortic aneurysm.

Syphilitic aneurysms of the ascending aorta, once common, now rare, produce in the full blown stage dramatic radiological findings, such as erosion of the sternum, thin, so-called egg-shell calcifications of the aortic wall, and cardiac enlargement compatible with aortic valvar insufficiency.

Atherosclerotic aneurysms of the ascending aorta causing valvar insufficiency by ring dilatation are rare.

Medionecrosis of the aortic wall may cause a characteristic bulbous dilatation of the ascending aorta, often involving the aortic sinuses and causing aortic valvar insufficiency on the basis of ring dilatation. Medionecrosis is a feature of Marfan's syndrome, but it may occur independently from other manifestations of this syndrome. Myxomatous involvement of the aortic valve causing valvar incompetence also occurs in Marfan's disease.

An angiocardiographic differentiation between these two mechanisms of valvar insufficiency in Marfan's disease will be possible rarely unless significant dilatation of the ascend-
aneurysms can cause aortic insufficiency if they are large enough to put traction on the aortic ring.\textsuperscript{10} Mycotic aneurysms, ankylosing spondylitis, systemic lupus erythematosus, Reiter's syndrome, pseudoxanthoma elasticum, and trauma are rare causes of aortic insufficiency.

The degree of aortic valvar insufficiency that can be tolerated by the left ventricle in terms of regurgitant fraction of the gross stroke volume is less than that of mitral valvar insufficiency. Arvidsson found the maximal regurgitant fraction to be 60% in aortic valvar insufficiency and 90% in mitral valvar insufficiency.\textsuperscript{18}

**Mitral Valvar Insufficiency**

Mitral valvar incompetence causes enlargement of the left ventricle and the left atrium. With increasing left atrial pressures, radiographic evidence of pulmonary venous hypertension is seen. The plain film radiographic changes of mitral valvar disease are demonstrated in figure 9. The left atrium is larger in cases of mitral valvar incompetence with or without valvar stenosis than in purely stenotic lesions. However, the degree of regurgitation cannot be determined by plain film estimation of atrial or ventricular size. Calcification of the mitral valve infers a rheumatic origin for the valvar disease, but does not aid in the identification of regurgitant valves as opposed to stenotic ones.\textsuperscript{22} Calcification of the mitral anulus is seen almost exclusively in elderly females and is often associated with mild to moderate valvar incompetence. However, calcified anulus also may be seen with normally functioning valves (fig. 10).

The angiocardiographic diagnosis of mitral valvar insufficiency rests upon the demonstration of contrast medium regurgitation from the left ventricle to the left atrium after selective injection of contrast medium into the left ventricle. A more specific diagnosis can be obtained by the analysis of the anatomic changes of the involved structures and the dynamics of the streaming blood.\textsuperscript{30} Complete anatomic demonstration is rarely possible because the anterior leaflet is almost completely surrounded by the U-shaped posterior leaflet, making separation in the tangential plane difficult, although the motion of the anterior leaflet is characteristic. Angiographically the mitral valve is best visualized in the right anterior oblique position.

Congenital and acquired lesions of the mitral valvar leaflets, as well as of the supporting structures, such as the chordae tendineae, the papillary muscles, and the left ventricular myocardium, may produce mitral valvar insufficiency. In addition, functional conditions, such as ectopic ventricular contractions, slow heart rate, and conduction abnormalities may render the valve incompetent in part of systole, probably because ventricular contraction occurs when the valve is open, which is not the case with normal ventricular contraction. The closure of the valve is then completed by the ventricular contraction. The angiocardiographic finding is characteristically demonstrated by a small initial puff of contrast medium entering the left atrium from the left ventricle. During the remainder of systole, the valve is competent, indicating that an organic lesion of the valve is not the cause of the insufficiency. As in the case of the tricuspid valve, pressure curve analysis and indicator dilution studies are not always conclusive in reaching the diagnosis.

Mitral valvar insufficiencies of differing geneses are
associated with angiographically demonstrated anatomic features which allow a specific diagnosis.

