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Differentiation of Ventricular Septal Defects from Mitral Regurgitation by Pulsed Doppler Echocardiography

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SUMMARY Forty youngsters with apical systolic murmurs, whose clinical evaluations did not allow conclusive differentiation between ventricular septal defect (VSD) and mitral regurgitation (MR), were studied by pulsed Doppler echocardiography (PDE). The PDE technique, which supplements M-mode echocardiography with Doppler flow detection, allowed determination of the site of turbulent blood flow in 39 of the 40 youngsters (97.5%). Twenty-one had PDE findings of VSD but no MR, and the 10 who underwent cardiac catheterization had VSD demonstrated. Seventeen had PDE findings of MR but no VSD, and the six who underwent catheterization were found to have MR, and no VSD. One younger had both VSD and MR demonstrated by PDE and catheterization, and one had no abnormalities detected by either PDE or invasive study. The sensitivity and specificity of PDE for determining the origin of troublesome apical murmurs has obvious clinical utility.

THE PRESENCE OF AN APICAL SYSTOLIC MURMUR is compatible with either a ventricular septal defect (VSD) or mitral regurgitation (MR). In many cases, the defect is mild and invasive studies are unwarranted. We here report a means of differentiating between VSD and MR by use of pulsed Doppler echocardiographic (PDE) equipment developed at this institution. Since the prognosis and management of patients having a VSD may differ significantly from those having MR, we believe the PDE technique, which supplements M-mode echocardiography with Doppler flow detection, has significant clinical utility.

Materials and Methods

The series includes 40 youngsters (22 female, 18 male, ages 1 month to 20 years, mean 6.6 years) with apical systolic murmurs whose clinical evaluations did not conclusively differentiate between VSD and MR. The series does not include patients whose clinical evaluations were typical for one diagnosis to the exclusion of the other. Eighteen youngsters underwent catheterization for evaluation of ventricular septal defect or mitral regurgitation, with significant precathe\oretization uncertainty as to whether or not the
other defect was absent. Their catheterization results and PDE findings were compared to establish initial sensitivity and specificity of PDE in distinguishing between VSD and MR. Except for one child subjected to catheterization elsewhere, the remainder of the series was comprised of youngsters whose defects were of insufficient severity to warrant invasive evaluation. Two youngsters were referred for evaluation having been previously labeled as having rheumatic mitral disease, and had been on rheumatic penicillin prophylaxis for some time. In both cases, the Jones criteria for the diagnosis of the supposed rheumatic episode were not satisfied in our opinion.

Using a previously described eighth generation research prototype echo and pulsed Doppler unit developed at the Center for Bioengineering at the University of Washington, or its commercially available offspring,* complete A and M-mode echocardiographic examinations and pulsed Doppler examinations were performed. Youngsters having catheterizations were examined during that hospitalization. The M-mode echocardiographic examination does not significantly differ from standard examinations performed on a routine basis throughout the country. For the purposes of this study, the M-mode examination provides certain anatomic information useful in localization of the pulsed Doppler sample volume, but provides little information useful in distinguishing between VSD and MR. Though the PDE examination has been described elsewhere, it will be briefly summarized here. The pulsed Doppler exam is performed in much the same fashion as the standard M-mode examination, both from suprasternal notch and precordium. The pulsed Doppler transducer emits a beam, with sampling gated at a single, but variable, position in the beam, and could be considered to function as a focused stethoscope. The sample volume has a teardrop shape with dimensions of approximately 2 × 4 mm. The sample volume can be positioned at any point along the beam, and the depth is displayed on both A and M-mode display. While the examination may well be guided by the A-mode echo, the system provides a simultaneous M-mode display for more conventional identification of structures. Also depicted on the M-mode recording is a spectral representation of the Doppler flow signal (fig. 1). This contains information concerning the quality (turbulent vs smooth) and direction (toward or away from the transducer) of the Doppler flow signal. The quality and direction of the flow signal are also assessed by a stereophonic audio output. When blood flow is laminar, the audio flow signal is smooth, occasionally tone-like, and the spectral flow signal is comprised of closely-oriented dots (fig. 1A). When flow is turbulent or nonlaminar, the audio signal is harsh, and the dots comprising the spectral signal are distinctly scattered (fig. 1B). The pulsed Doppler examination is done following a standard M-mode examination, and the Doppler sample volume is guided through the various cardiac chambers and great vessels, sampling direction and quality of blood flow at various sites.

