The clinical assessment of Doppler cardiac ultrasound in valvular heart disease

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Doppler ultrasound of the heart has been available for some years and may be used to evaluate cardiovascular function in a variety of ways; the measurement of transvalvular pressure gradient is of particular value. This physiological information on blood flow velocity is complementary to that obtained by cross-sectional echocardiography [1,2]. Recently, there have been advances in technology, with Doppler apparatus becoming less expensive, more widely available and simpler to use and interpret. We have assessed the use of continuous and pulsed wave Doppler with echocardiographic imaging in the management of 100 consecutive patients with valvular heart disease.

Patients and methods

Patients

One hundred consecutive patients referred to this hospital for the assessment of valvular heart disease formed the study group. Complex congenital and infant cases were excluded. Of the 100 patients, 52 had the lesion of aortic stenosis, 44 had aortic regurgitation, 26 had mitral stenosis, and 44 had mitral regurgitation. The mean age of the patients was 52 ± 11 years; there were 36 women and 64 men. The aetiology of valve disease was rheumatic in 44, congenital in 11, ischaemic in 8 and calcific aortic stenosis in 37. Ten patients had prosthetic heart valves (5 mitral and 5 aortic). Cardiac catheterisation and Doppler studies were performed within the same hospital admission, usually within 24 hours.

Doppler examination

The Doppler studies (both pulsed and continuous wave) were performed using Irex Systems III apparatus (2.5 MHz transducer) with spectral analysis of signals. All available acoustic windows were used which included right and left parasternal, subcostal, apical and subcostal positions. Doppler ultrasound was combined with cross-

sectional imaging for the initial examination and measurement of pulsed wave Doppler velocities. The continuous wave apparatus was used to measure velocities which exceeded the limit of pulsed wave systems. The limit depends upon the depth of the sample volume but in most adults it is less than 2 m/s and in the vast majority, measurements of continuous wave were made using a 5 MHz dual crystal transducer. From the recordings of Doppler spectral analysis, the maximum velocity was measured using a ruler.

Calculations

Transvalvular pressure gradient was calculated by the modified Bernoulli equation [2]:

Pressure gradient in mmHg \times V²

where V is the maximum transvalvular blood velocity in metres/second. This was applied to valvular gradients and to calculate the transtricuspid gradient. To obtain right ventricular systolic pressure, and therefore pulmonary artery systolic pressure (in the absence of pulmonary stenosis), the right atrial pressure measured clinically was added to this [1]. The mitral valve area in patients with mitral stenosis was calculated from pressure half-time: this is the time taken for the peak velocity of transmitral flow to decay by 50 per cent. The severity of mitral stenosis shown by Doppler was arbitrarily graded mild if valve area was less than 2 cm², moderate if less than 1.5 cm² and severe if less than 1 cm². Valvular regurgitation was assessed semi-quantitatively using standard methods which included mapping of the regurgitant jet and the density of spectral signals obtained [2].

Cardiac catheterisation

Aortic stenosis was assessed by the peak-to-peak catheter withdrawal gradient, which was defined as of sufficient severity to require aortic valve replacement if equal to or greater than 50 mmHg. The severity of aortic regurgitation was graded by an independent observer on a four-point scale using contrast ciné-aortography [3]. The severity of mitral stenosis was arbitarily graded by the

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mitral valve area as mild stenosis > 2 cm², moderate 1.5-2 cm² and severe less than 1 cm². The severity of mitral regurgitation was graded from the left ventriculogram as mild, moderate or severe by an independent observer. The systolic pulmonary artery pressure was measured by right heart catheterisation.

Results

The sensitivity and specificity of Doppler ultrasound in the detection of various cardiac lesions is shown in Table 1. Doppler ultrasound was sensitive in detection of all valvular lesions with a specificity exceeding 75 per cent.

Table 1. The sensitivity and specificity of Doppler cardiac ultrasound in valvular heart disease. A total of 218 lesions were assessed in 100 patients (64 male, 36 female, mean age 52 years).

Lesion category	Doppler	
	Sensitivity	Specificity
	(%)	(%)
Aortic stenosis $(n = 52)$	93	82
Aortic regurgitation $(n = 44)$	94	75
Mitral stenosis $(n = 26)$	95	83
Mitral regurgitation $(n = 44)$	90	90
Pulmonary hypertension $(n = 33)$	70	90

Aortic valve disease

Aortic stenosis was suspected in 52 patients of whom three were excluded because of technically inadequate Doppler signals. The aortic valve gradient at cardiac catheterisation varied from 10 to 160 mmHg and that obtained by Doppler from 10 to 152 mmHg. Correlation of the Doppler peak instantaneous gradient with the catheter peak-to-peak gradient yielded r = 0.88, (p < 0.001). Doppler ultrasound correctly identified all patients with surgically significant aortic stenosis (Fig. 1), and

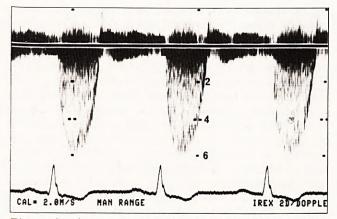


Fig. 1. Continuous wave Doppler ultrasound of the aortic value from the apex. A high-velocity jet of 6 m/s (peak gradient 144 mmHg) representing severe valvular aortic stenosis can be seen.

suggested mild aortic stenosis (gradient less than 36 mmHg) in four patients in whom none was apparent at catheterisation.