In rheumatic heart disease, the mitral valve becomes fibrotic and irregularly thickened, particularly along the free edges, preventing the leaflets from closing tightly in systole. The resulting regurgitation occurs between the shortened leaflets. Involvement of the chordae tendineae is common. Stenosis and insufficiency are often combined. In the latter case, a well defined regurgitant jet is often seen on angiocardiography.30

Infectious endocarditis most commonly occurs on a mitral valve which has been damaged by rheumatic endocarditis, particularly an insufficient valve.37 De novo infection of a healthy valve is distinctly uncommon, although involvement may occur by extension or engraftment from an infected aortic valve.

Angiographically, serial studies may show rapidly increasing valvar incompetence and deterioration of valvar function. Valvar aneurysms occasionally form during the course of the disease. These may be demonstrated angio-
graphically or, after their rupture, the resulting regurgitation can be shown.

As mentioned above, valvar incompetence may occur secondary to weakening or rupture of a chordae tendineae resulting from infection. Such infection of the chorda of the anterior leaflet may accompany aortic valvar infectious endocarditis.

A certain number of otherwise normal persons have echocardiographic and auscultatory findings of mitral valvar prolapse. A proportion of the patients with “floppy valve” syndrome also have mitral valvar insufficiency. Compilation of statistics on this problem is complicated by the lack of a clear definition of what constitutes a prolapsed mitral valve. For practical, reporting purposes, a prolapsed mitral valve is believed to be present when the mitral valve in systole protrudes beyond the plane of the mitral valve, this ring being defined as that part of the ventricle closest to the left atrium while still exhibiting active contraction in systole.

Localized protrusion of a part of the valve is referred to as scalloped mitral valvar leaflet. Small variations in the position and shape of the mitral valve in systole should probably be accepted as normal variations without prognostic significance.

Mitral valvar insufficiency resulting from ruptured chordae tendineae on an ischemic, infectious, or traumatic basis may display characteristic angiographic flow pattern. The loss of support to the anterior leaflet will direct the regurgitant flow downward behind the normally supported, posterior leaflet, and vice versa.

The rupture of a papillary muscle or chordae tendineae may produce ventricular contractions of a rocking character, probably brought about by the increased ventricular stroke volume and the lack of support of the ventricular myocardium by the ruptured papillary muscle. Coronary heart disease is often associated with slight to moderate mitral valvar insufficiency.

Ischemia, known to reduce the capacity of the myocardium to relax and to contract, does interfere with the closure of the mitral valve. However, the exact relationship between location and extent of myocardial damage on the one hand and the degree of mitral valvar insufficiency on the other is not clear. An akinetic or severely hypokinetic myocardial segment at the base of a papillary muscle may not cause any valvar insufficiency, while other seemingly less strategically located myocardial lesions may do so.

Endocardial cushion defects display varying degrees of mitral valvar insufficiency characteristically caused by a cleft in the anterior leaflet of the mitral valve, as well as abnormal attachment of the leaflet. The diagnosis of this condition is based on the demonstration of contrast medium regurgitation from the left ventricle to the left and right atria by way of the commonly associated atrial septal defect of primum type. The abnormal attachment of the anterior leaflet of the mitral valve creates a typical angiographic sign, the “gooseneck deformity” of the subaortic region of the left ventricle. The presence of this sign indicates that the mitral valvar insufficiency is caused by a cleft in the anterior mitral valvar leaflet, a feature which is often demonstrable by angiography.

In hypertrophic obstructive cardiomyopathy, the function of the mitral valve closure is interfered with primarily by anterior displacement of the free edge of the anterior leaflet in systole. This causes mitral valvar regurgitation which is present in practically all patients with a systolic intraventricular pressure gradient.

A normally functioning prosthetic mitral valve may show a minimal degree of insufficiency on angiocardiogram, whether the cardiac rhythm is regular or irregular. With multiplane projection, the regurgitant flow can be demonstrated to occur inside the ring of the prosthesis. If dehiscence is present, the regurgitant flow is seen outside the prosthetic ring. A combination of differential diagnostic interest is the formation of a dissecting wall aneurysm of the left atrium (fig. 11). The sutures of the prosthesis pull the inner wall of the atrium away from the outer wall, and a large cavity may form into which contrast medium from the left ventricle flows unhindered. The intramural cavity may simulate the left atrial lumen, but the lack of pulmonary venous flow into the cavity can usually be established without difficulty.

