The Effect of Pregnancy on the Murmurs of Mitral and Aortic Regurgitation

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SUMMARY
The effect of pregnancy on the intensity of murmurs of aortic and mitral regurgitation has not been reported previously. Twenty-five women with these murmurs, singly or in combination, were examined when pregnant as well as when not pregnant. During pregnancy the murmur of aortic regurgitation decreased in intensity or became inaudible in 10 patients, remained unchanged in four, and increased in one. The murmur of mitral regurgitation decreased in intensity or became inaudible in eight patients, became shorter in duration in two, was unchanged in three, and became louder in one. Three of the patients who did not show a decrease in intensity of the mitral or aortic regurgitation murmur and the patient whose murmurs increased during pregnancy had the appearance or persistence of systemic hypertension during pregnancy. Phenylephrine consistently increased the loudness of the murmurs, usually to the level present in the nonpregnant state. The diminished intensity of these murmurs may be related to the decrease in peripheral resistance known to occur during pregnancy. It is apparent that (1) aortic regurgitation or mitral regurgitation may be missed during pregnancy, since these murmurs may become inaudible, and (2) the severity of these cardiac lesions may be underestimated clinically because of a decrease in intensity of these murmurs during pregnancy. It is important to determine the existence of valvular heart disease, since endocarditis is a hazard during parturition, and prophylactic antibiotic therapy should be given to these patients.

Additional Indexing Words: Hypertension Phenylephrine Rheumatic fever
Rheumatic heart disease Tricuspid insufficiency

The effect of pregnancy on the intensity of the murmurs of mitral and aortic regurgitation has not been reported. It would be anticipated that these murmurs would become less intense during pregnancy, since the degree of mitral and aortic regurgitation is directly dependent upon systemic blood pressure and peripheral resistance,1-3 and these parameters decrease during pregnancy.4-8

Methods
Over a 5-year period, 25 women with either mitral or aortic regurgitation, or both, were examined during pregnancy and either before pregnancy or after delivery. Nine patients had aortic regurgitation alone, three had aortic regurgitation and mitral regurgitation, and nine had mitral regurgitation alone. During pregnancy, three of the 25 patients (J. B., S. H., and W. S.) had the murmur of mitral stenosis in addition to aortic regurgitation or mitral regurgitation, and...
The Effect of Pregnancy on the Murmur of Aortic Regurgitation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age during pregnancy Mo</th>
<th>Blood pressure (mm Hg)</th>
<th>Pulse (beats/min)</th>
<th>Murmur (grade)</th>
<th>Exam during gestation Mo</th>
<th>Blood pressure (mm Hg)</th>
<th>Pulse (beats/min)</th>
<th>Murmur (grade)</th>
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<td>(7-9 mo)</td>
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<td>100/60</td>
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<td>-</td>
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<td>9th</td>
<td>150/105</td>
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<td>III/VI</td>
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*Patients who had additional murmurs but not mitral insufficiency.
†Patients who also had mitral insufficiency. See table 2.
‡This patient had murmurs of aortic, mitral, and tricuspid insufficiency, as well as mitral stenosis.

one patient (M.L.) had murmurs consistent with aortic and mitral regurgitation, mitral stenosis, and tricuspid regurgitation.

The majority of patients were referred for cardiac evaluation during pregnancy because of the presence of rheumatic heart disease or a past history of rheumatic fever. They were all seen in outpatient clinics. The selection of patients was based on the presence of the murmur of mitral or aortic regurgitation during pregnancy or when

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<table>
<thead>
<tr>
<th>Exam during pressor effect</th>
<th>Postpartum exam</th>
<th>History of rheumatic fever</th>
<th>Comment</th>
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<td>III/VI</td>
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<td></td>
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<td>2</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>4</td>
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<td>I/VI</td>
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<tr>
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<td>130/80</td>
<td></td>
<td>I/VI</td>
</tr>
<tr>
<td>(1 wk)</td>
<td>130/80</td>
<td></td>
<td>II/VI</td>
</tr>
<tr>
<td>2⅓</td>
<td>130/84</td>
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<td>II/VI</td>
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<td>Patients in whom the murmur decreased during pregnancy</td>
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<td></td>
</tr>
<tr>
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<td>110/70</td>
<td></td>
<td>II/VI</td>
</tr>
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<td>I-II/VI</td>
</tr>
<tr>
<td>2</td>
<td>144/92</td>
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not pregnant. In addition, patients were included only if they were examined by the same physician during pregnancy and when not pregnant. Otherwise, all patients with these murmurs were included. The high incidence of patients with aortic regurgitation, unusual in women with rheumatic valvular disease, may be explained by the possibility that these patients were referred once our interest in the behavior of these murmurs during pregnancy become known to physicians.

