Chest Pain in Patients with Isolated Pulmonic Stenosis

By Richard P. Lasser, M.D., and Gabriel Genkins, M.D.

Chest pain like angina pectoris has been described in patients with pulmonary hypertension and cor pulmonale but its origin is obscure. In this article 5 patients are reported with isolated pulmonic stenosis and chest pain. On the basis of the clinical, electrocardiographic, hemodynamic, and pathologic findings the authors conclude that the chest pain is caused by ischemia of the right ventricular myocardium due to increased work and reduced coronary flow to the right ventricle.

This report concerns the occurrence of substernal and precordial chest pain in 5 patients with congenital stenosis of the pulmonic valve, a normal aortic root, and an intact interventricular septum. While not a commonly observed symptom in this particular congenital lesion nor frequently commented upon in the recent literature, chest pain and oppression are not at all rare in these patients and scattered reports can be found. Laubry and Pezzi in their text on congenital heart disease published in 1921 made note of chest discomfort in these patients terming the symptom "l'oppression d’effort" and distinguishing it from the dyspnea of effort. Stuckey, in 1955, reported angina pectoris in 6 of 38 patients with isolated pulmonic stenosis. Other reports, including several large series of cases, do not mention the occurrence of chest pain, nor is it mentioned in several of the texts on congenital heart disease.

In this report on 5 patients with pulmonic stenosis and a normal aortic root who experienced chest pain, we will attempt to define its clinical characteristics, to discuss the hemodynamic conditions under which it occurs, to ascertain the probable site of origin, and to indicate its grave prognostic import.

The diagnosis of pulmonic valvular stenosis with a normal aortic root was made in all 5 patients by cardiac catheterization and was confirmed at the time of surgery in each. They were from the medical ward services of the Mount Sinai Hospital, New York. Cardiac catheterization was performed preoperatively. Pressures were recorded by means of direct needle puncture of the right ventricle and pulmonary artery at surgery. Data were obtained before and after valvulotomy. All pressures at operation were recorded simultaneously by means of Statham pressure transducers adjusted to equal sensitivity. Several of the patients were also recatheterized approximately 1 year after operation.

Surgical procedures were performed by Dr. Mark M. Ravitch, using a transventricular approach to the pulmonic valve.

Clinical Features

The pertinent data concerning clinical details, intracardiac pressures, and systemic oxygen saturations are shown in table 1 and will be discussed individually.

All of these patients had right ventricular systolic hypertension that equaled, surpassed, or closely approached systemic blood pressure. Diastolic pressure in the right ventricle was moderately elevated in 2 patients, markedly elevated in 1, and normal in the other 2.

The electrocardiogram revealed a pattern of right ventricular hypertrophy in all patients.

All of the patients were in their teens or older. This age grouping may be more apparent than real because of the difficulty in eliciting a clear history of some of the subtler aspects of pain from a younger child.