The specific portions of the pulsed Doppler exam useful in distinguishing between VSD and MR are diagrammed in figure 2. The pulsed Doppler findings of ventricular septal defects depend upon the size of the left-to-right shunt. With moderately large shunts, turbulent flow may be encountered in the pulmonary arteries, and can be followed down across the pulmonary valve into the right ventricular outflow tract where turbulence may be maximal in the case of a high VSD. In apical or muscular defects, the turbulent jet may be encountered considerably further down the septum. The

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FIGURE 2. Cross section of the heart showing position of pulsed Doppler sample volume (shaded teardrop) in detection of A) mitral regurgitation, B) high VSD, and C) low VSD. CW = chest wall, RV = right ventricle, IVS = ventricular septum, LV = left ventricle, AO = aorta, LA = left atrium.

FIGURE 1. Representative PDE flow signals. ECG = electrocardiogram. Flow = Doppler flow record, with signals above the line indicating flow toward the transducer, below the line away from the transducer. Panel A shows normal systolic flow directed toward transducer. Note that the individual dots comprising the flow record are very closely organized, forming a distinct wave. Panel B shows moderately harsh systolic flow, the individual dots comprising the systolic portion of the record being distinctly scattered. Panel C shows harsh systolic and diastolic flow, with dots very scattered throughout systole and diastole.
Doppler sample volume is guided along the entire septum in search of turbulent flow. It is usually possible by remaining in the turbulent jet to then pass the Doppler sample volume through the defect into the left ventricle (fig. 3). In the smaller ventricular septal defects there may be minimal pulmonary artery turbulence, and indeed a thorough examination of the septum may be necessary to localize the turbulent jet and follow it through the septum. Following the turbulent jet through the septum is of some importance in evaluating turbulence localized to the right ventricular outflow tract, since this is the only way to eliminate a diagnosis of infundibular pulmonic stenosis. In the case of apical defects, following the jet through the septum is more difficult but confusion with infundibular pulmonic stenosis should not be a problem.

Considerable effort is sometimes required to angle the Doppler sample volume in the correct plane to follow turbulent jets through the septum. In the case of high, membranous defects, the thickness of septum that must be traversed or “threaded” is minimal, and angulation may not be much of a problem. With apical defects, however, the width of septum that must be “threaded” is considerably greater. This, coupled with the fact that most of the apical defects in this series were small, likely explains our inability to “thread” all of the apical jets through the septum. The ability of PDE to detect VSD depends upon the turbulence of blood flow through the defect, and hence factors which diminish turbulence (such as presence of large VSD with equal ventricular pressures, or elevations of pulmonary resistance) may limit the sensitivity of PDE in certain cases.

Mitral regurgitation is detected by Doppler sampling in the left atrium, behind the mitral valve, and moving the sample volume throughout the left atrium in an attempt to detect turbulent flow. If the degree of regurgitation is large, the turbulence is widespread. Conversely, if the degree of regurgitation is small, the jet may be quite small and found only in portions of the left atrium (fig. 4).

Results

Of the 18 youngsters who underwent catheterization, PDE indicated the presence of VSD but no MR in 10, MR but no VSD in six, both VSD and MR in one, and no VSD or MR in one. At catheterization, 10/10 with PDE evidence of VSD were found to have VSD, 6/6 with PDE findings of MR had MR, one child had catheterization documentation of both VSD and MR, and the remaining younger who had undergone catheterization elsewhere for clinically suspected VSD, had no evidence of shunt by hydrogen inhalation. PDE was then sensitive and specific in determining the site of turbulent blood flow in all 17 youngsters whose hemodynamic abnormality could be demonstrated by standard invasive study.

Of the 22 youngsters who did not undergo catheterization, isolated VSD was detected by PDE in 11. These 11, combined with the 10 with catheterization-proven VSD, make a total of 21 youngsters with PDE findings of VSD. Fifteen of the VSD jets were found low in the RV apex, and 12/15 were followed through the septum in low, apical position while six were followed through the high portion of the septum. The other eleven youngsters who did not undergo catheterization had PDE findings of MR but no VSD. These, combined with those with catheterization documented isolated MR give a total of 17 youngsters with PDE findings of MR but no VSD. Four of these 17 had M-mode echo evidence of leaflet prolapse, but in 13, the M-mode echo of the mitral valve appeared to be normal. Two youngsters carrying dubious diagnoses of rheumatic fever, in
whom the murmur had been felt by others to be mitral in origin, were shown to have apical ventricular septal defects, and no evidence of mitral regurgitation.

Discussion

The current series demonstrates that pulsed Doppler echocardiography has useful sensitivity and specificity in distinguishing between sources of turbulence which may be responsible for apical systolic murmurs. When clinical findings are characteristic, there is usually little difficulty in the clinical distinction between VSD and MR. However, when the VSD is low or apical, and when the MR is not massive, or when clicks do not accompany mitral regurgitation secondary to prolapse, or when auscultation in the postoperative period may be less than ideal, questions arise as to the origin of apical systolic murmurs. In this series evaluation by pulsed Doppler echocardiography was helpful in determining the site of turbulent blood flow in 39 of 40 patients, 97.5%. In the remaining patient, the site of turbulent flow was not detected by invasive or noninvasive means.

