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# SECOND SYMPOSIUM ON CARDIOVASCULAR SOUND

**Guest Editor: Victor A. McKusick, M. D.**

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This series of papers constituted the second Symposium on Cardiovascular Sound conducted in association with the Scientific Sessions of the American Heart Association. The symposium took place on Friday, October 25, 1957, at the Hotel Sherman in Chicago. The chairman of the morning sessions was Dr. Hans H. Hecht of Salt Lake City; Dr. J. Willis Hurst of Atlanta was chairman for the afternoon sessions. The first symposium was published in *Circulation* 16: 270, and 414, 1957. This publication was made possible by a grant-in-aid from the National Heart Institute, U.S. Public Health Service, and by contributions from Mr. Arthur L. Humphries, The Rena and Walter Burke Foundation, Lakeside Laboratories, Burroughs-Wellcome Company, and the Wyeth Laboratories.

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## Endocardial and Intimal Lesions (Jet Impact) as Possible Sites of Origin of Murmurs

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**U**NDER certain circumstances, abnormal cardiovascular dynamics cause structural changes which remain in the necropsy specimen to invite postdictive interpretation of their relation to previously existing functional derangements. Among the more graphic of these imprints are the "jet" lesions. These are focal fibrous reactions in the linings of the heart or blood vessels which apparently result from trauma by repeated impacts of abnormal jetlike streams of blood.

The sites of such impacts are located where high-velocity streams of blood emanating from a high-pressure source and entering a relatively low-pressure compartment are suddenly either stopped or deflected. The vibration of the area and associated turbulence in the blood might be expected to cause a murmur. Since the jet lesions locate the sites of impact, they may be utilized as a tool for fruitful investigation when their position in the specimen is correlated with the other ana-

tomic defects and the location of the previously recorded murmurs in the patient.

This presentation describes the positions of jet lesions encountered in a variety of cardiac and vascular disorders usually associated with stenotic orifices. Particular emphasis is placed on the variation in the positions of the jet lesions that have occurred in any one category of hemodynamic disturbance.

The variations in anatomic location of jet lesions at times correlate well with variations in position of maximal intensity of the murmurs for that condition during life.

The jet lesions, in addition to labeling the sites of possible origin of bruits, may be helpful in determining the previous pressure or the absence of regurgitant flow through a valve in the direction of blood flow that had existed in abnormal communications during the life of the patient.

### NATURE OF JET LESIONS

The lesions of jet impacts have been variously designated as pockets of Zahn,<sup>1</sup> impingement plaques, endocardial pockets,<sup>2-4</sup> regurgitant lesions, frictional lesions,<sup>5</sup> and jet lesions.

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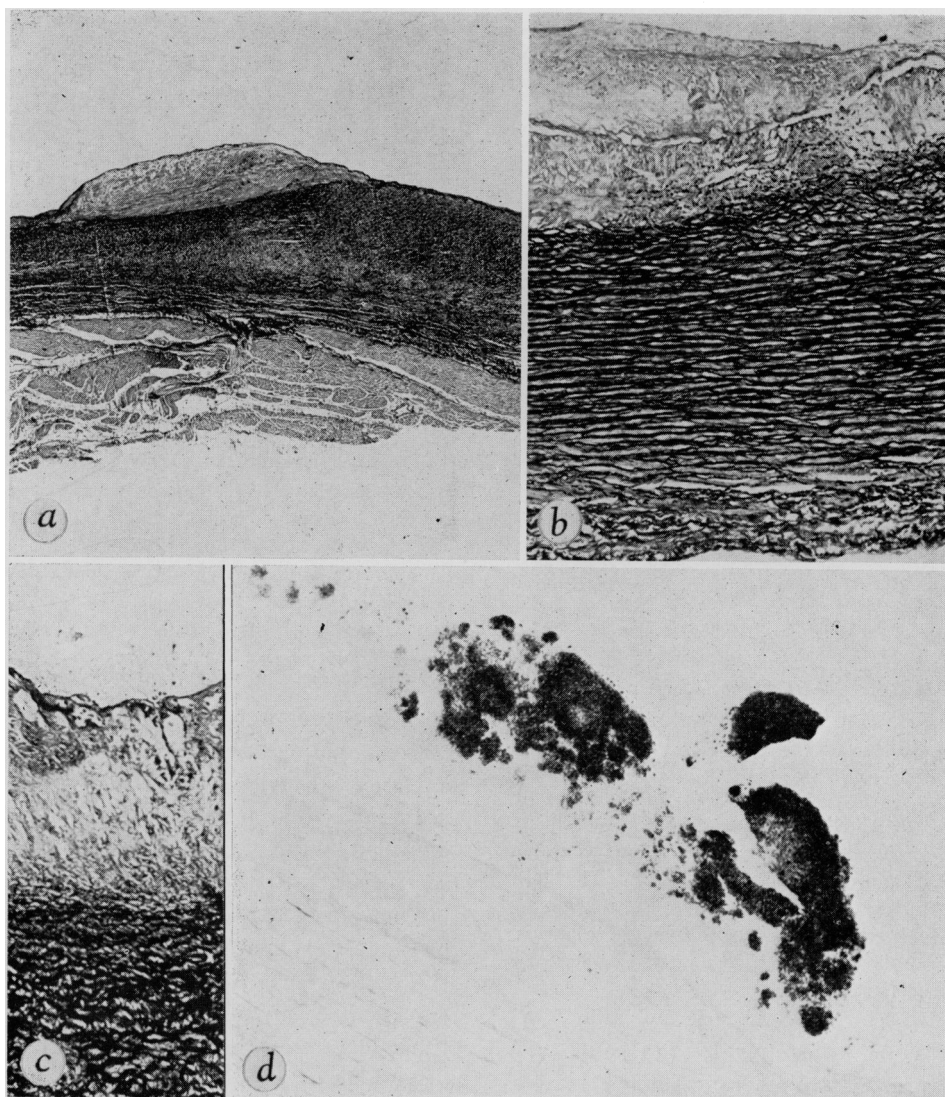


FIG. 1. Photomicrographs of jet lesions. *a.* Left atrium in a case of mitral insufficiency. Endocardial thickening by nonspecific fibrous tissue (Verhoeff's elastic-tissue stain, counterstained with van Gieson's connective-tissue stain;  $\times 15$ ). *b.* Ascending aorta in a case of aortic stenosis (ELVG;  $\times 55$ ). *c.* Pulmonary trunk in a case of tetralogy of Fallot with pulmonary valvular stenosis. Nonspecific fibrous-tissue thickening of intima. At the surface of the lesion there is a deposit of platelets, fibrin and occasional leukocytes (ELVG;  $\times 90$ ). *d.* Mitral valve in a case of aortic stenosis and insufficiency with bacterial endocarditis. The jet lesion on the subjacent mitral valve is infected and is composed to a large extent of colonies of bacteria (Brown Gram stain;  $\times 140$ ).

Conditions which may be associated with jet lesions are the various kinds of valvular stenosis or insufficiency, stenosis of ventricular outflow tracts, certain septal defects, and abnormal communications between the aorta or its branches and a cardiac chamber, a pulmonary artery or a vein.

