Pulsed Doppler Echocardiography in Cardiac Diagnosis

PETER WILDE, BM, MRCP(UK), FRCR
Consultant Cardiologist, Bristol Royal Infirmary

DAVID PITCHER, MB, MRCP(UK)
Consultant Cardiologist, Hereford County Hospital

The Doppler principle allows blood flow to be measured within the body by the analysis of the altered frequency of returned ultrasound waves reflected from a moving column of blood. Pulsed Doppler echocardiography (PDE) allows sampling of flow at a specific depth within the patient by appropriate timing in the system[1]. Some modern ultrasound instruments can combine the PDE system with a sector scanner so that flow can be sampled at any selected site within a two-dimensional image[2]. This capability has considerably increased the flexibility of ultrasound diagnosis in heart disease because a single diagnostic examination can incorporate the complementary modalities of two-dimensional (2D), M-mode and Doppler echocardiography.

The Doppler information is returned initially as an audio signal which has a characteristic sound in different situations but, for full analysis, the wave form is computer-processed by the Fourier technique which allows a graphic display of the frequency components present within the signals[3]. These frequency components are closely related to the blood velocity.

The angle between the examining beam and the direction of flow is particularly important. The most accurate Doppler information is obtained when the direction of the examination beam is close to the direction of blood flow. Turbulent flow can, however, be detected from many directions because the nature of the flow is multidirectional.

Inspection of the spectrally analysed Doppler signal together with the two-dimensional image and the simultaneous ECG allows several important features of blood flow to be assessed, namely: (a) site of flow within the image; (b) direction of flow (towards or away from transducer); (c) timing of flow (systole or diastole); (d) changing flow patterns within systole or diastole; (e) laminar or turbulent flow; (f) unusually high or low velocity flow, and (g) unusually high or low intensity signals.

Diagnostic PDE features of normal and abnormal cardiac blood flow have been reviewed by various workers[4-8]. Grading of severity of many lesions has also been achieved[6, 9-11].

This article assesses the diagnostic contribution of this new modality in a busy clinical echocardiography department with referrals from both cardiological and non-cardiological sources.

Methods

Equipment and Techniques

All scans were performed in the Radiology Department of the Bristol Royal Infirmary on an ATL (Advanced Technology Laboratories) Mark 600C mechanical sector scanner with a PDE facility. The PDE ‘sample volume’ (region of Doppler examination) was controlled by a

Fig. 1. Suprasternal trace from a normal ascending aorta. The initial increase in velocity is shown by a thin line which indicates laminar flow.
movable marker on a radial line within the image. The instrument was also capable of recording M-mode traces. A simultaneous ECG was always recorded.

Adults were studied using a medium focus 3 MHz transducer and children with a medium focus 5 MHz transducer. A small non-imaging 3 MHz transducer was also used when access to the suprasternal notch was difficult with the larger imaging transducer.

Flow through valves was assessed by positioning the 'sample volume' in line with the flow leaving the valve orifice. Normal flow through the valves was laminar in quality and low in velocity. Valvar stenosis was characterised by high velocity turbulent flow. The degree of stenosis was not assessed in this study (Figs 1-5).

Valvar regurgitation was diagnosed by the detection of high velocity turbulent flow proximal to a closed valve (Figs 3, 6-8). Severity of regurgitation was assessed by mapping the regurgitant jet with the movable sample volume. Low intensity turbulence only detectable near to the closed valve was graded as mild regurgitation. If turbulence was detectable up to halfway into the receiving chamber, moderate regurgitation was recorded. High intensity turbulence detected throughout most of the receiving chamber was graded as severe regurgitation. 2-D and M-mode assessment of the left ventricle was sometimes an additional help in assessing severity of regurgitation.

In paediatric cases the sample volume was moved along the right side of the interventricular septum to detect any turbulent high velocity flow arising from a ventricular septal defect[8, 12]. Pulmonary arterial flow was evalua-
ed to detect diastolic turbulence caused by a patent ductus arteriosus\cite{13} or systolic high velocity turbulence due to pulmonary stenosis. The flow in the descending portion of the aortic arch was assessed in order to detect the high velocity turbulent flow of coarctation. Valve function was assessed as described above for adults.