All of the patients had symptoms in addition to chest pain, with complaints ranging from exertional dyspnea or fatigue to frank right heart failure. These latter symptoms were not always prominent, however, and the primary complaint in 1 of the severe cases (R. D.) was...
Table 1.—Clinical and Hemodynamic Data in Five Patients with Chest Pain and Isolated Pulmonic Stenosis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Pressure mm. Hg</th>
<th>Hgb. (Gm.)</th>
<th>Os Sat. (%)</th>
<th>Pain</th>
<th>Dyspnea</th>
<th>X-ray dilatation of P.A.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>RV</td>
<td>PA</td>
<td>Systemic†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S.L.</td>
<td>Pre</td>
<td>20</td>
<td>105/5</td>
<td>30/15</td>
<td>120/90</td>
<td>16</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>53/5</td>
<td>25/10</td>
<td>130/90</td>
<td>14</td>
<td>94</td>
<td>0</td>
</tr>
<tr>
<td>E.F.</td>
<td>Pre</td>
<td>36</td>
<td>160/22</td>
<td>Mean 0</td>
<td>100/60</td>
<td>14.0</td>
<td>92</td>
</tr>
<tr>
<td></td>
<td>*Post</td>
<td>72/22</td>
<td>39/15</td>
<td>90/60</td>
<td>12</td>
<td>99</td>
<td>+</td>
</tr>
<tr>
<td>I.B.</td>
<td>Pre</td>
<td>16</td>
<td>96/6</td>
<td>14/5</td>
<td>136/73</td>
<td>12</td>
<td>99</td>
</tr>
<tr>
<td></td>
<td>*Post</td>
<td>51/8</td>
<td>21/11</td>
<td>90/60</td>
<td>12</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>R.D.</td>
<td>Pre</td>
<td>35</td>
<td>134/10</td>
<td>—</td>
<td>130/70</td>
<td>16.0</td>
<td>97</td>
</tr>
<tr>
<td></td>
<td>*Post</td>
<td>84/9</td>
<td>36/13</td>
<td>90/60</td>
<td>12</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>V.E.</td>
<td>Pre</td>
<td>52</td>
<td>129/3</td>
<td>Mean 6</td>
<td>151/88</td>
<td>14.0</td>
<td>94</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>114/8</td>
<td>25/8</td>
<td>140/90</td>
<td>14.0</td>
<td>95</td>
<td>0</td>
</tr>
</tbody>
</table>

Pre = preoperative
Post = postvalvulotomy
* Pressures obtained in operating room.
† Systemic pressure obtained by direct needle puncture of femoral or brachial artery or at time of operation from the aortic arch.

Characteristics of Chest Pain

S. L., a 30-year-old white woman with a known cardiac murmur since birth, had noted progressive exertional dyspnea since her early teens. For 3 months prior to admission she had suffered from distinct, rather severe substernal oppression, which did not radiate, was provoked by the mildest exertion, and was promptly relieved by rest. This sensation was distinct and different from dyspnea in that the latter was present almost constantly while the former occurred only on exercise with prompt relief by rest. The oppressive feeling was not true pain but might be described rather as a sensation on the verge of pain. Whether progression to a true anginal syndrome might in time have taken place is not known, as pulmonary valvulotomy completely abrogated the substernal oppression. The exertional dyspnea was, however, not significantly affected. In general, exercise tolerance improved significantly after surgery.

Symptomatology of such a nature would appear to correspond with "l'oppression d'effort" of Laubry and Pezzi. It is an example of the mildest type of chest pain syndrome noted.

The second patient, E. F., a woman 36 years of age, complained of substernal oppression and burning brought on by exertion and relieved by rest. The duration of each episode was approximately 30 min. Again, these symptoms were separate and distinct from exertional dyspnea. The symptoms of exertional pain in this case were also completely relieved by valvulotomy.

The third patient, I. B., 16 years of age, described 2 distinct types of pain. Upon moderate exertion, she experienced a sharp, knife-like pain in the fourth...
left intercostal space that grew more intense on further exertion but was relieved rapidly by rest and nitroglycerin. Simultaneously, she would note a dull, heavy, constant substernal oppressive sensation that was more protracted and did not diminish as readily on resting. Nitroglycerin, however, always terminated both types of pain. These symptoms were completely eliminated by pulmonary valvulotomy.

R. D., a robust 35-year-old laborer, complained of severe precordial pain as his only symptom. For many years he had been aware of episodes of "fluttering" in the chest, occurring 1 to 6 times per year. These were at first accompanied by no symptoms other than slight dizziness that subsided spontaneously after 10 minutes to 1 hour. For the past 3 years, however, these episodes were accompanied by a severe burning precordial pain radiating to the neck and left arm that would cease approximately 1 to 2 hours after the conclusion of the attack. These episodes were felt to represent attacks of paroxysmal tachycardia. The sensation of dizziness became more severe and progressed at times to almost complete syncope. In addition, during the same period of time, the same type of pain was also noted to occur during periods of exertion at work. This pain was identical with that occurring during the bouts of palpitation. At all times rest would relieve the pain within 10 minutes although a distinct substernal soreness might persist for 1 to 2 hours. For 1 year prior to admission, significant exertional dyspnea (1 flight, 10 blocks) had appeared. No manifestations of cardiac failure were noted.