Grossly, jet lesions appear as focal crops of thickenings of the endocardium or of the intima of involved blood vessels. There is a tendency, especially when the stream apparently strikes the wall obliquely, for the components of the jet lesion to have a cusplike orientation. The cusps formed are directed



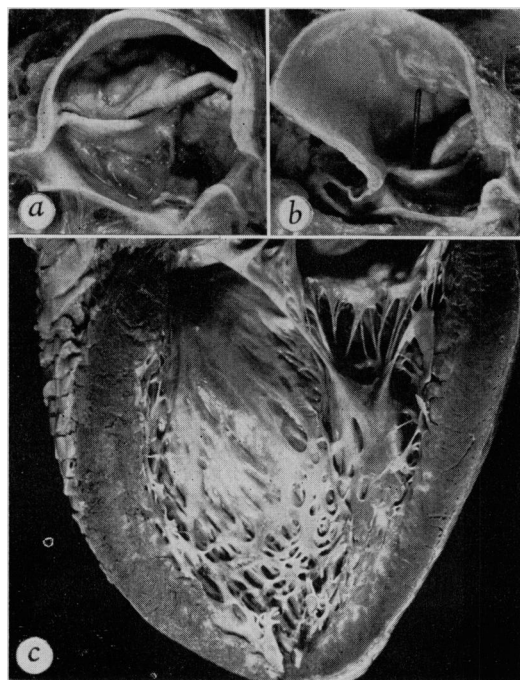


FIG. 2. Congenital bicuspid aortic valve with stenosis and insufficiency. *a*. The aortic valve from above. Congenital bicuspid valve with calcification causing irregularity responsible for both stenosis and insufficiency. *b*. Above the valve the probe points to a jet lesion on the ascending aorta (photomicrograph of this lesion appears as figure 1*b*). *c*. The left ventricle. At the apical portion there is widespread fibrous thickening of the endocardium representing jet-lesion formation from the element of insufficiency of the diseased aortic valve.

with their concavities toward the source of the jet stream. Histologically, jet lesions are composed of nonvascular fibrous tissue, mainly collagenous, although in old lesions some elastic tissue may be found as well (fig. 1*a* and *b*).

The usual absence of blood vessels in the jet lesion indicates that it appears to be a primary overgrowth of connective tissue of the lining of the involved vessel or cardiac chamber. In some instances, however, there are deposits of platelets and fibrin on the surface of the lesion suggesting that at times some of the plaque or excrescence may result from a process of organization of such surface deposition of blood elements (fig. 1*c*). There is no atheromatous material in the jet lesion, although, at times, jet lesions in arteries have

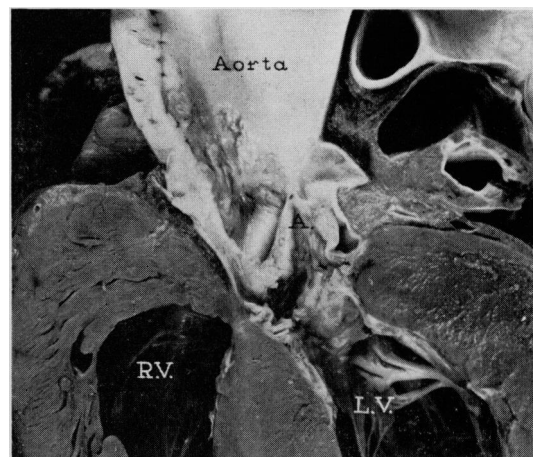


FIG. 3. Subaortic stenosis in a 14-year-old boy. Aortic valve and adjacent areas. Sagittal section of heart. Beneath the aortic valve (*A*), the outflow tract of the left ventricle (*LV*) shows stenosis characteristic of subaortic stenosis. On the aortic valve and on the ascending aorta there are jet lesions. *RV* = right ventricle.

been misinterpreted as representing atheromatous plaques.

Under special circumstances wherein there is intravascular infection at the site of origin of a jet stream, bacteria may be responsible for infection of the wall of the cardiac chamber or blood vessel at the site of the impact by the jet stream. Under these circumstances, the jet lesion has a different appearance. It is represented by an accumulation of vegetations characteristic of bacterial endocarditis or endoangiitis at the site of the impact (fig. 1*d*). Depending upon circumstances, such infectious jet lesions may be primary or secondary. They are primary when, for example, the insufficiency of a valve has resulted from a bacterial infection. On the other hand, when bacterial infection complicates the existence of a stenotic or insufficient lesion, then jet lesions of the inflammatory type are secondary, being superimposed on pre-existing fibrous elevations which represent jet lesions that had developed in the stages of cardiac dysfunction before the inflammatory disease appeared.

Most of the lesions with which we are concerned resulted from purely mechanical rather than infectious factors.

## SITES OF JET LESIONS

*Aortic Stenosis.* Frequently, in aortic valvular stenosis, a jet lesion is present on the wall of the ascending aorta.

The natural direction of blood passing through the aortic valve obliquely toward the right is responsible for the fact that in many instances of aortic valvular stenosis, the jet lesion involving the ascending aorta lies along the right aspect of this structure, beginning a short distance above the aortic valve. At times the jet lesion lies upon the surface of the posterior aspect of the ascending aorta, on that part of the aorta which lies in contact with the anterior aspect of the atrial septum and the adjacent anterior walls of the atrial chamber (fig. 2). When the tendency for the stream to strike the posterior wall occurs, it is believed to result from peculiarities of the valvular disease in the particular case rather than from the primary direction of blood flow leaving the left ventricle.

*Subaortic Stenosis.* In subaortic stenosis, jet lesions occur not only upon the right wall of the ascending aorta but also upon the ventricular aspect of the aortic valve (fig. 3). One is led to wonder whether the valvular jet lesions in this disease result in part from the trauma of the jet stream striking the closed aortic valve in the earliest part of systole just before the aortic valve opens. After the valve opens, the jet may traumatize the wall of the ascending aorta and cause a lesion there. In addition, the hemodynamic situation exists wherein intense fluttering of the valve leaflets about the high-velocity stream might be expected.

*Aortic Insufficiency.* Mural jet lesions in the left ventricle are common in aortic insufficiency of various types.

When left ventricular jet lesions occur as a result of incompetence of the aortic valve, whatever its cause, the lesions lie closely subjacent to the aortic valve, but they occupy a variety of foci in this region. A common site for a left ventricular jet lesion is the ventricular surface of the anterior leaflet of the mitral valve, as we have seen in some cases of congenital bicuspid aortic valve with aortic in-

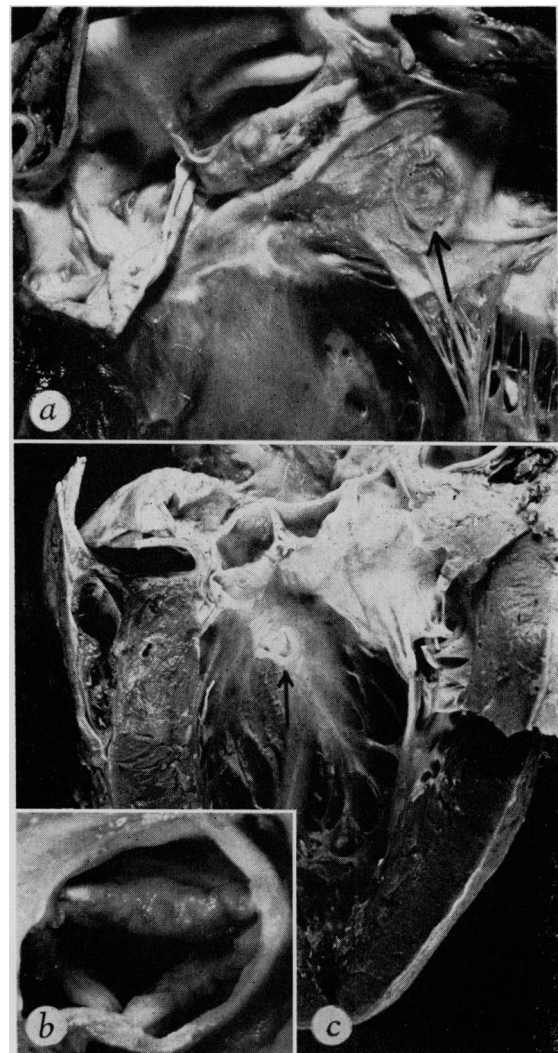


FIG. 4. Aortic insufficiency. *A.* Congenital bicuspid aortic valve in a 28-year-old man with aortic coarctation. Jet lesion is present on the ventricular surface of anterior mitral leaflet (*above point of arrow*). *b.* and *c.* A case of rheumatic aortic insufficiency in an adult. *b.* Aortic valve seen from above. In *c.* beneath the opened aortic valve, a jet lesion (*point of arrow*) is seen on the left ventricular surface of the ventricular septum.