**Patients**

All patients having a complete cardiac ultrasound examination in a six-month period were reviewed (Fig. 9). Thirty patients examined without PDE and 32 patients with inadequate data were excluded from the series.

The 294 patients comprised 75 catheterised cases and 219 non-catheterised cases. In the catheterised group 32 adults had ultrasound examination and catheterisation within seven days of each other; 43 paediatric cases had ultrasound and catheterisation on the same day. In the latter group ages ranged from five days to 21 years with a median age of two years. Of the 219 adult patients who were not catheterised, 125 were referred by cardiologists and 94 by other clinicians.
Results

Adult Catheterised Patients (32 cases)

Table 1 compares catheterisation (and angiography) with PDE in the diagnosis of mitral and aortic valve stenosis and regurgitation.

In only one ‘false negative’ case of mitral regurgitation was a lesion of moderate severity missed by PDE. In the remaining eight ‘false negative’ and ‘false positive’ cases of aortic and mitral regurgitation the discrepancies were all due to lesions judged by angiogram or PDE to be mild. No case of regurgitation judged severe by angiography was missed or considered mild by PDE.

All stenotic lesions were correctly identified.

Paediatric Catheterised Patients (43 cases)

Table 2 shows the diagnosis of simple malformations by cardiac catheterisation and angiography, compared with diagnosis by PDE and 2-D techniques. Complex abnormalities of connection and situs are not included in this table.

In no diagnostic category was any false positive diagnosis made by PDE.

A number of additional aspects of PDE diagnosis were apparent on review of these and the other cases in this group.

(a) PDE techniques are very sensitive in detecting variations from the normal flow patterns. Mild increased turbulence and velocity of flow were detected in a variety of cases where the invasive data showed a mild abnormality to be present. This group included three cases of surgically corrected coarctation of aorta, one case of postoperative transposition of the great arteries with mild obstruction of the superior systemic venous channel, and one case of mild pulmonary stenosis in transposition of

Table 1. Comparison of cardiac catheterisation and angiography with PDE in the diagnosis of aortic and mitral valve lesions (32 cases).

|                      | Total cases with positive catheter diagnosis | Positive Catheter Diagnosis | Negative Catheter Diagnosis |
|----------------------|---------------------------------------------|-----------------------------|-----------------------------|
|                      | Positive PDE diagnosis | Negative PDE diagnosis | Positive PDE diagnosis | Negative PDE diagnosis |
| Mitral or aortic stenosis | 18 | 18 | 0 | 0 | 14 |
| Aortic regurgitation | 16 | 14 | 2 | 0 | 5* |
| Mitral regurgitation | 19 | 15 | 4 | 3 | 7** |

*Aortography in 21 cases
**Left ventriculography in 29 cases
the great arteries. In none of these cases did the pulsed Doppler echocardiography technique suggest a more severe lesion than was actually present.

(b) In two cases of pulmonary atresia, one with a small patent ductus arteriosus and the other with aorto-pulmonary collaterals, there was low-intensity continuous flow in the hypoplastic main pulmonary artery.

(c) In four out of nine atrial septal defects (ASDs), left to right flow was detectable across the septal defect. The low velocity of this flow made it hard to reliably diagnose ASDs by using PDE. All the ASDs were detected on 2-D imaging.

(d) In the one case of Fallot’s tetralogy, right to left shunting across the VSD was detected.

(e) In the case of congenital mitral regurgitation the lesion was easily detected using PDE.

(f) In a patient with isolated pulmonary hypertension and right heart failure PDE clearly showed tricuspid and pulmonary regurgitation.

(g) In one case there was an aneurysm of the membranous septum clearly evident on the 2-D images. This was not associated with any turbulent flow on the PDE examination, and angiography confirmed that the septum was intact.

(h) In eight cases with complex disease and large VSDs of various types, the anatomy of connections and malformations was determined by using 2-D images. The VSDs were all clearly visualised. Useful PDE signals were hard to obtain from the ventricular cavities in these patients, because of the relatively low-velocity flow across the defects.

