Terje Skjærpe

NONINVASIVE QUANTITATION OF GLOBAL PARAMETERS ON LEFT VENTRICULAR FUNCTION:

THE SYSTOLIC PULMONARY ARTERY PRESSURE AND CARDIAC OUTPUT

University of Trondheim
Faculty of Medicine
Trondheim – Norway

TAPIR

ERRATA

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When the right ventricular and pulmonary artery pressure are normal, and in absence of no pulmonary stenosis, a high ....
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Side 25, the three last lines should be replaced with:
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Calculation of streamlines and velocities in a simplified (straight, axis symmetric) model of the aortic root. The calculations are based on the Navier-Stokes equations for viscous, incompressible fluid, using the finite element method (S. Ø. Wille, H. P. Langtangen, Institute of Informatics, University of Oslo. Reproduced by permission). The dimensions of the aorta and the velocities at the annulus are from a patient included in group 1, paper VI. The ejection time was 210 ms., peak velocity 1.1 m/s. In a curved vessel the large eddy will be located at the inner curvature, and give rise to velocity recordings like those in figure 4, paper VI.

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UNIVERSITETET I TRONDHEIM
DET MEDISINSKE FAKULTET

NONINVASIVE QUANTITATION OF PARAMETERS ON GLOBAL LEFT VENTRICULAR PERFORMANCE: THE SYSTOLIC PULMONARY ARTERY PRESSURE AND CARDIAC OUTPUT

TERJE SKJÆRPE

Overnevnte avhandling er funnet verdig til å forsvare offentlig for den medisinske doktorgrad. Disputasen finner sted i Store Auditorium, Regionsykehuset i Trondheim, 28. mai 1988.

SAMMENDRAG

Doktorarbeidet har gått ut på å utvikle metoder for å måle viktige parameter på hjartefunksjonen ved hjelp av ultralyd.

Dei parametra vi har sett på er trykket i lungekrinslaupet og mengden blod hjarta pumper pr. minutt, det såkalla minuttvolumet. Dette er viktige mål på den totale hjartefunksjonen og er som regel av stor interesse både ved skader av hjartemuskelen (hjarteinfarkt), ved ulike typer klaffefeil (forsnevringer og lekkasjer i hjarteklaffene) og ved medfødde hjartesjukdomer. Vanligvis er det naudsynt med ein såkalla hjartekateterisering for å måle desse parametra.

Resultata viser at trykket i lungekrinslaupet kan målast med stor nøyaktighet med ultralyd hos dei fleste pasientene. Minuttvolumet kan også målast med omlag same nøyaktighet under hjartekateterisering. Ein videreutvikling av metoden for minuttvolum målinger gjer det mulig å måle grad av forsnevring av klaffen mellom venstre hjartekammer og hovedpulsåra.

Betydningen av resultata er at ein kan unngå ei ressurskrevande hjartekateterisering hos mange pasienter. Ultralydundersøkinga er langt mindre ressurskrevande og gir mindre plager og risiko for pasienten. Den kan gjerast om igjen så ofte det er naudsynt, og sjukdomsutviklinga hos pasienter med ulike typer hjartefeil kan følgast langt betre. Operasjonstidspunktet kan difor i større grad optimaliserast.

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Terje Skjærpe

Noninvasive Quantitation of Global Parameters on Left Ventricular Funtion: The Systolic Pulmonary Artery Pressure and Cardiac Output.

Publication from the University of Trondheim
Department of Medicine
Section of Cardiology
Trondheim University Hospital
N-7006 Trondheim, Norway

Head of Department: *Professor Rolf Rokseth*

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NONINVASIVE QUANTITATION OF GLOBAL PARAMETERS ON LEFT VENTRICULAR FUNCTION:

THE SYSTOLIC PULMONARY ARTERY PRESSURE AND CARDIAC OUTPUT

TAPIR
1987

CONTENTS - GENERAL PART

ACKNOWLEDGEMENTS.

I would first of all like to express my gratitude to dr. Liv Hatle who taught me Doppler. Her deep insight in clinical cardiology and hemodynamics has been the basis of many ideas and stimulating discussions on how to use Doppler ultrasound to obtain clinical important information.

I also want to specially thank the chief of the Section of Cardiology at the Regional Hospital of Trondheim, Professor Rolf Rokseth. Without his continuous support and encouragement, the present work would have been difficult to finish.

The cooperation with dr. Lars Hegrenæs constitutes an important part of the work presented.

I am indebted to dr. Jørn Bathen for his constant willingness to allow time enough for discussions on practical problems. Despite his own thigt schedule he has accepted extra burdens to increase the possibility to bring this work through. I also want to thank dr. Jan Eriksen and dr. Jon Dale for help and advice in preparing manuscripts.