competence (fig. 4*a*). Another common site is the surface of ventricular septum subjacent to the aortic valve. In rheumatic aortic insufficiency, especially when associated with aortic stenosis, the tendency is great for a jet lesion to be found in this location (fig. 4*b* and *c*). We have also observed lesions in this position in syphilitic aortic insufficiency and



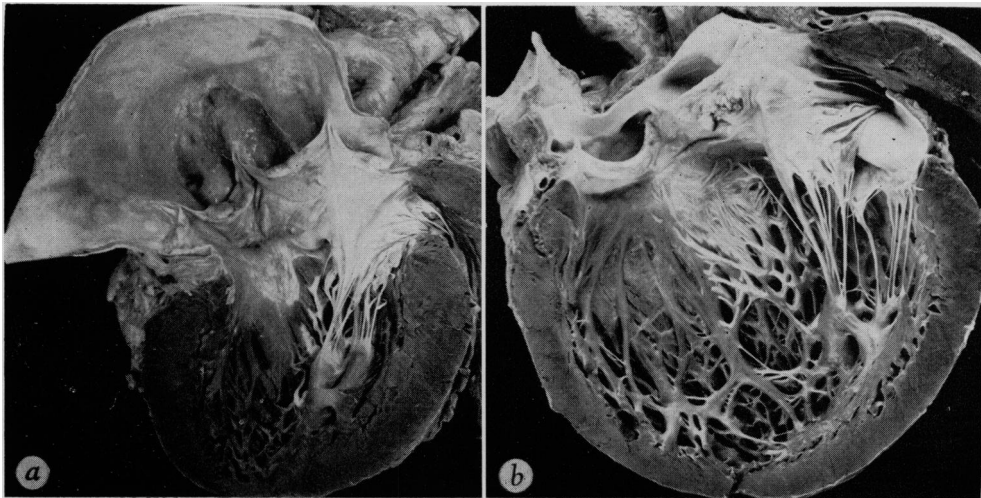


FIG. 5. *a.* Left ventricle and ascending aorta. There is senile dilatation of the aorta. A jet lesion resulting from aortic insufficiency is present on the ventricular septum immediately beneath the aortic valve. *b.* Left ventricle and aortic valve in a case of congenital bicuspid aortic valve with healed bacterial endocarditis. There is perforation of one leaflet, and jet lesions are present on the ventricular septum inferior to the perforated leaflet.

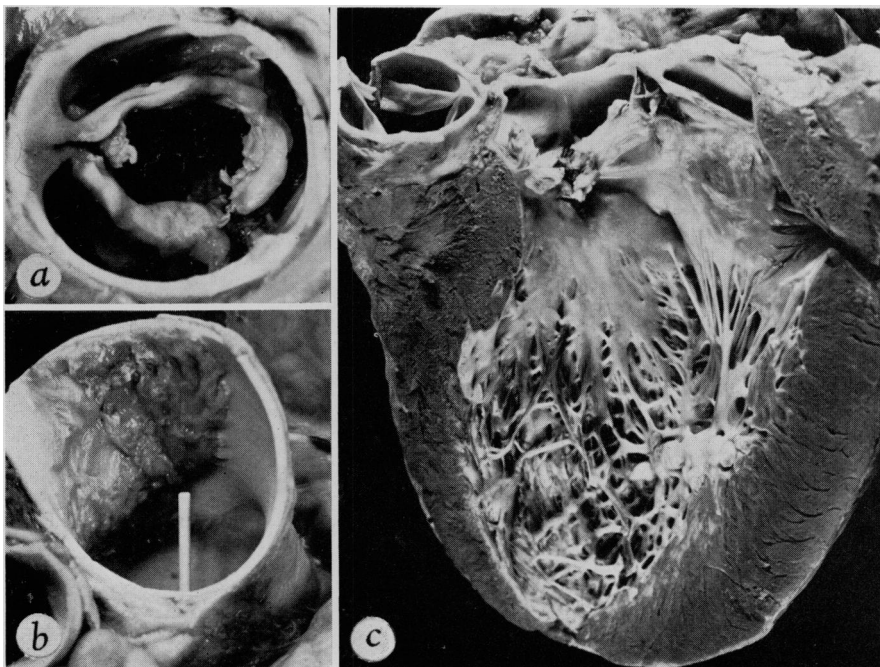


FIG. 6. Congenital aortic stenosis and insufficiency in a 16-year-old girl. *a.* The aortic valve from above, showing congenital stenosis and insufficiency. Valvulotomy had been done on this valve. *b.* Ascending aorta. A jet lesion (above probe) is seen on the right posterior lateral wall of the ascending aorta. *c.* The aortic valve and left ventricle opened. The apical half of the left ventricle shows extensive endocardial scarring representing jet-lesion formation as a result of the aortic incompetence.

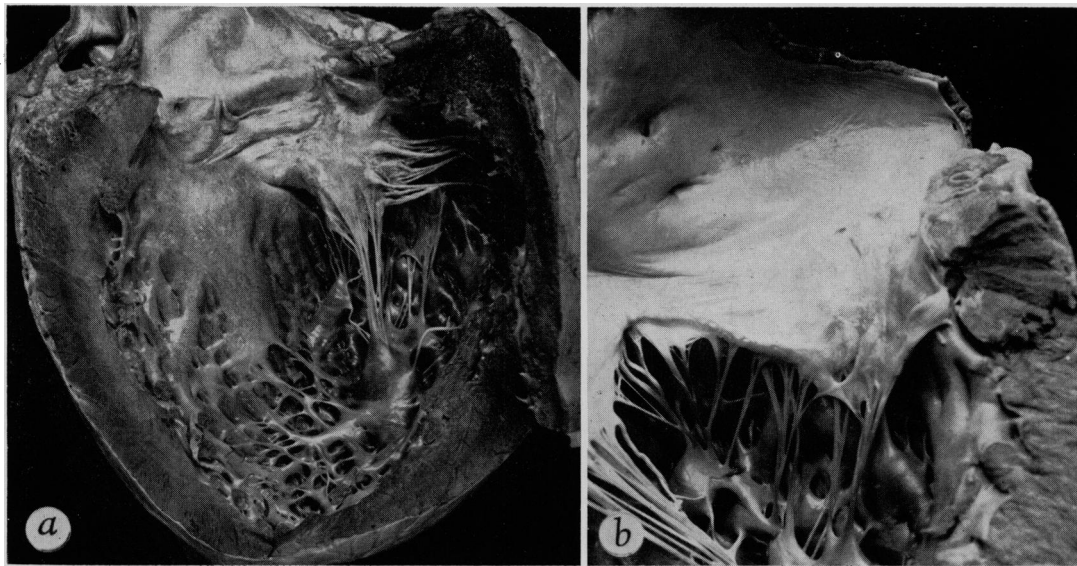


FIG. 7. Rheumatic aortic insufficiency in a young man with complicating mitral insufficiency. *a.* Aortic valve and left ventricle. There is left ventricular enlargement as a consequence of rheumatic aortic insufficiency. *b.* The mitral valve and the left atrium. The posterior leaflet of the mitral valve and the adjacent left atrial wall show jet lesions resulting from mitral insufficiency. The latter appears to have complicated the state of enlargement of the left ventricle with tension on the mitral chordae.