During cardiac catheterization an episode of tachycardia was provoked by the exploring catheter tip and the full syndrome of substernal pain as well as electrocardiographic demonstration of myocardial ischemia were noted (fig. 1). Relief of the pain as well as freedom from prolonged bouts of tachycardia were brought about by transventricular valvulotomy although transient arrhythmias were present in the immediate postoperative course.

The fifth patient, V. E., a 51-year-old woman, was the most severely ill of all and experienced the greatest degree of disability from chest pain. For 1 year previously, she was greatly limited by severe precordial pain that appeared with effort and radiated to both shoulders and arms. It lasted about 15 minutes and was followed by hours of precordial soreness. The severe pain was relievable by nitroglycerin and had also been ameliorated by oxygen inhalation.

One day prior to admission, she was stricken by an excruciating, crushing pain felt all over the anterior surface of the chest. This pain remained severe for about 6 hours and was finally relieved by analgesic injection. Residual soreness remained for several days. This attack had all the appearance of myocardial infarction, but failure of the electrocardiogram to develop characteristic changes, failure of sedimentation rate to rise, absence of fall in blood pressure, and the subsequent clinical course all made it extremely unlikely. It was finally considered that the attack represented severe and protracted coronary insufficiency. Pulmonary valvulotomy was done, a sufficient fall in right ventricular systolic pressure was effected, and after postoperative recovery had occurred, the chest pain was entirely relieved.

In summary then, 2 types of chest pain have been described by these patients. The first type is characterized by being (1) provoked by effort; (2) relieved by rest; (3) of variable duration, but generally less than 10 minutes; (4) located either substernally or precordially; (5) of variable severity ranging from an oppressive sensation without true pain to severe pain without oppression; (6) experienced during tachycardia; (7) relieved by nitroglycerin.

The second type of pain is (1) protracted; (2) generally but not always provoked by effort; (3) not promptly relieved by rest; (4) of considerable severity occasionally simulating myocardial infarction; (5) relieved inconstantly by nitroglycerin or by oxygen inhalation.

Electrocardiographic Features

The electrocardiograms of these patients in all cases showed a pattern of marked and obvious right ventricular hypertrophy. All showed right axis deviation, a QRS complex of normal duration and the presence of V1 of one of the forms of QRS complex associated with hypertrophy of the right ventricle. Three
patients showed a tall R wave and absent S wave in V1. The R wave was either slurred or deeply notched on the upstroke. One patient showed an rs'R' complex in V1, a pattern that is seen commonly in patients with right ventricular hypertrophy with or without conduction interference wherein the terminal R wave is taller than the initial one. One patient showed an R complex in V3 with an R/S ratio of about 3:1. A marked depression of the S-T segments and inversion of T waves in precordial leads V1-V4 was noted in 1 patient (V. E.). This patient was the 1 of the group who experienced the most severe and protracted chest pain. Low or inverted T waves were found in standard lead II in 3 patients and in leads V1-V4 in 1 patient. None of the electrocardiographic findings mentioned were confined solely to this group of patients with right ventricular hypertrophy and chest pain. These same abnormalities of S-T segments and T waves were seen in other patients with marked hypertrophy of the right ventricle who may not have chest pain.

In the patient (V. E.) in whom electrocardiograms were taken during and after a protracted episode of chest pain, we did not observe parallel fluctuation of the depressed S-T segment corresponding with the clinical condition.

In another patient (E. F.) an electrocardiogram was obtained immediately following a short period of exercise (Master's single 2-step test). Because of fatigue and dyspnea, it was not possible to complete more than 1 min. of exercise, but chest pain was not provoked nor was a change observed in the electrocardiogram. A report of electrocardiograms taken after exercise in a series of 26 cases of pulmonic stenosis was published in 1954.14 The authors found that very few changes were induced. The one observed chiefly was a shortening of the QTc interval. However, none of these patients was reported to have had chest pain, 15 were without any cardiac symptoms at all, another 9 showed only exertional dyspnea, 1 was in heart failure, and only 2 had markedly impaired exercise tolerance. Thus, most of these cases were examples of mild pulmonic stenosis or were in a stage of complete cardiac compensation.