Thanks are due to the audio-visual department, chief T. Stuedal, for the preparation of the illustrations, and to B. Tonning for photographic assistance.

I want to thank The Norwegian Research Council for Science and the Humanities for the Fellowship I received and for further financial support. I acknowledge the support given by the Norwegian Council on Cardiovascular Disease.

The technical support given by Vingmed has been of immense value.

LIST OF PAPERS.

The papers will later be referred to by their Roman numbers.

I. Skjaerpe T, Hatle L. Diagnosis and assessment of tricuspid regurgitation with Doppler ultrasound. In: Rijsterborgh H, ed. Echocardiology The Hague: Martinus Nijhoff Publishers, 1981: 299-304.

II. Skjaerpe T, Hatle L. Noninvasive estimation of pulmonary artery pressure by Doppler ultrasound in tricuspid regurgitation. In: Spencer MP, ed. Cardiac Doppler diagnosis. Boston: Martinus Nijhoff Publishers. 1983: 247-54.

III. Skjaerpe T, Hatle L. Noninvasive estimation of systolic pressure in the right ventricle in patients with tricuspid regurgitation. Eur Heart J 1986; 7: 704-710.

IV. Skjaerpe T, Hegrenaes H, Ihlen H. Cardiac output. In: Hatle L, Angelsen B. Doppler ultrasound in Cardiology. Philadelphia: Lea & Febiger. 1985.

V. Skjaerpe T. Influence of the geometry of the ascending aorta upon the velocity profile. In: Spencer MP, ed. Cardiac Doppler diagnosis, Volume II. Dordrecht: Martinus Nijhoff Publishers. 1986: 65-71.

VI. Skjærpe T, Hegrenæs H, Bathen J. Evaluation of the optimal level for noninvasive measurements of cardiac output in the left ventricular outflow tract and the ascending aorta. Comparison with two invasive methods. (Submitted for publication).

VII. Skjaerpe T, Hegrenaes L, Hatle L. Noninvasive estimation of valve area in patients with aortic stenosis by Doppler ultrasound and two-dimensional echocardiography. Circulation 1985; 72: 810-818.

INTRODUCTION.

Blood velocities can be measured with ultrasound by transmitting it, continuously or in pulses, toward the blood stream and analyzing the frequency shift (Doppler shift) of the back scattered signal. Assuming that the velocity of ultrasound in the tissues of the body is constant, and keeping the frequency of the transmitted ultrasound constant, the Doppler shift depends only on the velocity of the blood and the angle between the ultrasound beam and the direction of flow (the angle of incidence). Accordingly, the key to successful measurements of blood velocities is to reduce the angle of incidence to less than +/-20°. This angle introduces an error of 6% on calculated velocities, which is generally accepted to be clinically insignificant. The studies of Holen (1) and Hatle (2) showed how pressure drops can be calculated from the velocities by a very simple formula, the so called simplified Bernoulli equation: Pressure drop = $4*Vmax^2$. It is assumed that the velocities proximal to the jet is low enough to be neglected in comparison with the velocities in the jet, and that the size of the orifice is above a certain limit, about 0.1 cm^2, to allow the viscosity of blood to be neglected. Provided adequate examination techniques and Doppler instruments of high sensitivity, these findings made it possible to quantitate pressure drops across stenotic valves (1-8).

Stenotic valve areas compatible with life usually are larger than 0.1 cm^2, the limit below which the viscosity may invalidate the use of the simplified Bernoulli equation. In regurgitant valves the flow area may be so small that the viscosity can no longer be neglected. To calculate pressure drops across leaking valves, it is therefore necessary to evaluate the validity of the simplified Bernoulli equation in small regurgitations, in addition to the problem of obtaining a

small angle of incidence. Because of the large value of having information on the pulmonary artery pressure in a variety of cardiac patients, it was considered very important in the present studies to evaluate these problems.

To extract information on volume flow from the recorded velocities, additional problems are encountered. Because the sample volume usually are much smaller than the vessel lumen, the main problem is to what extent the spatial distribution of the velocities are represented by the recorded velocities. Another is the accuracy with which the flow area can be measured. Because of the lack of reference methods to evaluate these problems separately, the combined effect must be evaluated against invasive methods commonly accepted to provide useful estimates on cardiac output.