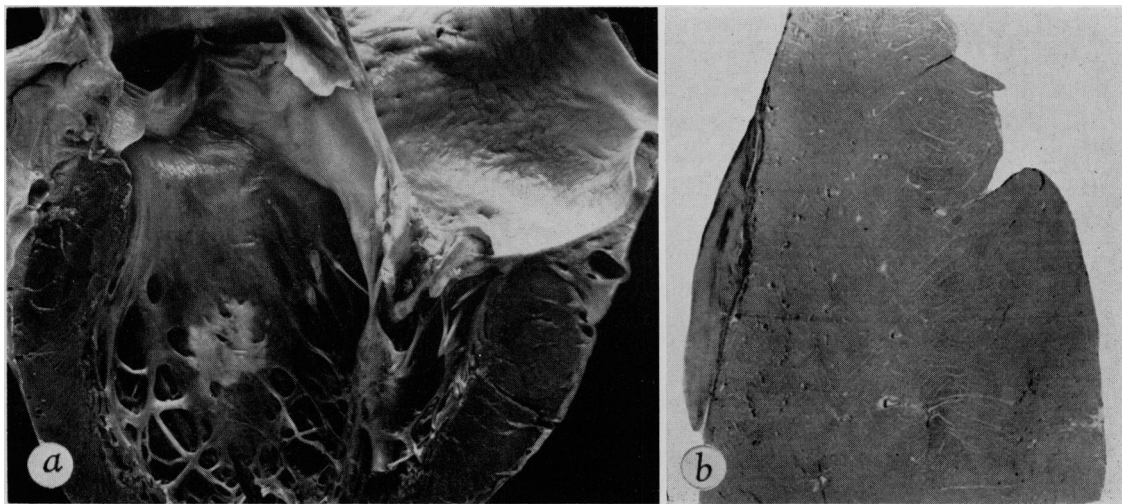


FIG. 8. Mitral stenosis with jet lesion on septal wall of left ventricle. *a.* Gross specimen in sagittal section. *b.* Photomicrograph (ELVG;  $\times 3$ ).

in a rare case of aortic incompetence which appeared to have resulted from senile dilatation of the aorta (fig. 5*a*).

In the relatively uncommon situations when left ventricular jet lesions occur below the upper third of the chamber, the lesions tend to be broadly distributed in contrast to the

relatively confined sites of involvement seen, as a rule, in the upper third of the chamber.

In a case of congenital bicuspid aortic valve with healed bacterial endocarditis which we studied, there was a perforation in the more posterior of the two aortic cusps. The central portion of the midseptal wall of the left



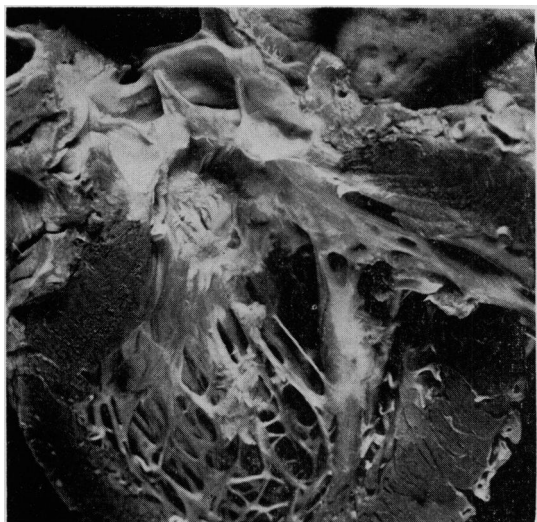


FIG. 9. Coexistent mitral stenosis and aortic insufficiency. The septal wall of the left ventricle shows two jet lesions. The first, resulting from the aortic insufficiency, lies immediately beneath the aortic valve. The second, resulting from the mitral stenosis, lies about midway between the apex and the base of the septum.

ventricle showed a large patch of fibrous thickening resulting from the aortic regurgitant stream (fig. 5*b*).

In those cases of congenital, coexisting, aortic valvular stenosis and insufficiency that we have observed, the entire lower half of the left ventricular endocardium was thickened irregularly by collagen, a process interpreted as jet-lesion formation (fig. 6). A similar process was observed in a case of congenital bicuspid aortic valve which had become involved by acquired stenosis and insufficiency. The possibility of these lesions being a focal endocardial fibroelastosis is not admitted since the tissue of the lesion is collagenous rather than containing wavy deposits of elastic tissue.

Additional mention should be made of aortic insufficiency associated with active bacterial endocarditis. Here there is a great tendency for the regurgitant streams to strike the anterior mitral valve and its attached chordae. Under these circumstances infection develops in the structures receiving the impact. Not infrequently aneurysm of the valve leaflet may occur and if the secondary inflammatory lesions are sufficiently destructive, perforation of the anterior mitral leaflet may

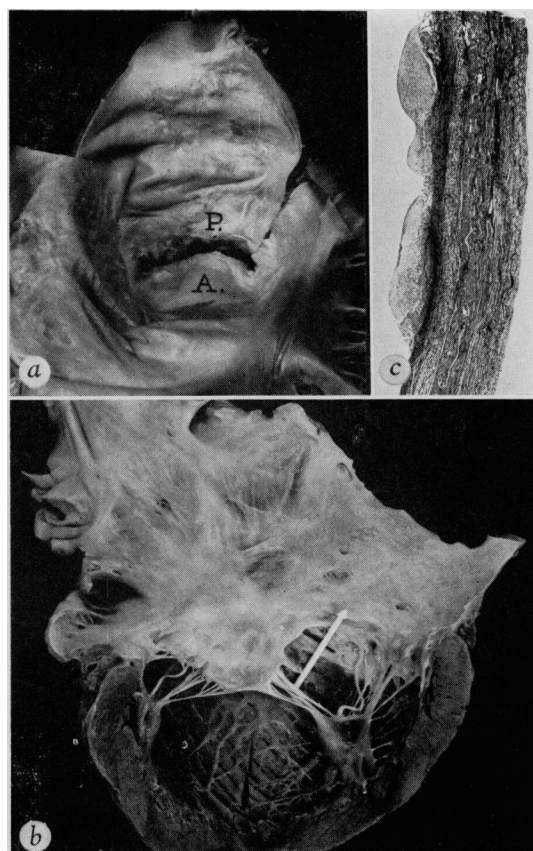


FIG. 10. Mitral insufficiency. *a*. Rheumatic mitral insufficiency. Unopened valve is viewed from above. Above the posterior leaflet (*P*) jet lesions are on the posterior wall of the left atrium. *A* = anterior mitral leaflet. *b*. and *c*. Healed bacterial endocarditis. *b*. Rupture of chordae and erosion of valvular tissue are evident in relation to the posteromedial commissure. The posterior wall of the left atrium shows an area of jet lesions (above arrow). *c*. Photomicrograph of jet lesions (ELVG;  $\times 9$ ).

follow. More commonly, however, rupture occurs in those chordae which insert into the central part of this mitral leaflet. The resulting mitral insufficiency complicates the picture, and left atrial jet lesions develop.

While mitral incompetence logically may follow the destructive complications of aortic valvular bacterial endocarditis, there is yet another circumstance in which mitral incompetence may complicate aortic insufficiency. In a case which we observed, a young man had severe pure aortic insufficiency of rheumatic origin. No jet lesions were present in the left ventricle, but such lesions were pres-