These patients did not have hemodynamically severe regurgitation. This evaluation was based on the observations that none of the patients had a history of heart failure prior to or during pregnancy, that only one patient had a murmur.

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## Table 2

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age during pregnancy (yr)</th>
<th>Pregestation exam</th>
<th>Blood pressure (mm Hg)</th>
<th>Pulse (beats/ min)</th>
<th>Murmur (grade)</th>
<th>Exam during gestation</th>
<th>Blood pressure (mm Hg)</th>
<th>Pulse (beats/ min)</th>
<th>Murmur (grade)</th>
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<td></td>
<td>Patients in whom the murmur decreased during pregnancy</td>
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<td></td>
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<td>24</td>
<td>3</td>
<td>II-III/VI</td>
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<td></td>
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<td>100/70</td>
<td>80</td>
<td>0</td>
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<tr>
<td>E.H.</td>
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<td>II/VI</td>
<td>8th</td>
<td>110/68</td>
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<td>II/VI</td>
<td>7th</td>
<td>100/70</td>
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</table>

| B.S.    | 19                        | 3                 | II-III/VI              | 1st               | II/VI         | 6th                   | 110/70                 | -                 |               |
| J.S.    | 19                        | 7                 | -                      | 110/70            | II/VI         | 8th                   | 100/68                 | -                 |               |
| S.J.    | 22                        | 7                 | -                      | 110/80            | I/VI          | 6th                   | 110/70                 | -                 |               |
| J.B.†   | 34                        | 7                 | -                      | 100/70            | II/VI         | Monthly, 2nd-9th     | 100-110/50            | -                 | I/VI          |
| R.L.†   | 21                        | 12                | 130/70                 | 84                | II/VI         | 7th                   | 120/80                 | -                 | II/VI         |
| R.W.†   | 24                        | 7                 | -                      | 120/80            | II/VI         | 6th                   | 100/60                 | -                 |               |
| M.L.‡   | 36                        | 9                 | 140/84                 | 90                | II/VI         | 9th                   | 88/56                  | -                 |               |

| C.S.†   | 17                        | 2                 | 120/80                 | II/VI             | 3rd           | 120/80                 | 9th                   | 150/105           | III/VI        |

*Patients who had an additional murmur but not aortic insufficiency.
†Patients who also had aortic insufficiency. See table 1.
‡This patient had murmurs of aortic, mitral and tricuspid insufficiency, as well as mitral stenosis.

The murmur of mitral regurgitation was defined as a high-pitched murmur of maximal intensity at the apex. The majority of these murmurs were pansystolic. Less frequently there was a mid-to-late systolic crescendo murmur consistent with mild mitral regurgitation. A few patients had a high-pitched apical systolic murmur that ended before the second heart sound. This murmur was judged to be due to mitral regurgitation after it became pansystolic following the infusion of phenylephrine. The

* louder than grade III/VI after delivery, and that the patients with aortic regurgitation had normal diastolic and pulse pressures after delivery.

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The effect of pregnancy on cardiac murmurs

Exam during pressor effect

<table>
<thead>
<tr>
<th>Exam during pressor effect</th>
<th>Blood pressure (mm Hg)</th>
<th>Pulse (beats/ min)</th>
<th>Murmur (grade)</th>
<th>Postpartum exam</th>
<th>Blood pressure (mm Hg)</th>
<th>Pulse (beats/ min)</th>
<th>Murmur (grade)</th>
<th>History of rheumatic fever</th>
<th>Comment</th>
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<tr>
<td>Mo</td>
<td></td>
<td></td>
<td></td>
<td>Mo</td>
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<td>4</td>
<td>110/60</td>
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<td>120/60</td>
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<td>IV/VI</td>
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<td>130/80</td>
<td>II/VI</td>
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<td>2 1/2</td>
<td>130/84</td>
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</table>

Patients in whom the murmur decreased during pregnancy

A thrill was felt post partum but was not palpable during pregnancy.