Even when only the ascending aorta is considered, a multitude of different ways to do the noninvasive measurements seem to give good results compared to invasive methods (9-18), but the results of some studies are incompatible with others, unveiling the existing controversies. Reviewing the literature, there seems to be a lack of studies that try to evaluate factors that may influence the accuracy of cardiac output estimates. Several animal studies exist (19-24). They are probably of limited value in relation to man, especially since conflicting results are obtained in different species. A study on factors that might influence the velocity distribution in the ascending aorta in man is therefore of interest.

The current studies were designed to look at some of the aspects mentioned above. The first paper presented was published in 1981, and the last in 1986. During these 5 years the equipment used for velocity measurements has been developed into very sophisticated instruments. The most important change occurred with the implementation of high speed spectral analysis, dramatically increasing the ability to display high velocities in jet flow. Simultaneously, the international interest in Doppler as a diagnostic and quantitative tool in heart

disease, has changed from curiosity to a general acceptance of the importance of the method.

The papers presented here to some extent reflect these changes. The Doppler recordings in the first studies were mainly made with an instrument displaying the velocities by analog frequency estimation, while in the later ones spectral analysis was generally applied. Comparing the first article to some of the later ones, the development of Doppler from a promising investigative tool into an accurate quantitative method, is indicated. However, despite the limitations imposed by analog frequency estimation, the first two articles represent the first reports on one of the most clinically helpful applications of Doppler, the quantitation of systolic pulmonary artery pressure by the velocities in tricuspid regurgitation.

The reason why we chose to look at the quantitation of the pulmonary artery pressure and the cardiac output is that both parameters to some extent are measures of the global performance of the left ventricle, providing important clinical information in patients with valvular or myocardial diseases. Also in shunts and pulmonary diseases the same parameters are of great importance.

THE AIMS OF THE STUDY.

The aim of the study was to develop noninvasive methods to quantitate important parameters on global cardiac function; the estimation of systolic pressures in the right ventricle and pulmonary artery, and cardiac output measurements in the outflow part of the left ventricle.

More specifically the aims were:

1. To evaluate if it was possible to obtain semiquantitative information of the severity of tricuspid regurgitation with Doppler.

2. To see to what extent the velocities in tricuspid regurgitation could be used to measure the pressure drop across the valve, and if the systolic right ventricular pressure (pulmonary artery pressure in absence of pulmonary stenosis) could be estimated by adding the central venous pressure judged by the neck vein congestion.

3. To evaluate how different degrees of tricuspid regurgitation, as judged by Doppler, influenced the accuracy of systolic pulmonary artery pressure estimations by two noninvasive methods: The right ventricular isovolumic relaxation time and the velocities in the tricuspid regurgitation.

4. To evaluate the theoretically optimal level(s) for cardiac output measurements in the left ventricular outflow tract (LVOT) and the ascending aorta by combining knowledge on fluid dynamics and the caliber variations of the distal LVOT and the first part of the ascending aorta. Or, in other words, to find out to what extent the anatomy in these regions provide the anatomic basis for a flat velocity profile.

5. To test the theories in a clinical study

6. To test and exemplify the usefulness of the noninvasive method for cardiac output measurements in a study on a completely noninvasive method for estimating valve area in aortic stenosis, designed to be independent of any coexisting regurgitation.

METHODS.

Patients.

There were some overlap of patients in some of the studies. In paper I, 85, and in paper II, 92 patients were included. Thirty-one patients in the first study in whom measurements of the maximal velocities in the tricuspid regurgitation were attempted, were also included in the second study. Twenty-one patients in the second study where the recorded velocities were displayed by spectral analysis, were also included in the study presented in paper III. Group 1 patients in paper VI were all included in the study on caliber variations of the ascending aorta (V).

In all patients where comparisons were made to invasive measurements, catheterization was necessary for clinical reasons.

Instruments.

1) Pedof (Vingmed A/S). (Papers I,II,VI) A separate Doppler unit displaying the velocities by analog frequency estimation. The maximal velocity is nondirectional (velocity curve always positive), while the mean velocity is directive (velocities toward the transducer give a positive deflection of the velocity curve, and velocities away from the transducer give a negative deflection). The loudness of the Doppler signal is displayed by an intensity curve where short, strong signals (valve movements) are displayed as distinct spikes. Automatic calculation and analog display of velocity integrals (maximal or mean velocity) are provided. Individual adjustment of gain is possible for each curve. The curves can be recorded on a regular ECG recorder. The sample volume in pulsed mode is about 7 mm i diameter, and 7 mm long.

2) A prototype cardiac output unit (Vingmed A/S). (Paper VI) This instrument is basically a Pedof with additional features. Vessel diameter can be keyed in for instantaneous calculation of cardiac output, averaged over the 10 latest beats. The results, together with patient i.d., instrument set-up and the curves as displayed on the screen can be printed out on a dot matrix printer.