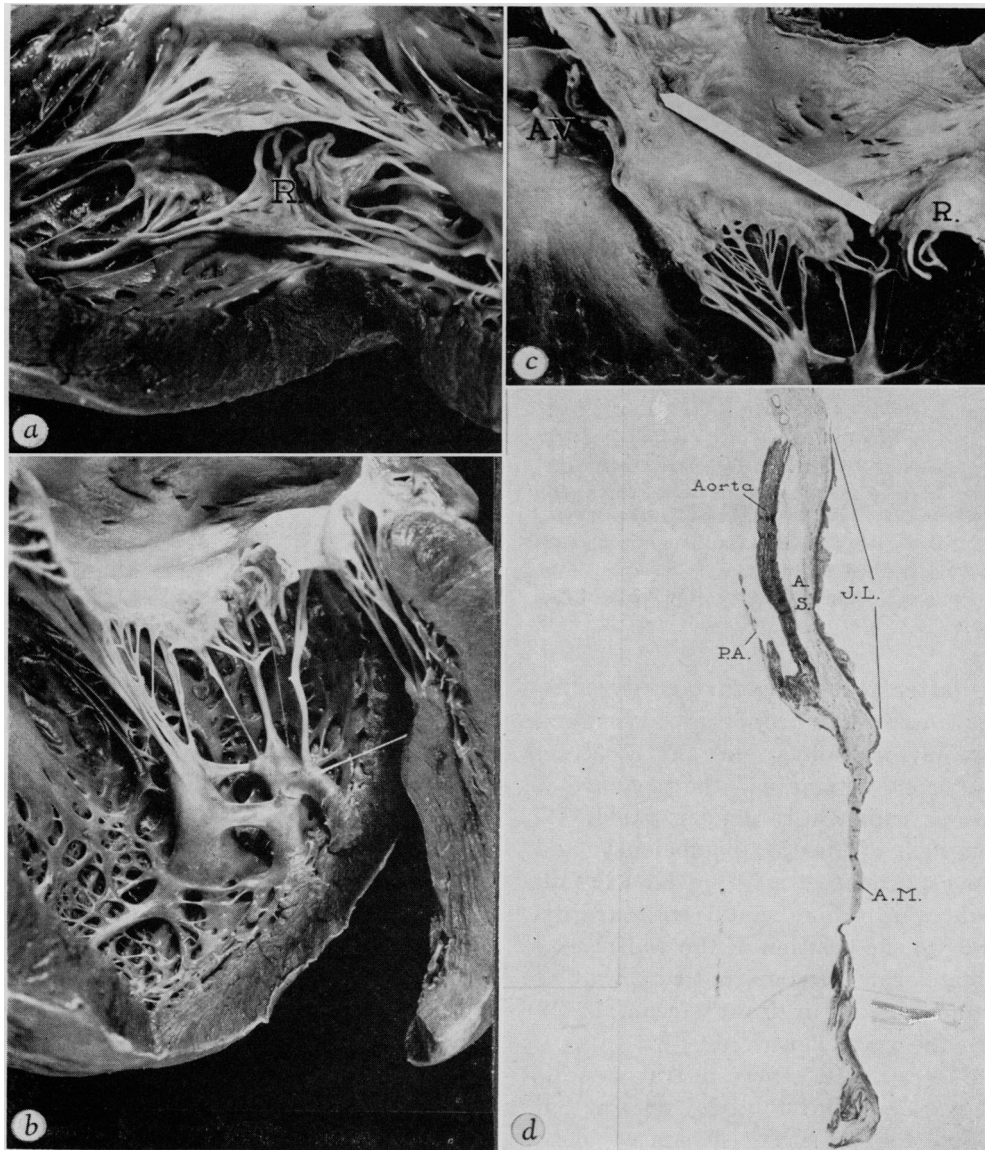


FIG. 11. Mitral insufficiency resulting from rupture of chordae attached to posterior leaflet in a 24-year-old man. *a*. Mitral valve unopened, viewed from below. Chordae to a portion of the posterior leaflet (*R*) have ruptured. *b*. Mitral valve opened. That part of the valve from which the chordae have ruptured shows a hoodlike deformity. *c*. Sagittal section of a portion of the left side of the heart. Probe shows path of regurgitant stream which had originated at the site of the ruptured chordae (*R*) and then struck the septal wall of the left atrium. The aortic valve (*AV*) lies close to the site of impact by the regurgitant stream. An artifact is seen beneath the base of the probe. *d*. Low-power photomicrograph of aortic valve, atrial septum and related structures from a case similar to that illustrated in *a*, *b* and *c*. The jet lesions (*JL*) on the atrial septum (*AS*) lie close to the posterior cusp of the aortic valve (*PA*). *AM* = anterior mitral leaflet (ELVG;  $\times 2$ ).

ent upon the atrial aspect of the posterior mitral leaflet and upon the contiguous portion of the left atrium (fig. 7). The mitral valve was otherwise normal. The left ventricle was

greatly dilated and elongated. We interpreted these changes as probably resulting primarily from the aortic insufficiency and causing undue tension upon the mitral chor-



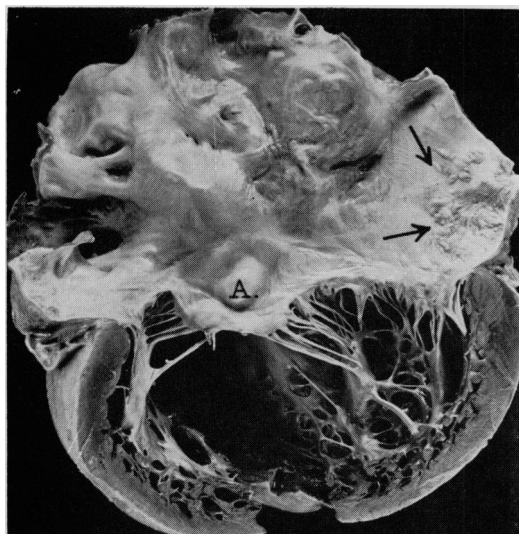


FIG. 12. Mitral insufficiency resulting from rupture of chordae attached to anterior leaflet (A). The jet lesions (arrows) lie on the posterior wall of the left atrium.

dae. The latter feature is further hypothesized to have caused improper approximation of the two mitral leaflets, and the resulting mitral insufficiency explained the presence of the jet lesions which were demonstrated.

The complex of aortic insufficiency and ventricular septal defect may yield jet lesions on the right ventricular septal endocardium just inferior to the position of the ventricular septal defect. Such lesions indicate that at least part of the regurgitant stream is directed into the right ventricle.

**Mitral Stenosis.** In severe mitral stenosis associated with a high left atrial pressure, it is conceivable that a jetlike stream of blood would enter the left ventricle during diastole of this chamber. That this occurs is evidenced by the fact that in an occasional case of mitral stenosis a jet lesion may be identified in the left ventricle. Such lesions may be found on the surfaces of the papillary muscles or on the ventricular septum opposite the mitral orifice (fig. 8). Our experience indicates that it is the exceptional case of mitral stenosis that exhibits such traumatic lesions in the left ventricle.

Several factors may explain the absence of left ventricular jet lesions in mitral stenosis. If the orifice is relatively large or if the left

atrial pressure is not particularly high, a high-velocity stream does not exist and so no basis for unusual trauma to the left ventricular chamber is present. Another factor may be the orientation of the mitral-valve orifice. In those cases in which a jet lesion occurred on the ventricular septum, the mitral orifice was so positioned as to suggest that the blood flowed in a somewhat horizontal direction toward the septum. When jet lesions were absent, the orientation of the mitral orifice was such as to suggest that the stream of blood flowed into the left ventricle in a vertical direction toward the anatomic apex of this chamber. It is conceivable that in the latter type of case the kinetic energy of the jet stream is dissipated in the left ventricular cavity and so does not impact against the left ventricular endocardium.

Of special interest are rare cases of mitral stenosis associated with aortic stenosis and insufficiency when left ventricular jet lesions occur from involvement of both valves. In a case which we observed, there was such a lesion on the septal wall immediately below the aortic valve which we interpreted as resulting from the aortic insufficiency. Below this lesion the septal wall contained a second and distinct patch of fibrous tissue caused by the mitral stenosis (fig. 9).

**Mitral Insufficiency.** Mitral insufficiency of rheumatic origin is not frequently associated with distinct jet lesions in the left atrium. This may possibly be explained by the direction of the regurgitant stream into the cavity of the left atrium, wherein the energy of the stream is dissipated. In those cases of rheumatic mitral insufficiency in which jet lesions are identifiable, they usually occur on the posterior wall of the left atrium immediately above the posterior leaflet of this valve (fig. 10a).