One patient (R. D.), as previously demonstrated, did show electrocardiographic evidence of myocardial ischemia during a bout of supraventricular tachycardia that developed during cardiac catheterization. The electrocardiogram showed depression of the S-T segments during the tachycardia, which returned to normal following return to regular sinus rhythm. Electrocardiograms of 3 of these patients taken approximately 6 months to 1 year postoperatively, were available. The postoperative tracings were characterized by a distinct return to "normal." The right axis deviation was entirely absent in 1 and of much less extent in the other 2. The amplitude of the R wave in proportion to the S in V1 diminished to an entirely normal relationship in 1 patient and to near normal in the others. Inversion of T waves has not reverted entirely to normal. An example is shown in figure 2.

**Discussion**

What is the site of origin of this chest pain and what is the mechanism of its production? We believe it to be due to myocardial ischemia. Further, the right ventricular myocardium is felt to be the ischemic site. Another possible source of pain that will be considered is the pulmonary artery itself. This structure has been considered to be responsible to atypical chest pain experienced by patients with idiopathic pulmonary hypertension and also in patients with chronic cor pulmonale18 who present some clinical features in common with the group under discussion.

Evidence pointing to the probable existence of right ventricular myocardial ischemia has been derived from: (1) consideration of hemodynamic relationships and (2) pathologic findings.

**Hemodynamic Considerations**

The probable existence of right ventricular myocardial ischemia is indicated by the additive deleterious effects of an increased right ventricular myocardial oxygen demand in the presence of a hemodynamic state that is unfavorable to coronary flow to the right ventricle. The increased oxygen demand results from a greatly hypertrophied right ventricular myocardium, which is an anatomic expression
CHEST PAIN IN PATIENTS WITH ISOLATED PULMONIC STENOSIS

262

FIG. 2. E. F., preoperative (December 12, 1954, left) and postoperative (March 21, 1955, right) electrocardiograms. Note diminished amplitude and more normal contour of P waves; disappearance of right axis deviation, of the late R wave in aVR, and tall R wave in V1, reflecting the change to normal from right ventricular preponderance.

FIG. 3. F. B., Simultaneous needle punctures of pertinent areas, recorded by means of strain gages adjusted to equal sensitivity, at time of operation. Note that right ventricular pressure exceeds aortic pressure from the onset of aortic ejection to .04 sec. beyond closure of the aortic valves.

of the high level of right ventricular work, about 5 times that of the normal right ventricle in patients of this type.19

Factors acting to impair coronary circulation are as follows:

1. The marked elevation of right ventricular intracavity pressure during ventricular systole and the protracted duration of this period by comparison with events in the aorta. This pressure relationship is unfavorable to right ventricular coronary flow by virtue of its influence upon intramural perfusion. Perfusion of ventricular musculature during its contraction proceeds when a difference exists between aortic pressure, which acts to drive flow forward, and the pressure that is exerted on the intramural coronary vessels by muscular contraction, which opposes flow by compressing the vascular lumina. Since the intramural pressure is roughly equivalent to the intracavity pressure,20 it is evident that intramural perfusion of the right ventricle (particularly of the innermost layers) is probably substantially prohibited during much of right ventricular systole when the right ventricular pressure exceeds the aortic pressure. These pressure-time relationships between aorta and right ventricle are illustrated in figure 3. The curves were obtained by direct needle puncture at the time of operation according to a technic previously reported.21 This patient had isolated pulmonic valvular stenosis with a moderate degree of congestive failure but did not complain of distinct chest pain or oppression.

2. A low level of systemic cardiac output, which is relatively fixed and cannot be augmented in a normal fashion during exercise.9, 17
3. The presence of a moderate to marked elevation of right atrial and ventricular diastolic pressure. This factor acts to reduce both the coronary arteriovenous pressure gradient and Thebesian vein drainage, thus increasing the intravascular resistance to flow during diastole. Though direct measurement of right coronary flow has not been accomplished in man, experimental work in dogs has specifically demonstrated myocardial ischemia and reduced coronary flow in the presence of right ventricular systolic hypertension such as that produced by acute narrowing of the pulmonary artery or mitral valve orifice.