3) Irex III B (Irex Corporation). (Papers II,III,V-VII) A two-dimensional echocardiographic machine with a 3.5 mHz phased array sector transducer. A PCD-4 (basically a Pedof) is implemented to enable 2 mHz pulsed and continuous wave Doppler recordings to be made simultaneously with imaging. A separate Doppler transducer is provided for optimal velocity recordings. High speed spectral analysis (Chirp-Z) of the Doppler signal is made and displayed. Optionally, analog curves as described for Pedof, can be displayed.

4) ATL Mark III (Advanced Technology Laboratories). (Papers V-VII) A two-dimensional echocardiographic instrument with a 2.5 mHz mechanical sector transducer.

Ultrasound measurements.

For methodological problems relating to specific measurement sites, I refer to the various papers. Some general comments on the Doppler technique are appropriate.

The problem of using a separate Doppler instrument, is how to localize the recorded velocities. Concerning valvular flow, the separation of flow through the aortic and pulmonary valves from flow through the atrioventricular valves is, of course, easy since one is systolic and the other diastolic. Tricuspid was separated from mitral flow by the distinct respiratory variations and the lower velocities of the tricuspid flow. In significant tricuspid regurgitation both characteristics may be less apparent. In such cases flow through both

valves was recorded from the apex. From this position the tricuspid flow is recorded medial to the mitral flow.

Pulmonary flow was separated from aortic flow by its lower acceleration, lower peak velocities and timing (P2 occurring after A2). Especially in case of pulmonary hypertension, but also when there was increased flow because of pulmonary regurgitation or a left to right shunt, other characteristics were again used. The pulmonary flow was usually best recorded from the third intercostal space, directing the transducer posteriorly, cranially and somewhat to the left. By directing the transducer medially, usually also increasing the depth of the sample volume (pulsed mode), aortic flow was also localized. By directing the transducer back and forth, differences in shape and timing of the flow curves were noted to ensure that separate flows were recorded. A two-dimensional echocardiographic study was always made to confirm a normal anatomic relationship between the great vessels.

The depth of the sample volume relative to the the valve leaflets was determined as described by Hatle by paying attention to the opening and closure signals (25).

Especially when using continuous wave, care was taken to relate the velocities to the valve movements. Only when the recorded velocities were observed to start and stop at valve opening and closure, or vise versa for regurgitant flow, they were accepted as representing valvular flow.

Maximal jet velocities were always obtained by recording from different directions (I,II,III,VII). For better intercostal access, and optimal sensitivity, a separate Doppler transducer was used whenever considered necessary. No attempts were made to correct for the angle between the ultrasound beam and the assumed direction of flow.

Except when doing cardiac output·measurements, all measurements were made off-line on paper print outs. To increase measurement accuracy, the paper was run at a speed of 100 mm/s when measuring time intervals, and velocity curves were usually scaled to be as large as

possible.

In the two first papers, mainly analog estimation of velocities were used. That means that the instrument at any moment must decide whether a frequency component represents noise or velocity. Since this decision is based on differences in intensity, correct gain adjustment becomes critical. Too little gain may make the estimators track too low velocities, while too high gain may cause the estimators to track noise components which may contain high frequencies, causing overestimation of velocities.

By spectral analysis a complex sound signal is analyzed during a short time interval, and the frequency components are displayed irrespective of wether they represent noise or velocity signals. It is left to the eye to separate velocities from noise. Since the analyzed time intervals are displayed consecutively, the individual velocity components will form a curve which the eye can easily separate from noise.

SUMMARY OF RESULTS.

Abbreviations used:
TR: Tricuspid regurgitation. RA: Right atrium. RV: Right ventricle. SPAP: Systolic pulmonary artery pressure. CW: Continuous wave Doppler. Pc-To interval: Interval from pulmonary valve closure to tricuspid valve opening. LVOT: Left ventricular outflow tract.

Paper I

TR was diagnosed with Doppler in 85 patients when reverse flow in systole, originating at the tricuspid orifice, could be followed back into the RA. Attempts on assessing the severity were based on the velocity of forward flow, the intensity of the regurgitant jet signal and the extension of the jet into the atrium. The regurgitations were classified as mild, moderate and severe.

In 31 patients where RV angiography or contrast echocardiography were performed, the diagnosis was confirmed in all. Of 62 catheterized patients 27 had abnormal V-waves, 1 of 22 with mild TR, 17 of 29 with moderate TR and 9 of 11 with severe TR. A clinical diagnosis was made in 32 of 85 patients, in 1 of 35 with mild TR, 15 of 39 with moderate TR, and in 7 of 11 with severe TR. In 29 patients the maximal velocities in the TR were measured, and the RV-RA systolic pressure drop was calculated as $4*Vmax^2$. The correlation to invasively recorded pressure drops was r=0.93.