Aortic regurgitation. Comparison of Doppler to aortographic grading showed that in 23 (56 per cent) they were similar and in 14 (32 per cent) they differed by one grade. In five, the difference in grading was two or more; in three cases aortic regurgitation was detected in the absence of a diastolic murmur and aorto-left ventricular reflux of contrast medium. By Doppler there was no systematic over- or under-estimation of regurgitant flow.

Mitral valve disease

Mitral stenosis was suspected in 41 patients of whom 38 had evidence of this on cardiac catheterisation. The Doppler and catheterisation grading was similar in 29 (66 per cent) and differed by one grade in 13 (29 per cent (Fig. 2).

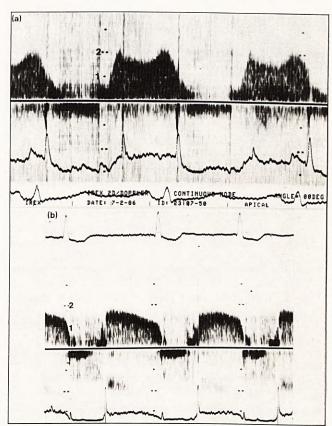


Fig. 2. (a) Continuous wave Doppler ultrasound of a severely stenotic rheumatic mitral valve from the apex. The maximum velocity is increased from the normal of approximately 1 m/s to 2 m/s. The pressure half-time is grossly prolonged and a prominent A peak of atrial systole is seen in this patient in sinus rhythm. (b) Pulsed wave Doppler of severe rheumatic mitral stenosis in atrial fibrillation from the apex. There is similar increase in maximum velocity and prolonged pressure half-time but the A peak is lost.

Mitral regurgitation was readily detected in the 44 patients with this suspected lesion. The Doppler detected mitral regurgitation in four patients without a pansystolic murmur or evidence of left ventricular-left atrial reflux of contrast medium. There was no obvious systematic error in Doppler assessment of mitral regurgitation, although a trivial jet of regurgitation was frequently detected when not suspected either angiographically or clinically.

Pulmonary artery pressure measurement

The systolic pulmonary artery pressure was assessed in all 33 patients in whom a right heart catheterisation was performed. No tricuspid regurgitation was found in seven patients, of whom five had had normal pulmonary artery pressure (mean pulmonary artery pressure 20 mmHg or less). In the remaining 26 patients, the correlation of Doppler to catheter systolic pulmonary artery pressure was r = 0.96, (p < 0.001) (Fig. 3).

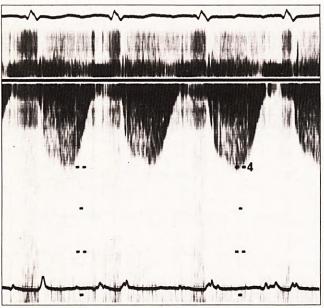


Fig. 3. Continuous wave Doppler ultrasound of tricuspid regurgitation from the left parasternum. The peak velocity exceeds 4 m/s representing a right ventricular and systolic pulmonary artery pressure of 64 mmHg.

Suspected prosthetic valve dysfunction

Suspected prosthetic valve dysfunction was noted in four homografts, three xenographs, and three Starr-Edwards valves. Valvular regurgitation was demonstrated by Doppler in two homografts in the aortic position, and two xenographs and one Starr-Edwards in the mitral position. Obstruction with apparent valve stenosis was seen in one mitral and one aortic tissue valve replacement.

Discussion

This study has evaluated the clinical usefulness of Dopp-

ler ultrasound in 100 patients undergoing routine noninvasive and invasive cardiac investigations. Crosssectional echocardiography is now in widespread clinical use and yields an anatomical image. The Doppler method, by contrast, provides haemodynamic information and depends upon the alteration in frequency imparted to the incident beam by the moving red blood corpuscles. Doppler is accurate in assessing aortic stenosis, showing a good correlation with the peak-to-peak gradient obtained by cardiac catheterisation (r = 0.88, p < 0.001); of considerable clinical importance was the high specificity (82 per cent) when separating 'surgical' from 'non-surgical' cases [4,5]. This cannot be achieved by clinical examination or other non-invasive methods such as cross-sectional echocardiography. For physicians without ready access to cardiac catheterisation, Doppler ultrasound provided reliable information in elderly patients with coexisting hypertension and immobile calcified valves in whom clinical signs and imaging is often unhelpful, as well as young patients. There is, however, one important pitfall with Doppler assessment of all valvular lesions which is particularly important in aortic stenosis. The high-velocity jet may be localised and eccentric, and to determine the peak velocity, multiple ultrasound windows may need to be interrogated [6,7]. To obtain diagnostic quality traces requires skill and training, particularly using a continuous-wave dual crystal transducer; failure to demonstrate a high-velocity jet in aortic stenosis by an inexpert operator may lead to an inadequate diagnosis.