Mitral insufficiency resulting from bacterial endocarditis has a varied picture. In instances in which there is excavation of tissue of either leaflet, the jet lesions appear on the posterior wall of the left atrium (fig. 10b and c). A particularly interesting group of cases consists of those in which the bacterial endocarditis is associated with rupture

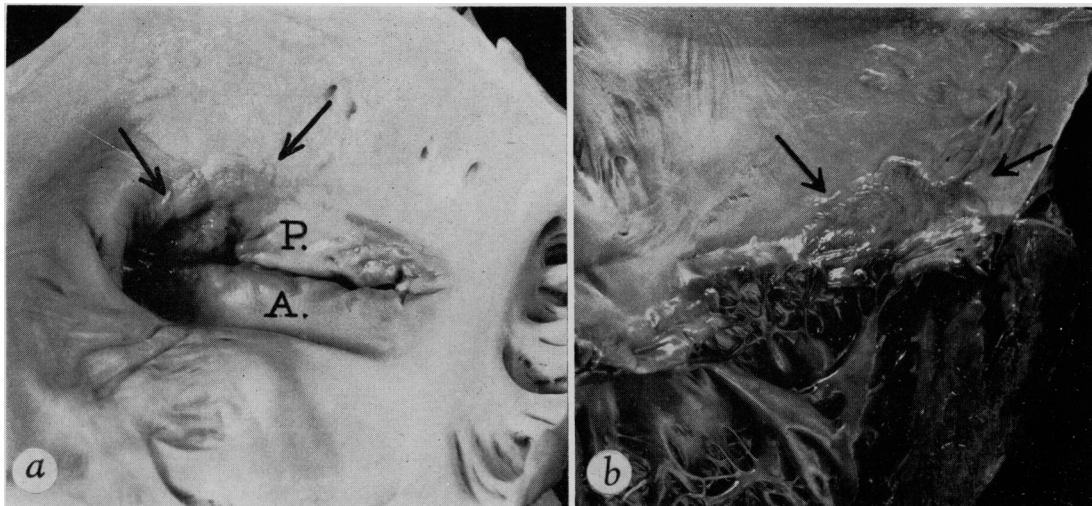


FIG. 13. Congenital mitral insufficiency in a 5-year-old child who also had a ventricular septal defect. *a*. The mitral valve is viewed from in front and above. In the posterior leaflet (*P*) there are two deficiencies. Above the larger there is a fan-shaped jet lesion (*between arrows*) on the posterior wall of the left atrium. *A* = anterior leaflet of mitral valve. *b*. The valve opened. Above the larger congenital deficiency of the posterior leaflet the jet lesion (*between arrows*) lies on the posterior wall of the left atrium.

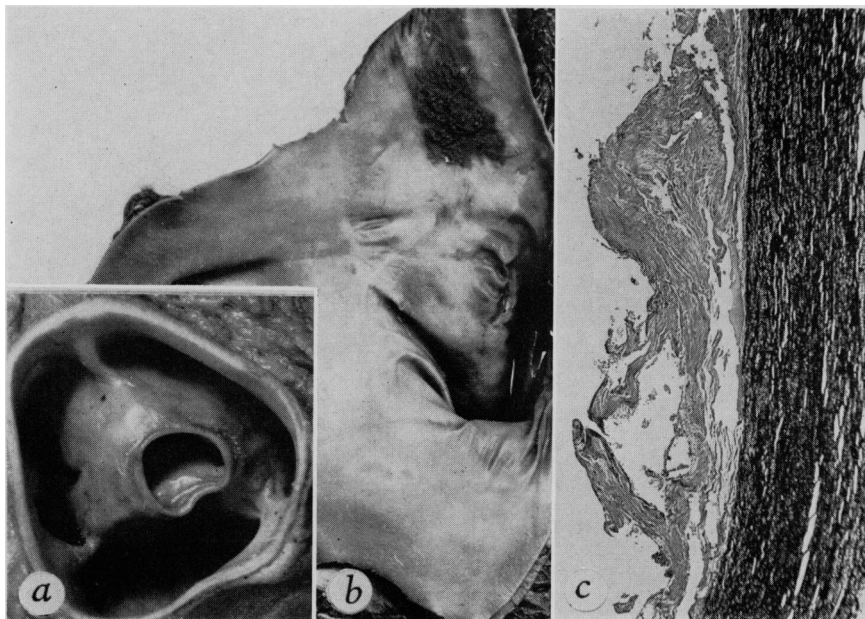


FIG. 14. Pulmonary valvular stenosis with intact ventricular septum. *a*. The unopened pulmonary valve from above. *b*. At the origin of the left pulmonary artery near the bifurcation of the pulmonary trunk (right upper portion of illustration) is an elevation of the intima which is a jet lesion. *c*. Photomicrograph of the jet lesion shown in *b* (ELVG;  $\times 30$ ).

of chordae. Usually, if the patient survives such an episode, the extent of ruptured chordae is restricted to a relatively small region. This creates a change of the valve in which, at the site of the ruptured chordae, the valve

leaflet protrudes into the atrium as a hood-shaped structure. This hoodlike deformity seems to have an influence in deflecting the blood to that part of the left atrium which lies opposite the site of the ruptured chordae.



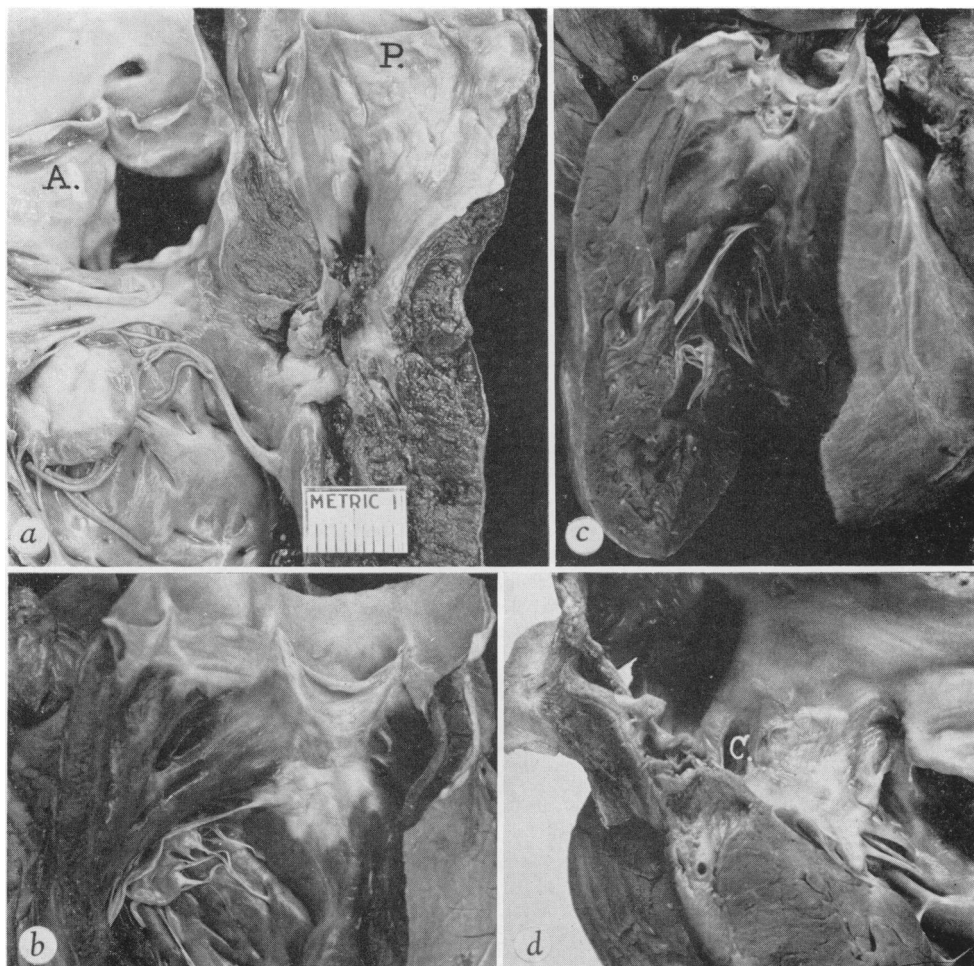


FIG. 15. *a.* Outflow tract of right ventricle and pulmonary valve in tetralogy of Fallot with severe infundibular stenosis. Above marked narrowing of the lower ostium of the infundibulum, where endocardial thickening of a considerable degree is present, there is a series of jet lesions involving the wall of the infundibulum and the adjacent leaflets of the pulmonary valve (*P*). *A* = aortic valve. *b.* Rheumatic pulmonary valvular insufficiency. Jet lesion beneath valve. From a 61-year-old man who also had involvement of the aortic and mitral valves. *c.* Pulmonary valvular insufficiency in an adult with patent ductus arteriosus and pulmonary hypertension. Jet lesion is present over a wide area immediately beneath the valve. *d.* Tricuspid insufficiency in an adult with pulmonary valvular stenosis and intact ventricular septum. A jet lesion can be seen on the septal wall of the right atrium just anterior to the ostium of the coronary sinus (*C*).