Pulmonic Valvar Insufficiency

Acquired pulmonic valvar insufficiency may be seen in carcinoid lesions, from surgical relief of pulmonary stenosis or as the result of bacterial attack on the valve. Most commonly, pulmonic valvar insufficiency occurs secondary to pulmonary hypertension, often resulting from mitral valvar disease. Acquired pulmonic valvar insufficiency is generally considered to be of little hemodynamic significance. However, no conclusive quantitative studies are available. Experimental studies showing little effect of the destruction of the anterior wall of the right ventricle may not be applicable, since a small, scarred right ventricle may be less deleterious to total cardiac function than a large, volume-overloaded right ventricle.

![Figure 11](https://i.imgur.com/3Q5zQ5z.png)  
**Figure 11.** Dissecting aneurysm of left atrium. Contrast medium regurgitates from left ventricle, around prosthetic valve (outlined by arrows) into the left atrial wall dissection.
The angiographic diagnosis of pulmonary insufficiency is illustrated in figure 12. The catheter tip is placed in the main pulmonary artery into which the contrast medium is injected. The contrast medium regurgitates into the right ventricle. For quantitation of regurgitant flow, the right ventricular stroke volume is measured, and from it is subtracted the effective forward stroke volume which is equal to the normal left ventricular stroke volume. The latter can be measured either by the Fick principle or by angiocardiography.

The right ventricular enlargement and dilatation of the main pulmonary artery which occur with pulmonic valvar insufficiency are rarely diagnostic on plain films because these changes are relatively small and nonspecific. For instance, a slight right ventricular enlargement, occurring after surgery for pulmonic stenosis, may be attributable to myocardial insufficiency or to reduced myocardial hypertrophy which facilitates diastolic filling.

Congenital anomalies of the pulmonic valve may result in valvar insufficiency. The degree of involvement of the valve varies from minor irregularities of the cusps (preventing them from closing completely) and small defects in the cusps to absence of the valve. In the latter event, the cusps may be completely absent or small residues of the cusps may be present. Marked ring stenosis is usually found at the valvar level. This combination of stenosis and insufficiency produces marked turbulence in the pulmonary artery. Depending upon the direction of the outflow tract toward the right or the left pulmonary artery, marked dilatation of these arteries occurs which often, but not always, can be detected on the chest film (fig. 13).

Patients with absent pulmonic valve often have a ventricular septal defect as well, and therefore the clinical presentation is that of tetralogy of Fallot. However, auscultation frequently reveals the pulmonic insufficiency. The right ventricle is usually larger than in tetralogy of Fallot, as the pulmonic valvar insufficiency causes volume overload of this chamber.

The pulmonic valve may be markedly incompetent in infants with large patent ductus arteriosus. It is usually assumed that this insufficiency results from dilatation of the main pulmonary artery with ensuing lack of cusp competence in diastole. The regurgitant flow occurs in some patients during systole, when the valve is normally open. The resulting enlargement of the right ventricle may exceed that of the left ventricle.

The Tricuspid Valve

Tricuspid valvar insufficiency may occur as a result of congenital anomaly or through destruction of the valve by infectious agents, rheumatic heart disease, trauma, or by carcinoid agents. The most common and perhaps least understood type of tricuspid insufficiency occurs secondary to pulmonary embolism, left sided cardiac lesions, such as mitral valvar disease, and left ventricular failure. The specific pathophysiological mechanisms which may explain this type of tricuspid valvar insufficiency include: 1) dilatation of the tricuspid valvar ring to such a degree that the leaflets only incompletely cover the valvar ostium in systole; 2) myocardial failure of the right ventricle, causing weak contractions and incomplete closure of the valve; 3) dilatation of the right ventricle, which may cause retraction of the chordae tendineae and the papillary muscles, thus interfering with their function; 4) atrial fibrillation which is known...
to increase the regurgitation, and slow heart rate which may in turn cause diastolic overflow.