Table 2. Comparison of cardiac catheterisation and angiography with PDE and 2-D echocardiography in the diagnosis of simple congenital heart malformations (43 patients).

| Patients with positive catheter diagnoses | Catheter positive diagnosis | PDE positive diagnosis | 2-D diagnosis |
|------------------------------------------|-----------------------------|------------------------|--------------|
| VSD                                      | 11                          | 10                     | 5            |
| PDA                                      | 7                           | 6                      | 2            |
| Coarctation                              | 4                           | 3                      | 4            |
| Aortic stenosis                          | 2                           | 2                      | 2            |
| Subaortic stenosis                       | 2                           | 2                      | 0            |
| Pulmonary stenosis                       | 6                           | 2                      | 4            |

1 Anatomy of interventricular septum otherwise normal.
2 Two separately identified VSDs in one case. Muscular VSD missed in one post-operative case of Fallot’s tetralogy due to non-examination of septum (see discussion).
3 Suspected in a further 3 by the presence of irregularity of the membranous septum.
4 One PDA missed by PDE was angiographically tiny and not detected by saturations.
5 Suspected but not certain in a further 3 cases.
6 PDE examination omitted from coarctation region in 1 case.
7 Subaortic stenosis suspected from 2-D images in both cases but not certain. Aortic valve seen to be normal in both cases.
8 Three cases of pulmonary stenosis also had VSD. Turbulence in pulmonary artery in systole could have been due to VSD so confident diagnosis not possible.
9 Detail of pulmonary valve not well seen in 2 cases.

Table 3. Comparison of clinical findings with pulsed Doppler echocardiography in the diagnosis of important mitral and aortic regurgitation. (The figures in brackets are cardiological referrals.)

|                      | No. of cases | At least moderate lesion by PDE | Clinically diagnosed important lesion, PDE negative |
|----------------------|--------------|--------------------------------|-----------------------------------------------|
| Aortic regurgitation | (157)        | (14)                           | (0)                                           |
| Mitral regurgitation | (157)        | (6)                            | (1)                                           |

All adult cases, including catheterisation patients, are reviewed in this group.

Aortic and Mitral Regurgitation. Our results in the catheterisation group of adults (see Table 1) show some discrepancy between PDE and angiogram in the diagnosis of mild valvar regurgitation (especially mitral). The results in moderate or severe regurgitation are, however, extremely accurate and furthermore are clinically most relevant. The clinical findings in cases with moderate or severe regurgitation (detected by PDE) are shown in Table 3.

Twenty-nine cases of important valvar regurgitation diagnosed by PDE were undetected clinically. In only five of these cases was the missed lesion an isolated abnormality, the remaining cases all having at least one other valve lesion.

In a further four cases clinically suspected important valvar regurgitation was not confirmed by PDE. Although the lesions may have been missed by PDE, it is considered probable that these four lesions were incorrectly assessed clinically.

Thus in 251 patients there were 33 valve lesions that may well have been assessed incorrectly at clinical examination.

Tricuspid Valve Disease. In three cases, tricuspid stenosis was diagnosed by PDE, the diagnosis being made by 2-D images in two cases. The clinical signs did not allow a confident diagnosis in any of the three cases, all of whom had mitral valve disease. Three clinically undiagnosed cases of important tricuspid regurgitation were diagnosed by PDE. In all these cases of tricuspid valve disease there was associated mitral valve disease.

Prosthetic Valve Function. Thirty-four patients with prosthetic valves were assessed but none was catheterised in the review period. One mitral xenograft was suspected of stenosis by the detection of unusually high-velocity flow through the valve, which was sustained throughout diastole. In two cases, clinically severe mitral regurgitation
was diagnosed by PDE and in one of these cases a flail xenograft leaflet was identified on the 2-D study.

In six cases of clinically diagnosed prosthetic aortic regurgitation, the diagnosis and grading were confirmed by PDE. In one further case of a ‘stuck’ aortic prosthesis, the malfunction was shown on 2-D images and the clinical diagnosis of severe aortic regurgitation was confirmed by PDE. In five more cases, moderate aortic prosthetic regurgitation was shown by PDE but was not detected clinically. In two of these a confident statement had specifically been made that there was no aortic regurgitation.