The assessment of pulmonary artery pressure is central to the management of many forms of heart disease. Doppler detected tricuspid regurgitation is almost invariably found in pulmonary hypertension (often when clinically inapparent) and this provides a means of measuring the transtricuspid gradient [1,8]. As Doppler and imaging echocardiography can reliably exclude pulmonary stenosis, systolic pulmonary artery pressure can be calculated. Since this estimate of pulmonary artery pressure can be readily made and appears reliable it should provide a means to follow up patients with heart disease at risk of pulmonary hypertension. Mitral stenosis may be readily assessed by cross-sectional echocardiography which allows non-invasive measurement of mitral valve area [9]. Doppler ultrasound also facilitates a calculation of mitral valve area by the pressure half-time method; this probably adds little to the clinical assessment of most patients but is particularly useful following mitral valvotomy and stenosis of prosthetic tissue valves [10,11]. Imaging echocardiography may only provide indirect signs of regurgitant valvular flow by detecting valvular abnormalities and the enlargement of the left ventricle. However, Doppler ultrasound may directly interrogate regurgitant flow and the sensitivity of Doppler in detecting mitral and aortic regurgitation was 90 per cent and 94 per cent respectively. However, while sensitive in detection, quantitative assessment is difficult and can be misleading. Angiographic and Doppler assessment of severity concurred in only 56 per cent of aortic regurgitation and 60 per cent of cases of mitral regurgitation. The methods we used were semi-quantitative and subjective

but more refined Doppler techniques, such as comparing aortic and mitral valve orifice blood flow and the use of colour flow mapping, allow for greater accuracy in assessment of regurgitant lesions [12–14]. We suggest that Doppler ultrasound adds a new diagnostic technique that complements existing non-invasive methods and will replace cardiac catheterisation in the assessment of valvular lesions as it allows outpatient assessment in a District General Hospital, of many cardiac lesions previously requiring referral to specialist units.

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Naming diseases

In 1839, the Registrar General's office was set up in Somerset House and made ready to receive the certificates giving the cause of every death. Lister, the first Registrar General, was known for his writing of plays and novels but not for his knowledge of statistics. So he sought the advice of Sir James Clark, physician to Queen Victoria.

Clark, with his interest in chest disease, had employed William Farr to write most of his book on tuberculosis and so wrote to Lister recommending Farr as 'a gentleman of the medical profession whose scientific and intimate acquaintance with statistical enquiries were ample pledges of his peculiar fitness for the post.' Clark was right in his judgement, although Farr only held the licence of the Apothecaries, was a failed general practitioner and had managed to survive by medical journalism and taking in lodgers. He proved to be a triumphant success in the Registrar's office, developing a classifica-

tion of disease that was echoed in the nomenclature devised by WHO. Farr tried and failed to get an international classification of disease agreed at the International Statistical Conferences held in 1853 and 1860. At the latter conference, held in London, delegates visited the house of Florence Nightingale, where she received them while reclining on her couch.

Farr continued his good work until 1880. However, he was not without critics. He came under attack in the hot summer of 1868 when infantile gastroenteritis killed many English infants. Under Farr's classification these deaths were recorded as due to 'cholera'.

Everyone knew that this really meant 'diarrhoea' and not Asiatic cholera but the Spanish and Portuguese government issued a decree imposing a ten-day quarantine on all vessels from any port in Britain or its dominions. Self-styled British patriots found this insult hard to bear.

Health planning

An English schoolboy's memory of the French Revolution is likely to be of the terror, not the philosophy of the revolutionaries. But there was a mass of liberal concept that had to be translated into action. In 1789 the newly formed Assembly of the States General appointed a committee to remedy the poverty of the masses and their ill health. Curiously this committee was headed by a blueblooded aristocrat, Duc de la Rochefoucauld-Liancourt, and had two doctors as members. One of the doctors headed a sub-committee dealing with health. The committee worked very hard, holding 70 meetings in 18 months, visiting countless hospitals and corresponding with experts abroad.

It was held that public assistance was an obligation of

society and thus a responsibility of the state. So charities were abolished and their funds sequestrated. At the same time religious orders were abolished. This left the health committee with no funds for the hospitals and, because the nuns had to go, no nurses. In the interests of equality all 'privileged associations' were abolished. These included scientific societies and medical schools. So no new doctors were trained.

The philosophy of health care led in practice to the closure of hospitals and the loss of nurses and doctors. This was not exactly a triumph for the doctor who headed the health committee. His name is not remembered for creating an administrative shambles but for his practical invention. The doctor was Joseph Ignace Guillotin.