An insight into the compensatory mechanisms of the heart is afforded by the observations that in dogs the immediate effect of elevation of right ventricular systolic pressure is actually an increase in right coronary arterial flow. When the experimental lesion is maintained for a few hours, however, coronary flow consistently diminishes below the resting level. Gregg has explained those findings as follows: "Thus in the latter stages of an experiment...the flow-reducing effect of increased extravascular support [intramural tension] is dominant over whatever flow-promoting mechanism the heart has retained, and would appear to be left almost unopposed by any concomitant dilatation of the coronary bed."

It is obvious that the human heart, too, is able for many years to compensate for these various factors that reduce coronary perfusion and increase the myocardial oxygen demand. Finally, some new factor intervenes, such as tachycardia, or further reduction of the coronary flow and increase in the degree of hypertrophy exceed the limits of coronary compensation and symptoms of angina or signs of congestive failure appear.

**Pathologic Findings**

Pathologic examination of postmortem specimens has furnished direct evidence of the prior existence of a diffuse right ventricular myocardial ischemia. A review of 5 cases of patients with severe, isolated pulmonic stenosis revealed extensive fibrosis of a patchy nature distributed throughout the right ventricle. The left ventricle was spared, indicating that the coronary insufficiency was not ubiquitous but was rather confined to the right ventricular myocardium. The most severe example of this pathologic lesion is shown in figure 4. These

![Figure 4](image-url)

**Fig. 4. A. L., Right (left) and left (right) ventricular sections (both X 260) revealing the severe right ventricular myocardial scarring, and a normal appearing left ventricular myocardium.**
sections were obtained from a 21-year-old white man, A. L., who was found at post-mortem examination to have combined pulmonary valvular stenosis and infundibular stenosis with intact ventricular septum and interatrial septal defect. He had died as a result of the rupture of a frontal lobe brain abscess. Marked exercise intolerance had been noted for many years but no history of chest pain was obtained.

The right ventricular myocardium was 2.5 cm. in thickness, exceeding the thickness of the left ventricular wall. Grossly, the right ventricular myocardium revealed marked fibrosis in contrast to a normal appearing left ventricular musculature. The extent of this difference was strikingly illustrated by the representative areas of the 2 ventricles in figure 4. There was mild right coronary artery preponderance. No coronary sclerosis was observed. This extensively scarred right ventricular myocardium would certainly seem to be a site of chronic coronary insufficiency. Other investigators have reported similar pathologic findings in these patients.28-30

The presence or absence of anginal pain, then, in any 1 individual patient might be a function of the acuteness with which local anoxia occurred under stress. Somewhat similar type of chest pain has been reported in patients with pulmonary hypertension due to various causes including idiopathic pulmonary hypertension,31 chronic pulmonary fibrosis and emphysema,18, 32 and mitral stenosis.32-34 Viar and Harrison18 have proposed that chest pain in these patients is not due to myocardial ischemia but has its origin in the distended pulmonary artery trunk itself. Is it possible that such a hypothesis applies to the cases reported here? In our cases of pulmonic stenosis, the main and initial branches of the pulmonary artery are dilated, often considerably, but the one essential element of pulmonary hypertension is lacking. Actually, the pressure within the pulmonary artery is at normal or lower than normal levels.

Is it possible then that poststenotic dilatation of the pulmonary artery alone is capable of giving rise to pain? Certainly, at operation these pulmonary arteries are not flaccid but are seen to be tense and distorted by the impact of a jet stream of blood from the right ventricle ejected under considerable force. Pain that is localized to the site of an aneurysmal dilatation of either the aorta or pulmonary artery secondary to syphilitic vasculitis is well known. On the other hand, patients with only moderate degrees of pulmonary stenosis may have pronounced dilatation of the pulmonary artery and in our experience do not experience chest pain. In our group, as mentioned, there was no correlation whatever between the size of the pulmonary artery and the severity of chest pain.