There are several situations in which pulsed Doppler echocardiography has been of significant clinical value in distinguishing VSD from MR. As in the majority of youngsters in this series, the magnitude of the defect in question is usually insufficient to warrant invasive study, and indeed may be so mild as to be difficult to demonstrate by invasive means. Still the distinction may be important. For example, a small muscular VSD is benign, has a very good prognosis, and there is a fair chance that the youngster may proceed to spontaneous closure of the defect. Mitral insufficiency on the other hand carries a less cheerful prognosis. In older children and young adults, mitral regurgitation due to prolapse may, in some cases, be associated with arrhythmias, adverse symptomatology, or even sudden death. In cases of mitral regurgitation not due to prolapse, the very long-term outlook may be worse than that for youngsters with small VSDs, as the deformed mitral valve could be expected to function less well with increasing age; whereas uncomplicated small VSDs are not thought to lead to any significant hemodynamic stress with increasing age.

The discovery of a murmur (especially apical) during a febrile illness may raise the question of rheumatic fever even though the Jones criteria are not fulfilled. While youngsters with a ventricular septal defect or congenital mitral regurgitation may develop rheumatic fever, knowledge that the apical murmur is that of a ventricular septal defect and not mitral regurgitation is of obvious utility. Since it is the presence of the murmur that may raise the question of rheumatic fever in an otherwise nonspecific febrile illness, it
would be of use to know by pulsed Doppler echocardiography whether or not the origin of the murmur in question is consistent with a rheumatic process. When there has been no firm basis for the diagnosis of rheumatic fever, and pulsed Doppler echocardiography shows only a VSD, removal of the diagnosis of rheumatic fever and discontinuation of rheumatic fever prophylaxis may be considered.

In cases where the severity of VSD or MR may approach the surgical range, invasive studies are more appropriate, and there is usually less difficulty in distinguishing between VSD and MR. Questions may remain, however, especially if the VSD is of endocardial cushion type, with associated mitral abnormalities, or in situations where left ventricular angiography performed via the transatrial route is associated with so-called catheter induced mitral insufficiency.

Pulsed Doppler echocardiography is sensitive and specific in determining the origin of troublesome apical systolic murmurs due to VSD or MR, and in the situations discussed extends noninvasive ultrasound diagnosis beyond the capabilities of M-mode echocardiography.

References

The Pulsed Doppler Coronary Artery Catheter

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SUMMARY A new catheter which measures instantaneous changes in coronary artery blood flow velocity is described. The linear relationship between flow velocity measured with the catheter and volume flow through small arteries is documented with a correlation coefficient of r = 0.99. Coronary flow velocity has been measured from the proximal right and left coronary arteries and aortic ostia of saphenous vein bypass grafts in patients undergoing diagnostic coronary arteriography. The increase in coronary flow following injection of contrast media in normal coronary arteries is similar to the increase in coronary flow reported following near-maximum exercise. This increased flow response following injection of contrast media is severely limited by coronary artery stenosis and may provide a useful method for assessing hemodynamically significant stenosis in patients with coronary artery disease.

THE SYMPTOMS of coronary artery disease are usually precipitated by some type of hemodynamic stress. However, of all the laboratory techniques commonly used to assess the presence or severity of coronary artery disease, only the exercise electrocardiogram imposes a hemodynamic stress on the cardiovascular system. Since the symptoms of coronary artery disease presumably arise from an inability to adequately increase flow through stenosed coronary arteries, diagnostic tests which more clearly define the flow limiting characteristics of specific coronary artery lesions might be of considerable help in evaluating patients with this disease.

The significant increase in coronary blood flow that follows selective coronary artery injection of contrast media has been appreciated for several years. Initial studies were performed in acute, open-chest animal preparations.1 4 Recently, coronary blood flow measurements in patients undergoing cardiac catheterization have demonstrated a similar increase in coronary blood flow following selective coronary arteriography.7 8

Gould et al., using an open-chest animal preparation, have demonstrated the potential usefulness of this flow response in assessing the critical nature of coronary artery stenosis.4 In their study, resting coronary flow was not decreased until the stenosis exceeded 85%. However, peak flow following selective coronary injection of contrast media was limited by a 50% stenosis. It was postulated that a quantitative measure of this post contrast media flow response would therefore provide a meaningful assessment of the flow limiting nature of a specific coronary artery lesion.

The clinical application of this contrast media-induced hyperemic response in man requires an easily applied method for measuring coronary artery blood flow. The method must also provide a measure of rapid changes in coronary blood flow since flow is constantly changing following injection of contrast media, and a prolonged steady state is never maintained during the hyperemic response phase. During the past 25 years, a variety of techniques for measuring coronary artery blood flow in conscious man have been

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