During pregnancy the apical murmur was not holosystolic and was not diagnostic of mitral regurgitation. After phenylephrine and postpartum, the murmur became holosystolic.

Murmur was II/VI at 1st mo; I/VI at 6 mo, and not heard at all in 8th mo.

(Msame comment as for M.G. above)

Murmur decreased in intensity by the 5th mo.

Patients in whom the murmur did not decrease during pregnancy

The murmur was late systolic and was introduced by a mid-systolic click during and after pregnancy.

Murmur increased from II/VI to III/VI with the onset of hypertension during pregnancy.

The murmur of aortic regurgitation was defined as a high-pitched murmur occurring early in diastole. The murmur was heard best at the left sternal border. The intensity of the murmurs was graded on the basis of I to VI. Patients were examined while both in the supine and the sitting positions for the murmur of aortic regurgitation, and in the left lateral position for the murmur of mitral regurgitation. At each examination, auscultation was performed before the physician looked at the patient's record where his previous findings had been recorded.

Phenylephrine, 0.25 mg, was injected intravenously in seven women during pregnancy.

*CPhenylephrine was given to other pregnant women suspected of having aortic or mitral regurgitation, but murmurs indicative of these lesions were not elicited during the test. Post partum, these murmurs were not heard; therefore, these patients were not included in this report.

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this dose did not raise the systolic pressure by 15 to 20 mm Hg in 2 to 3 min, 0.5 mg was then given. The induced hypertension usually caused a decrease in heart rate of 10 to 20 beats/min.

Results

The effect of pregnancy on the murmur of aortic regurgitation is presented in table 1. The murmur was not heard during pregnancy in eight of 15 patients with aortic regurgitation. When these same patients were not pregnant, a grade I to III/VI high-pitched diastolic murmur was heard. The murmur of aortic regurgitation decreased in intensity in one patient and became audible late in pregnancy in another. The murmur was unchanged in four patients, one of whom was hypertensive during pregnancy. The murmur increased in intensity in one patient (C.S.), who had an increase in blood pressure between the third and the ninth month of pregnancy corresponding with the increase in loudness of the murmur. Patient M.T. may have had pulmonic regurgitation. In this patient there was a clearly discernible interval between the second sound and the onset of the murmur. This uncertainty existed even after right heart catheterization, pulmonary angiography, and intracardiac phonocardiography.

Phenylephrine was given to three patients suspected or known to have aortic regurgitation, but in whom the murmur of aortic regurgitation was not heard at the time of the test. An audible murmur consistent with aortic regurgitation was elicited in all three patients after the phenylephrine was given (table 1).

The effect of pregnancy on the murmur of mitral regurgitation is presented in table 2. Fourteen patients had this murmur before pregnancy or after delivery. During pregnancy this murmur was inaudible or became softer in eight patients, became shorter in duration in two (M.G. and J.S.), was unchanged in three, and increased in one patient. The diagnosis of mitral regurgitation was aided by the injection of phenylephrine in patients M.G. and J.S., since its administration resulted in a lengthening of the murmur to that of a typical pansystolic murmur. After delivery, these two patients had pansystolic murmurs.

Miscellaneous Observations

Two patients, L.L. and R.L., with aortic regurgitation had a prominent ejection click. In both patients the murmur could not be heard during some part of pregnancy. However, the ejection click persisted and was unchanged in intensity during pregnancy. Three patients, J.B., W.S., and M.L., had apical diastolic rumbling murmur heard only during pregnancy. This finding was interpreted as indicating the presence of mitral stenosis. None of these subjects was thought to have sufficient mitral regurgitation to account for the presence of a diastolic "flow" murmur or sufficient aortic regurgitation to have an associated Austin Flint murmur.

M.L. had tricuspid regurgitation. This diagnosis was based on the finding of a systolic murmur heard best at the lower left sternal border. Before pregnancy this murmur was grade I/VI and increased to grade II/VI with inspiration and also with amyl nitrite inhalation. During pregnancy it was grade II/VI during expiration, and increased to grade IV/VI during inspiration. In pregnancy the murmur had a "whooping" sound during inspiration.