Summary

A group of 5 patients with proved isolated pulmonic valvular stenosis manifested anginal pain of varying severity, ranging from mild substernal oppression to severe, intractable precordial pain. In all cases, the diagnosis was proved at catheterization and in each instance the symptomatology was completely relieved by surgical intervention.

The right ventricular myocardium is thought to be the source of this anginal pain. This concept is analyzed through electrocardiographic, hemodynamic, and pathologic data. It is pointed out that this cardiac lesion requires the right ventricle to labor under a strikingly increased stroke work load, under hemodynamic circumstances that are unfavorable to coronary flow. Under stimulus of effort or in the presence of arrhythmias these difficulties are enhanced and clinical and electrocardiographic evidence of myocardial insufficiency may be noted.

Pathologic evidence of severe right ventricular myocardial fibrosis was demonstrated in a patient with pulmonic stenosis who died of an unrelated cause.

The syndrome of "right-sided" angina may prove to be of prognostic significance in these patients with pulmonic stenosis, as its appearance would seem to herald an imbalance between the demands made on and the resources of the right ventricular myocardium. It would seem logical to apply this concept to other cardiac lesions, congenital or acquired, that have led to right ventricular hypertension and hypertrophy.
LASSER AND GENKINS

SUMMARIO IN INTERLINGUA

Un gruppo de 5 patientes con provate stenosis pulmono-valvular isolate manifestava dolores anginal de varie grados de severitate, ab leve oppression substernal usque a sever e intrac-tabile dolores precordial. In omne casos le diagnose eseva confirmate per catheterisation, e in omne casos le symptomologia eseva completamente alleviata per le intervention chirurgic.

Nos opinà que le myocardio ventricular es le fonte del dolor anginal. Iste conception es analysate super le base de datos electrocardiographic, hemodynamic, e pathologic. Es signalate que iste lesion cardiac require ab le ventriculo dextere que illo labora sub un fraffantemente augmentate carga de labor pulsatile e sub conditiones hemodynamic que es disfavorabile al fluxo coronari. Le stimulo de effortio o le presentia de arrythmias resulta in un augmento de iste difficultates, e manifestazione clinica e electrocardiographic de insufficiency myocardial deveni observabile.

Signos pathologic de sever grados de fibrosis myocardial dextero-ventricular eseva demonstrate in un patiente con stenosis pulmonic qui moriva ab un causa sin relation al condition hic discutite.

Le syndrome de angina “dextero-lateral” pote esser de signification prognostic in iste patientes con stenosis pulmonic, proque su manifestation pare announce un disbalanci inter le demandas que le myocardio dextero-ventricular debere satisfacere e le ressources que illo possede. Il pare logic applicare iste concepto a altere lesions cardiac, congenite o acquiretie, que ha resultate in le disvelopamento de hypertension e hypertrophia dexteroventricular.

REFERENCES

hemodynamics of the left side of the heart as studied by simultaneous left atrial, left ventricular, and aortic pressures; particular reference to mitral stenosis. Circulation 12: 69, 1955.


Two hundred thirty-one euthyroid, seriously ill cardiac patients have been treated with radioactive iodine since February 1950. Of particular interest is the analysis of the original 100 patients treated 4 years ago. The patients in this series were treated for severe angina pectoris or severe congestive heart failure, or a combination of both. The rationale of treatment was to produce a state of beneficial relative hypothyroidism by lowering the total metabolism of the body so that the heart had less work to do. An interesting feature in this series of cases was the use of “thyrogram” (a diagram of the thyroid gland made by a scintillation counter) to check gland size and function. Fifty-six per cent of 94 patients with angina pectoris showed excellent results, and 37 per cent of this number showed good results. Of 78 patients with congestive heart failure 53 per cent showed excellent and 28 per cent good results. A group of 59 patients with angina pectoris and congestive heart failure combined showed 48 per cent excellent and 32 per cent good results.

Kitchell
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