**Differential Diagnosis.** In seven cases differentiation between mitral regurgitation and aortic stenosis was sought. In one case neither lesion was detected by 2-D or PDE. In three cases PDE showed mitral regurgitation and excluded aortic stenosis and in the remaining three cases PDE showed no mitral regurgitation and aortic stenosis was shown by imaging and PDE techniques. In two cases the alternative diagnoses of mitral regurgitation or ventricular septal defect were raised. In one there was clear PDE evidence of mitral regurgitation without evidence of a septal defect. In the second a clinically undetected ASD was shown on 2-D images and PDE revealed associated tricuspid regurgitation.

**Discussion**

It is apparent that the pulsed Doppler techniques are best integrated into the normal echocardiographic examination. In many instances the Doppler technique serves only to confirm clinical as well as M-mode and 2-D diagnoses, for example in mitral stenosis (although Doppler examination can be used to obtain accurate quantitation). In other cases, PDE will provide diagnostic information that is not available by other means. It may be difficult in a case of aortic stenosis to confirm or exclude the presence of co-existing mitral regurgitation but PDE can do this quite specifically.

Satisfactory images cannot always be achieved with M-mode and 2-D ultrasound, especially from the left parasternal window. When images are poor the recording of a normal mitral flow signal from the apex can allow confident exclusion of mitral stenosis. Aortic stenosis can be similarly confirmed or excluded from the suprasternal position. In our own series, all cases of mitral or aortic stenosis diagnosed by catheter were also shown by PDE. In the remaining patients 2-D and PDE recordings showed full agreement in the diagnosis of these lesions.

The PDE technique has proved very useful in the detection and approximate grading of valvar regurgitation. PDE is particularly sensitive in the diagnosis of aortic regurgitation, published results showing a diagnostic sensitivity of 94–95 per cent and a specificity of 82–100 per cent[14,15]. In our own small group the results are similar, with a sensitivity of 88 per cent and a specificity of 100 per cent. This cannot be matched by any other diagnostic technique apart from angiography which is currently the most accurate technique available for the diagnosis of regurgitant valves. This level of diagnostic accuracy has been shown in our own and other work[6] to be superior to clinical examination. The detection of aortic regurgitation by fluttering of the mitral valve on M-mode studies cannot attain this accuracy[17].

PDE is also very accurate in the diagnosis of mitral regurgitation, with a sensitivity of 91–94 per cent and a specificity of 89–94 per cent[10,14]. Our own results in this group are lower, sensitivity being 80 per cent and specificity being 70 per cent. On further analysis, however, the results in all but one of the false negatives have been due to estimates of mild mitral regurgitation either clinically or by PDE. If haemodynamically important lesions only were calculated (i.e. moderate or severe) then our results would show a sensitivity of 95 per cent and a specificity of 100 per cent. The scanning technique can be more difficult for mitral regurgitation, and our own experience suggests that as familiarity with the technique increases, the diagnostic accuracy will improve. Mitral regurgitation, especially of mild degree, is well known to fluctuate from day to day with varying clinical circumstances. We have also found PDE to be useful after catheterisation in assessing possible catheter-induced mitral regurgitation.

The evaluation of right-sided lesions has proved interesting. Tricuspid regurgitation may be clinically undetectable except in severe cases but is easily diagnosed by PDE. This is important because tricuspid regurgitation is a frequent accompaniment of mitral valve disease and left ventricular impairment.

Our results show that PDE is particularly valuable in patients with multiple valvar lesions. PDE is no more or less accurate for each specific lesion in these cases but accurate clinical diagnosis becomes increasingly hard as the number of lesions rises. In our series the majority of 'missed' lesions were in patients with more than one valvar abnormality.

Prosthetic valve evaluation is becoming increasingly important and PDE is a great aid in these patients for whom further invasive investigation may be more hazardous than usual. The mild stenotic pattern of prosthetic valves must be recognised. The echogenic material of prostheses can make scanning more difficult, but careful technique will usually show mitral or aortic regurgitation. It is now also possible to distinguish between paravalvar and valvar leaks[14].

The study of acute and severe haemodynamic upset such as rupture of the ventricular septum or papillary muscle after myocardial infarct, infective endocarditis or the acute derangement of a prosthetic valve is well suited to PDE[18]. In such cases the correct clinical diagnosis is often suspected, but echocardiography with 2-D imaging and PDE can provide rapid confirmation and allow surgical intervention in some cases without subjecting critically ill patients to invasive investigation.