Time Course of the Change in Intensity of Aortic and Mitral Regurgitation Murmurs with Pregnancy

Three patients were examined on several occasions during pregnancy. L.L. had a grade II/VI murmur of aortic insufficiency before pregnancy. She was examined when 3 months pregnant. The murmur of aortic regurgitation could not be heard, nor could an aortic insufficiency murmur be detected in the fifth and seventh months. A grade II/VI diastolic blowing murmur was heard on the first postpartum day. In R.L. the murmur of aortic regurgitation started to decrease in intensity by the fifth month and was inaudible during the sixth and ninth months. A similar time course was present in this patient with regard to the decrease in the murmur of mitral
regurgitation. In patient B.S., the murmur of mitral regurgitation was unchanged during the first month of pregnancy, was decreased at the sixth month, and could not be heard at all during the eighth month.

Discussion

It is clear from these observations, extending over 5 years, that the murmurs of mild aortic and mitral regurgitation will generally become softer or even inaudible during pregnancy (tables 1 and 2). Attempts were made to document these findings by phonocardiography; however, the majority of these murmurs were not only of high frequency, but were soft. The combination of low amplitude and high frequency required amplification sufficient to produce a waveling base line. Therefore, the records were usually unsatisfactory. Nonetheless, these observations are thought to be valid for the following reasons: The study was prospective, and each patient was examined during pregnancy and either before or after pregnancy by the same physician. The element of bias cannot be excluded from these observations since grading of murmurs is subjective. Certain notations made during this study were helpful in verifying the validity of the change in the murmur during pregnancy. For example, it was noted during the second pregnancy of patient L.L. that the murmur of aortic regurgitation was not heard when the patient was supine but was heard only when the patient was in the upright position. Post partum, it was readily heard when the patient was supine. In patient E.H. an apical systolic thrill was present post partum but was not felt during pregnancy. In 11 patients the murmur of aortic or mitral regurgitation could not be heard at all during pregnancy but was heard before or after pregnancy. In six of these patients the murmur was of grade II/VI or III/VI when the patient was not pregnant. It is unlikely that these murmurs would have been missed. During the course of the study it was considered possible that the murmurs of aortic and mitral regurgitation did not change in intensity but rather were altered in location of maximum intensity because of a more horizontal position of the heart. This hypothesis could not be verified by auscultation of the entire precordium in many patients. Also, phenylephrine infusion during pregnancy increased the intensity of the murmurs, at which time they were heard best at the anticipated location. Finally, spontaneous variations in the intensity of the murmur, unassociated with pregnancy, could not be excluded as an explanation for the change in murmurs during pregnancy. The consistency of change in intensity correlated with pregnancy is against this possibility. Serial examinations on the same patients such as L.L., who was examined before and after two pregnancies, also served to exclude spontaneous variations in the murmurs.

The reasons for the decrease in the intensity of the murmurs of aortic and mitral regurgitation during pregnancy may be related to the diminution in systemic vascular resistance accompanying pregnancy. The hemodynamic alterations occurring during pregnancy that appear to be pertinent to this study may be summarized as follows. An increase in cardiac output can be demonstrated by the eighth to the twelfth week of pregnancy. Cardiac output continues to rise until the 24th to 28th week. Thereafter, traditionally, it has been taught that the cardiac output declines in the third trimester. However, this is based upon data gathered during measurements in the supine position. Recently, Lees and associates and Kerr documented that the increase in cardiac output is maintained during the latter part of the second and throughout the third trimester. They measured cardiac output with the subjects in the lateral, as well as in the supine positions, and determined that the decrease in cardiac output observed in the supine position in the latter part of pregnancy is due to decreased venous return caused by partial vena caval compression by the fetus. There is general agreement that systemic vascular resistance decreases during the first and second trimesters, since the mean blood pressure is unchanged or decreased despite the increase in.
cardiac output. Sites of low vascular resistance include the placenta, the kidney, skin, and probably the breasts. If we accept the observation that cardiac output is maintained at or near peak level until delivery, the systemic vascular resistance must also continue low, since the mean arterial blood pressure does not rise during the latter part of pregnancy.