Thus, in rupture of the chordae to the posterior leaflet, the direction of the stream is forward and medially. The stream impacts against the atrial septum. In this location the lesions are removed from the base of the aorta by only a few millimeters (fig. 11). We have observed that the systolic murmur and thrill created by impingement of the regurgi-

tant stream against the atrial septum may yield clinical signs that may mimic aortic stenosis.

When rupture of chordae involves those attached to the anterior mitral leaflet, the blood is deflected posterolaterally where it strikes the posterior wall. We observed a case in which bacterial endocarditis had

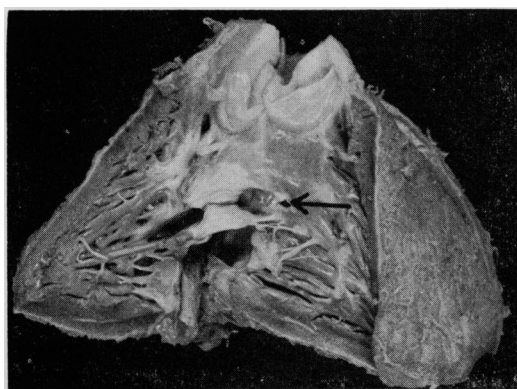


FIG. 16. Right ventricle in a case of small ventricular septal defect (arrow) in a 5-year-old boy. Jet lesion is on anterior wall of chamber.

started primarily on the aortic valve. This resulted in minor degrees of erosion of this valve. There was regurgitation onto the anterior mitral valve which showed a jet lesion on an unruptured aneurysm. Additionally, subjacent chordae which inserted into the central portion of the anterior leaflet had ruptured, causing the mitral valve to become incompetent. The jetlike stream from the mitral incompetence caused lesions on the posterior wall of the left atrium (fig. 12). In this patient, the systolic murmur was transmitted to the left axilla and back.

There is a large variety of congenital malformations of the mitral valve which lead to its incompetence. One of these is characterized by the presence of accessory chordae and commissures of the posterior leaflet. The deformity is responsible for inadequate apposition of one portion of the posterior leaflet upon the other with resulting incompetence through the deficiency. In such instances, the jet lesions appear directly above the deficiency in the posterior leaflet, upon the posterior left atrial wall (fig. 13).

**Pulmonary and Infundibular Stenosis.** Jet lesions associated with pulmonary or infundibular stenosis are most graphically observed in cases of congenital pulmonary valvular stenosis with intact ventricular septum. The jet stream strikes the top of the pulmonary arterial bifurcation or the nearby origin of the left pulmonary artery (fig. 14). In this regard it is significant that the systolic mur-

mur and thrill characteristic of this condition are often observed most intensely, not at the level of the pulmonary valve, but some distance above it.

In those cases of tetralogy of Fallot in which the major obstruction lies at the pulmonary valve, jet lesions occur on the wall of the pulmonary trunk, the exact position being determined by the orientation of the plane of the stenotic orifice.

In cases of tetralogy of Fallot wherein the major obstruction lies in the right ventricular infundibulum, lesions of the jet type are found on the ventricular face of the pulmonary valve. In particular when an infundibular chamber occurs beyond a point of severe localized infundibular stenosis, the wall of the chamber may likewise show such lesions (fig. 15).

**Pulmonary Insufficiency.** When pulmonary valvular insufficiency occurs, jet lesions may be found immediately inferior to the valve. Usually these lesions are represented by broad areas of relatively minor endocardial thickening (fig. 15*b* and *c*).

**Tricuspid Insufficiency and Stenosis.** Rheumatic involvement of the tricuspid valve usually is not associated with either right ventricular or right atrial jet lesions. This may perhaps be explained by relatively minor differences in pressure between the two chambers concerned and also by the fact that the orifice, although narrowed, is still of relatively good size.

In an occasional case in which a severe right ventricular systolic hypertension has existed, as in isolated pulmonary valvular stenosis, the tricuspid valve may be mildly incompetent. The heart in such a case may have jet lesions above the septal leaflet of the valve on the septal surface of the right atrium (fig. 15*d*).

**Septal Defects.** Since jet lesions depend upon high-velocity streams starting in a high-pressure source and entering a region of lower pressure, it is consistent that examples of pronounced lesions are seen in cases of small ventricular septal defect (fig. 16). The jet lesions are seen on the endocardium opposite the defect. In those hearts having the com-



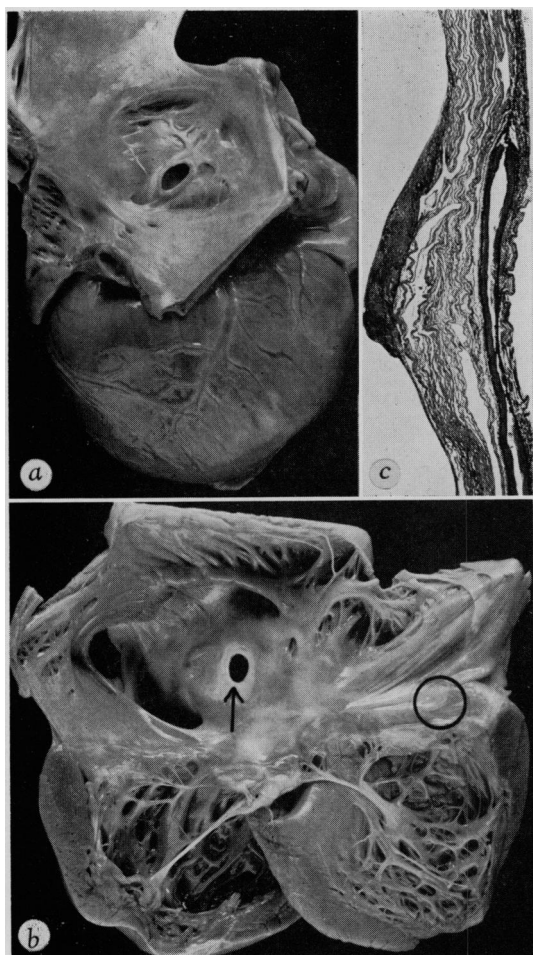


FIG. 17. Mitral atresia in a 14-year-old girl. *a.* The left atrium shows no mitral orifice. There is a small atrial septal defect. *b.* The right atrium. Opposite the small atrial septal defect the posterior wall of the right atrium shows a jet lesion (circle). *c.* Photomicrograph of the jet lesion of the right atrium (ELVG;  $\times 10$ ).

mon variety of defect, the anterior wall of the right ventricle near the tricuspid attachment is characteristically involved. In addition, the septal leaflet of the tricuspid valve, which at times overhangs the defect, is also frequently distorted by such lesions.

In the hearts of patients with large ventricular septal defects in whom the left and right ventricular pressures were about equal during life, there are no well-developed jet lesions.

In the hearts of patients having atrial septal defect as an isolated lesion, no jet lesions

are identifiable in the right atrium even though large volumes of blood enter this chamber. Recalling that the defect is usually large and that the pressure differences between the two atria are negligible makes absence of jet lesions in this condition understandable and, indeed, predictable.

We have observed a right atrial jet lesion in a heart having a small atrial septal defect in association with mitral atresia (fig. 17). In this condition the small atrial septal defect was the only effective outlet for the left atrium, which had received all the pulmonary venous blood. Though physiologic measurements had not been made, it is assumed that the left atrial pressure was elevated and a high-velocity jetlike stream of blood flowed through the small atrial septal defect. Jet lesions were present upon the posterolateral wall of the right atrium opposite the small atrial septal defect.