Doppler studies are a logical extension of paediatric echocardiography. The anatomical accuracy of diagnosis is now well recognised but Doppler studies complement the 2-D images in the conditions where imaging resolution is not totally accurate. Small ventricular septal defects, patent ductus arteriosus and coarctation can be hard to visualise or exclude by 2-D studies[19] but it is
just such lesions with their high velocity turbulent jets that are well suited to diagnosis by PDE.

The complementary nature of imaging and PDE examination is well suited to the diagnosis of some difficult lesions. In their description of the diagnosis of coronary artery fistulae, Miyatake et al. [20] emphasised the complementary roles of 2-D and Doppler techniques. Uncertain anatomical structures can often be more clearly evaluated if flow signals from them are obtained. In our series a case of membranous ventricular septal aneurysm without interventricular communication could not have been diagnosed with certainty on 2-D studies alone.

It is notable that the errors made in our series have usually been as a result of inappropriate scanning technique rather than failure to interpret the Doppler findings. It is for this reason that we feel even more accurate results will be possible as experience in PDE grows.

Quantitative Doppler evaluation of valvar gradients, intracardiac pressures and volume flows (cardiac output and shunt evaluation) are all exciting prospects that are now being evaluated. They are soon likely to join qualitative Doppler echocardiography in the routine non-invasive assessment of cardiac patients.

Acknowledgements

We are indebted to Miss J. Hugh and Miss N. Eberle for typing the manuscript. We are also grateful to our consultant cardiological colleagues in the Bristol Royal Infirmary for allowing us access to their patients’ clinical records.

References

1. Wells, P. N. T. (1969) Medical and Biological Engineering, 7, 641.
2. Griffiths, J. M. and Henry, W. L. (1978) Circulation, 57, 925.
3. Goldberg, S. J. and Sahn, D. J. (1982) American Journal of Cardiology, 50, 1394.
4. Richards, K. L., Cannon, S. R., Crawford, M. H. and Sorensen, S. G. (1983) American Journal of Cardiology, 51, 1122.
5. Veyrat, C., Kalmanson, D., Farjon, M., Manin, J. P. and Abitbol, G. (1982) British Heart Journal, 47, 596.
6. Hhatle, L., Angelson, B. A. and Tromsdal, A. (1980) British Heart Journal, 43, 284.
7. Thiuliez, C., Theroux, P., Bourassa, M. G. et al. (1980) Circulation, 61, 381.
8. Stevenson, J. G., Kawabori, I., Dooley, T. and Guntheroth, W. G. (1978) Circulation, 58, 322.
9. Holen, J. and Simonson, S. (1979) British Heart Journal, 41, 529.
10. Veyrat, C., Ameur, A., Bas, S., Lessanz, A. and Abitbol, G. (1984) British Heart Journal, 51, 130.
11. Diebold, B., Peronneau, P., Blanchard, D. et al. (1983) British Heart Journal, 49, 167.
12. Magherini, A., Azzolina, G., Weichmann, V. and Fantini, F. (1980) British Heart Journal, 43, 143.
13. Stevenson, J. G., Kawabori, I. and Guntheroth, W. G. (1980) Catheterisation and Cardiovascular Diagnosis, 6, 255.
14. Quinones, M. A., Young, J. B., Waggoner, A. D., Ostojic, M. C., Ribeiro, L. G. T. and Miller, R. R. (1980) British Heart Journal, 44, 612.
15. Veyrat, C., Lessana, A. et al. (1983) Circulation, 68, 998.
16. Giobanu, M., Abbasi, A. S., Allen, M., Hermer, A. and Spellberg, R. (1982) American Journal of Cardiology, 50, 339.
17. Pridie, R. B., Benham, R. and Oakley, C. M. (1971) British Heart Journal, 33, 296.
18. Richards, K. L., Hoekenga, D. E., Leach, J. K. and Blaustein, J. C. (1979) Chest, 76, 101.
19. Goldberg, S. J., Allen, H. D. and Sahn, D. J. (1980) Paediatric and Adolescent Echocardiography. (2nd ed) Chicago: Year Book Medical Publishers.
20. Miyatake, K., Okamoto, M., Kinoshita, N., Fusejima, K., Sakakibara, H. and Nimura, Y. (1984) British Heart Journal, 51, 508.