It has long been appreciated that the murmurs of aortic and mitral regurgitation can be altered by change in systemic arterial resistance. These murmurs will become louder following injection of vasopressor agents such as phenylephrine or methoxamine and will diminish in intensity after inhalation of a vasodilating drug such as amyl nitrite or the administration of a ganglionic blocking agent. The change in intensity of these murmurs directly reflects changes in the volume of mitral or aortic regurgitation. Wiggers and Maltby found that an increase in peripheral resistance caused an increase in the volume of aortic regurgitation in experimental animals. Rodbard and Williams, using an artificial model, and Braunwald and co-workers, using dogs, confirmed the observations of Wiggers and Feil that an increase in systemic vascular resistance increased the amount of mitral regurgitation. Increase in the degree of mitral regurgitation due to an augmentation of systemic vascular resistance induced by methoxamine has been documented in patients. Therefore, the decrease in intensity of the murmurs of aortic and mitral regurgitation may be explained on the basis of the decreased systemic vascular resistance during pregnancy.

Some patients whose murmurs of aortic or mitral regurgitation did not decrease during pregnancy had the onset or exacerbation of hypertension to account for the lack of change in these murmurs. In others, the reason for the lack of alteration of murmur with pregnancy is not clear. Jose and associates noted that methoxamine did not cause an increase in mitral regurgitation in patients thought to have fixed or rigid valves. Whether this observation is pertinent to the lack of change of the mitral regurgitation murmur in this series is not known. The few observations regarding the effect of pregnancy on murmurs other than those of aortic and mitral regurgitation serve only to suggest that the murmurs of mitral stenosis and tricuspid regurgitation may increase during pregnancy as they do with inhalation of amyl nitrite. The persistence of the ejection click in two patients who had murmurs of aortic regurgitation only when not pregnant suggests that this sign may be the only clue to the presence of aortic regurgitation in some pregnant patients with this lesion.

These observations have both diagnostic and therapeutic significance. It is taught that "organic heart disease may usually be excluded in the absence of a diastolic murmur, unequivocal cardiomegaly, a systolic murmur more than grade II in intensity, and severe arrhythmia." The present study indicates that organic heart disease must be suspected during pregnancy when there is a history of rheumatic fever or rheumatic heart disease, even in the absence of the murmur of aortic or mitral regurgitation. Under these circumstances, particular attention must be given to previous examinations before pregnancy. This study also suggests the importance of examining a patient post partum before stating that the patient has no auscultatory evidence of heart disease. If the physician wishes to determine the presence of aortic or mitral regurgitation during pregnancy, he may inject phenylephrine intravenously and listen immediately following infusion for these murmurs. Phenylephrine seems to be innocuous to the fetus. Vasoactive drugs, including phenylephrine, do not appear to cross the placenta in dogs or in sheep. Systemic hypertension is a relative contraindication to the use of this drug.

The murmurs of aortic and mitral regurgitation may be accentuated during squatting. The effect of this maneuver is to increase the systemic blood pressure. It is important to diagnose valvular disease during pregnancy, because of the hazard of subacute bacterial endocarditis occurring at
the time of delivery. If valvular heart disease is present or if there is a history of an organic murmur prior to pregnancy, the physician should prescribe antibiotic prophylaxis at the time of delivery and for several days thereafter. The diagnosis of subacute bacterial endocarditis or rheumatic fever may be suspected in women found to have the “appearance” of the murmur of mitral or aortic regurgitation after pregnancy (see case W.P.). Under these circumstances, the possibility should be considered and that the murmur disappeared or was missed during pregnancy.

The physician may underestimate the severity of the cardiac lesion if the patient is first examined during pregnancy, since one of the parameters for assessing the degree of mitral and aortic regurgitation is the intensity of the murmur. In surveys regarding the effect of pregnancy on the course of rheumatic heart disease, these observations must also be taken into account. It is interesting to speculate that one factor contributing to the favorable course of pregnancy in patients with aortic or mitral regurgitation may be the actual decrease in the degree of aortic or mitral regurgitation during pregnancy. This decrease in valvular regurgitation may in part compensate for the increase in the work of the left ventricle due to the hemodynamic demands of pregnancy.

The following two case reports are illustrative of the problems encountered before it was appreciated that the murmurs of aortic and mitral regurgitation may diminish during pregnancy.

Report of Cases

Patient W.P.