The diagnosis of tricuspid valvar insufficiency by non-radiological means is based on the detection of a systolic murmur and an analysis of right atrial pressure curves. Indicator dilution techniques may be used as well. All of these methods have significant flaws. The systolic murmur may be difficult to differentiate from other cardiac murmurs. The height of the systolic wave, the so-called V wave, does not correlate well with the degree of valvar insufficiency since the compliance of the right atrium is the determining factor for the pressure development. Also, V waves may be found in the absence of tricuspid insufficiency when the patient is in right-sided heart failure. Because of poor mixing conditions in the right ventricle and right atrium, dye injected into the right ventricle and sampled in the right atrium may not give an accurate assessment of the degree of regurgitation.

None of these methods demonstrates such relevant features as the anatomy of the tricuspid valves, the volume and shape of the right ventricle, the size of the valvar ostium, nor the movement of the blood in the atrium and the ventricle. For example, it is not possible to differentiate between systolic and diastolic regurgitation.

In isolated tricuspid valvar insufficiency, the stroke volume of the right ventricle is increased over that of the left ventricle in an amount equal to the regurgitant flow per heart beat. To the extent that this increase in right ventricular stroke volume is accompanied by an increase in the right ventricular end-diastolic volume, a selective right ventricular enlargement will result and, if sufficiently large, can be detected on plain chest films. As the amount of blood flowing through the right atrium is increased by an amount equal to the regurgitant flow, the right atrium will also become enlarged. Figure 14 shows the chest film of a three-year-old girl with destruction of the tricuspid valvar leaflets by bacterial endocarditis and marked enlargement of both the right ventricle and the atrium. Calcification of the tricuspid valve is distinctly uncommon.

When the tricuspid regurgitation is marked, systolic pulsations of the right atrium and the superior vena cava may be seen by fluoroscopy. The angiographic demonstration of tricuspid valvar incompetence rests on the demonstration of flow of contrast medium from the right ventricle to the right atrium.

Ebstein's malformation of the tricuspid valve is a congenital cause of tricuspid valvar incompetence. The demonstration of the tricuspid valvar anatomy is more significant than the angiographic demonstration of insufficiency. The valvar leaflets are displaced and inserted into the ventricular wall and septum more apically than is normal. The valve may form a sac-like structure which, in the extreme case, may reduce the size of the non-atrialized portion of the right ventricle to a slit-like structure, and the tricuspid valvar orifice may be small, producing subinfundibular stenosis. However, in most instances, the tricuspid valvar distortion is

**Figure 14.** Serial films (A before, B after) of a patient with tricuspid bacterial endocarditis.
left ventricle to the right atrium without admixture of the blood contained in the right ventricle.

In endocardial cushion defect, the tricuspid valve may be insufficient because of the congenital defect of the septal leaflet of the valve. This is a phenomenon parallel to the cleft mitral valve in this condition, but less frequently demonstrated.

Rheumatic involvement of the tricuspid valve is rare. It is said to occur in 10% of the patients in whom other rheumatic cardiac valvar lesions are found.

Although right-sided infectious endocarditis is rare, tricuspid valvar endocarditis does occur, particularly in drug addicts, infants, and postcardiac surgery patients. A common associated finding is the presence of multiple septic pulmonary emboli. Serial angiographic studies will show rapid destruction of the valve with maintenance of relatively normal valvar motility and thickness.

Carcinoid heart disease typically affects the tricuspid and pulmonic valves, leading to severe insufficiency.49

Extrasystoles, as well as slow heart rate and conduction abnormalities like total heart block, may produce functional tricuspid valvar regurgitation.

References

5. Larsson H, Kjellberg SR: Roentgenological heart volume determinations in patients with the special regard to pulse rate and the position of the body. Acta Radiol 29: 159, 1948
20. Graham TP Jr, Jarmakani JM, Cavenet RV Jr: Left heart volume characteristics with a right ventricular volume overload. Total anomalous
venous connection and atrial septal defect. Circulation 45: 144, 1972
44. Barr PA, Celermaier JM, Bowdler JD, Cartmill TB: Severe congenital tricuspid incompetence in the neonate. Circulation 49: 962, 1974
The radiological diagnosis of cardiac valvar insufficiencies.
E Carlsson, R Gross and R G Holt

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