*Communication Between the Aorta and a Cardiac or a Pulmonary Vessel.* In hearts having a ruptured aortic sinus aneurysm, when the history indicates that the patient had lived for some time with it, jet lesions exist in that right-sided chamber into which the rupture of the aneurysm occurred. In one of the cases we observed in which the aneurysm had ruptured into the right ventricular outflow tract and there was an associated ventricular septal defect, jet lesions had developed upon the anterior wall of the outflow tract of the right ventricle.

In another case, an aortic sinus aneurysm had ruptured, 2 years before the patient's death, through the lower part of the septal wall of the right atrium into the right atrial cavity. Jet lesions of well-defined nature were present on the posterolateral aspect of the right atrium, opposite the site of the rupture in the aneurysm (fig. 18).

The vessels in a classic patent ductus arteriosus often show focal endarteritis characteristic of the jet lesion in the pulmonary artery opposite or proximal to the pulmonary orifice of the ductus. Such lesions occur either in the left pulmonary artery or in the pulmonary trunk itself (fig. 19). The position of

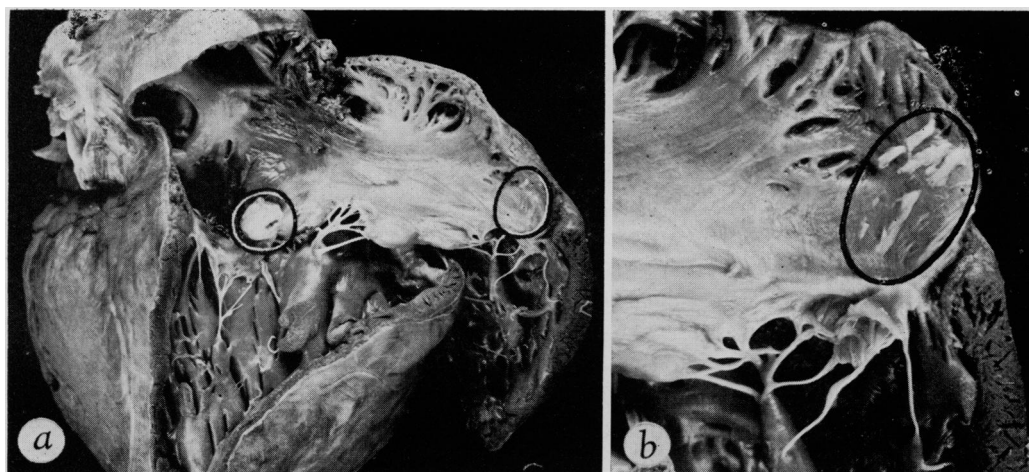


FIG. 18. *a.* Ruptured aortic sinus aneurysm presenting in right atrium (*left circle*). The opposite posterior wall of the right atrium shows a collection of jet lesions (*right circle*). *b.* Close-up view of jet lesions of right atrium. From a 42-year-old woman.

the jet lesion proximal to the pulmonary orifice of the ductus correlates well with the oblique position of the ductus in which the pulmonary orifice lies in a more medial position than the aortic orifice. A jet stream coming from a ductus would therefore be expected to strike the pulmonary arterial wall in a proximal location.

The counterpart of the jet lesions resulting from persistent patency of the ductus is seen in the great vessels of patients who have had a Blalock-Taussig anastomosis between the left subclavian and left pulmonary arteries. In one case in which the operation had been done on the left side, the direction of the anastomotic segment of the left subclavian artery was such that its aortic origin was more medial than its left pulmonary arterial connection. It is a reasonable conjecture that the blood stream was directed downward and laterally to account for the jet lesions that were found on the inferior wall of the left pulmonary artery distal to the site of anastomosis.

*Coarctation of the Aorta.* Beyond the region of aortic constriction in coarctation of the aorta, jet lesions may be present but are not universally found. When present, they tend to lie on the inferior wall of the aorta within a centimeter of the constriction. The presence of jet lesions in this location is readily under-

stood as a reaction to a high-velocity stream originating at the narrowed point in the aorta. Absence of a jet lesion is probably to be explained by peculiar orientation at the obstruction as a result of which the jet is directed toward the center of the aortic lumen and the kinetic energy is dissipated within the aortic lumen.

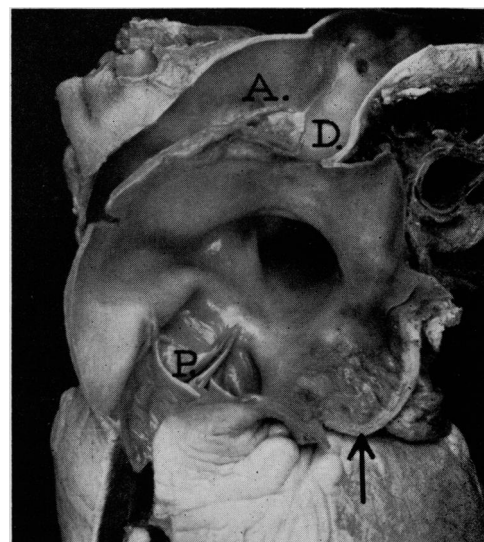


FIG. 19. Patent ductus arteriosus with infected jet lesion (*arrow*) near the pulmonary valve (*P*). *D* = patent ductus arteriosus; *A* = aorta. From a 14-year-old girl. Specimen contributed by Dr. James H. Peers.



## SUMMARY

High-velocity streams (jets) of blood are a hemodynamic characteristic when a high-pressure gradient exists across a small orifice. If such jet streams strike the wall of the receiving compartment, be it either cardiac chamber or blood vessel, the focal trauma causes a fibrous reaction designated herein as a jet lesion. At the site of impact where jet lesions are formed, the jet stream is either arrested or deflected and imparts energy to the area which may vibrate and give rise to an audible murmur. Jet lesions may be considered as designating possible sites of origin of murmurs in whole or in part, and in this regard may be utilized in the explanation of the particular positions of the maximal intensity of murmurs recorded during life. Random examples in support of this proposition are the systolic murmurs heard at maximal intensity in the left subclavicular area in some patients with congenital pulmonary valvular stenosis; in the left scapular area in patients with mitral regurgitation in which it is believed the anterior leaflet was of inadequate length to meet the posterior leaflet; and in the aortic area in patients with mitral regurgitation resulting from rupture of the chordae to the posterior leaflet which then behaves as a hood-like ~~baffle~~ directing the regurgitant stream forward and to the right.

## SUMMARIO IN INTERLINGUA

Currentes de sanguine de alte velocitate ("rapidos") es un characteristica hemodynamica que occurre quando un gradiente de alte pression existe a transverso un micre orificio. Si un tal rapido batte contra le pariete del compartmento receptori (que pote esser un camera del corde o un vaso de sanguine), le

trauma local causa un reaction fibrose que es designate in le presente reporto como "lesion rapidal." Al sito de impacto ubi le lesion rapidal es formate, le rapido es arrestate o illo es deflectite e imparti energia al area circumjacente que pote vibrar e producer assi un murmure audible. Lesiones rapidal pote esser considerate como indicadores de sitios possibile de murmures, in toto o in parte, e in iste respectu illos pote esser utilisate in le explication del positiones particular del intensitate maximal de murmures que es registrate durante le vita del patiente. Exemplos—cite non systematicamente—que supporta iste proposition es le murmures systolic que se audi a intensitate maximal in le area sinistro-subclavicular in certe patientes con congenite stenosis del valvula pulmonar; in le area sinistro-scapular in patientes con regurgitation mitral (ubi le supposition ese que le cuspid anterior es inadequamente longe pro junger se con le cuspid posterior); e in le area aortic in patientes con regurgitation mitral como resultado de ruptura del chordas del cuspid posterior que allora age como un deflector que dirige le corrente del regurgitation in un direction antero-dextrorse.

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