A 32-year-old female, gravida 7, para 6, was seen during her eighth month of pregnancy. At that time she complained of vague discomfort of her chest and cardiac consultation was requested. There was no history of rheumatic fever. Vital signs were as follows: The blood pressure was 110/60 mm Hg, and the pulse rate was 115/min and regular. The left ventricular impulse was displaced laterally and was more forceful than anticipated, even in pregnancy. The patient was examined by two cardiologists, neither of whom could hear a murmur suggestive of valvular heart disease. There was voltage criteria for left ventricular hypertrophy by electrocardiogram. A 6-foot PA roentgenogram of the chest showed mild cardiomegaly. In the absence of heart murmurs and the presence of cardiac enlargement, primary myocardial disease was thought to be present. The patient was seen 9 days prior to delivery, and again, no murmurs were heard. On this occasion blood pressure was 110/70 and pulse rate was 120/min. Delivery was uneventful and without complications.

Because of the findings of cardiomegaly and tachycardia, the patient was seen 3 weeks after delivery. At that time, her blood pressure was 140/80. A grade III/VI diastolic blowing murmur was heard. Because this murmur had not been heard previously, there was concern that she may have had a recent episode of acute rheumatic fever. Other possibilities included dissection of the aorta or endocarditis. She was hospitalized for further investigation. On admission, a blood pressure of 130/80 was recorded. Her pulse rate was 80/min and regular. She was afebrile. The jugular venous pulse and carotid pulse were of normal contour. The left ventricular apical impulse was in the fifth intercostal space at the mideclavicular line. The first heart sound was not accentuated. A grade III/VI systolic ejection murmur was present along the left sternal border, and a high-frequency diastolic blowing murmur of grade III/VI was heard along the left sternal border. Six blood cultures were negative. She remained afebrile. Her antistreptolysin (ASO) titer, C-reactive protein, CBC, sedimentation rate, LE cell preparation, and serum electrophoresis were all within normal limits. There was no evidence of enlargement of the ascending aorta by chest roentgenogram. Since there was no evidence to substantiate a diagnosis of acute rheumatic fever, aortic dissection, or endocarditis, the possibility was considered that the murmur of aortic regurgitation was not present during pregnancy because of the hypotension and tachycardia then present. To simulate these dynamic changes, auscultation was performed before and during the inhalation of amyl nitrite. The reflex tachycardia increased the pulse rate from 98 to 120. Amyl nitrate inhalation decreased the intensity of the diastolic murmur. Rheumatic fever prophylaxis was instituted. Although there was no history of rheumatic fever, this was the most likely etiology of the murmurs.

Patient S.R.

This 24-year-old woman was referred to the Cardiac Clinic of Georgetown University Medical Division for evaluation of possible heart disease. She had a history of dyspnea on exertion of 2 months’ duration. She had had rheumatic fever at
the ages of 12 and 19 years, although she was not
told of the presence of any heart disease at the
time of rheumatic fever.

On examination she had a blood pressure of
110/70 mm Hg; her pulse was 80 and regular.
There was no evidence of cardiomegaly by
examination. The first heart sound was split, the
second heart sound was single, and a grade II
ejection murmur was heard at the second left
interspace. Neither an apical systolic nor an aortic
diastolic murmur was heard. She was thought not
to have valvular heart disease. Antibiotic prophyl-
axis during delivery was not prescribed. Delivery
was uneventful. She was seen 4 months after
pregnancy. In the interim, she felt well and did
not have symptoms of rheumatic fever. The only
difference in cardiac examination, performed by
the same physician, was the presence of a grade
II/VI pansystolic murmur at the apex which
radiated to the axilla. No diastolic blowing
murmur was heard.

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**Bedside Medicine**

**Need for a Wider Horizon**

... The call for teaching at the bedside is old and there again is a demand to replace most lectures by bedside instruction. As I told you, I was not addicted to formal lectures myself but I have to point out that many aspects of clinical knowledge cannot be taught at the bedside. Prevention of diseases, in my opinion the highest form of medicine, cannot be taught at the bedside because the protected persons are well and walking around. Prenatal diseases which can be so devastating to the unborn child are another example. They cannot be taught at the bedside since neither the mother nor the embryo or fetus is in bed during the fateful days. At the bedside you can see a tiny sector of the area that must be known. But you have to learn to supplement these quick glimpses by intensive studies of books or with other devices.—From **WARKANY, JOSEF:** Convocation Address at the opening of the 150th year of medical classes at the University of Cincinnati, September 29, 1969. Medical Alumnal Bulletin of the University of Cincinnati 22: 1, Winter, 1969.
The Effect of Pregnancy on the Murmurs of Mitral and Aortic Regurgitation
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