Comparing the grading of the TR to the hemodynamic and clinical data indicates that semiquantitation of TR is possible with Doppler. The RV-RA systolic pressure drop can be calculated from the maximal velocities in the TR as recorded with Doppler and may provide a noninvasive way to estimate the RV systolic pressure.

Paper II

In 92 catheterized patients, TR was diagnosed and assessed as in paper I, using analog frequency estimation in 71, and spectral analysis in 21 patients. A recording of the maximal velocities in the TR were attempted in 76 patients, and the RV-RA pressure drop calculated ($4*Vmax^2$). The RA pressure was estimated by the neck vein congestion to obtain an estimate of the systolic RV pressure. The Pc-To interval was also attempted measured with Doppler and phonocardiography, and the pulmonary artery pressure was read from the table constructed by Burstin (26).

In 67 of 76 patients where CW Doppler was used, the maximal velocities in the TR could be recorded. Calculated RV-RA pressure drop correlated well with invasive measurements (r=0.93). In the patients where spectral analysis was used, the r-value was 0.97. Calculated and invasively measured SPAP also correlated well (r=0.90). The Pc-To interval was recorded in 75 of 83 patients. SPAP were underestimated in some patients with moderate and severe TR (up to 40 mmHg), while good estimates were obtained in mild TR. Combining the results, using the highest value where both methods were used, gave good estimates of SPAP in 79 of 92 patients with TR (r=0.89).

Paper III

Since earlier experience was that the velocities in TR were better displayed by spectral analysis than by analog frequency estimation, we measured the maximal velocities and displayed them by spectral analysis in 70 patients diagnosed as having TR by Doppler. The TR was graded (paper I) and the RA pressure was estimated as in Paper II. Estimated and recorded RA pressures correlated moderately well (r=0.79) while the correlation between calculated and invasively measured RV-RA pressure drops was good (r=0.97). Calculated and measured RV pressures also correlated well (r=0.96). A similarly good

correlation (r=0.95) was obtained in patients judged by Doppler as having mild TR, indicating that the viscosity of the blood does not invalidate the use of the simplified Bernoulli equation when the regurgitant area is small.

Paper IV

Because different ways of doing cardiac output measurements in the aorta and the LVOT have been used, and because we were not able to reproduce some results, an extensive literature study was undertaken. The problems especially considered were theoretically expected velocity patterns and results of clinical and experimental studies on the velocity profile and cardiac output measurements. Based on these considerations, and our own initial experience, we describe a measurement method and technique that is designed to minimize some of the problems encountered.

Paper V

To analyze factors that might influence the velocity profile in the ascending aorta, fifty-five patients, 3 to 74 years old, underwent echocardiographic measurements of diameters at four levels in the LVOT and the ascending aorta: 1) The aortic annulus, 2) the Sinus of Valsalva, 3) the level just distal to the sinus of Valsalva (sinotubular junction), and 4) a level about 2 cm distal to the Sinus of Valsalva. The results show that in most patients there is a sudden expansion of the cross sectional flow area distal to the annulus. The aortic geometry thus does not fit the model of a narrow flow channel branching of from a larger one (the left ventricle), a model in which a flat velocity profile is found in the inlet region. On the contrary, the aortic annulus represents an obstruction between larger flow channels, the proximal LVOT and the ascending aorta.

Attempts were made to quantitate the degree of skewing of the velocity profile that occurs because of the curved course of the

ascending aorta. Such measurements turned out to be very difficult, and were successful only in 6 patients. The results suggests, however, that only moderate overestimations of cardiac output measurements in the ascending aorta may be caused by the skewness of the profile.

Paper VI

In 40 patients (group 1) diameters were measured at the 1) aortic annulus, 2) sinotubular junction and 3) about 2 cm distal to the sinotubular junction. Maximal and mean velocity recordings were made at level 2 simultaneously with invasive measurements (Fick in 26 and thermodilution in 30 patients, both in 16 patients). Cardiac outputs were calculated from the velocity integrals and any of the diameters. Only when the mean velocity integral was combined with the diameter at the annulus were significant overestimations avoided. In 18 patients (group 2) velocity measurements were also made from the apex at the annulus. The results were somewhat different from group 1 in that maximal velocity integrals at level 2 tended to be lower, giving better correlation to invasive measurements when combined with the diameter at the annulus. Doing both velocity and diameter measurements at the annulus provided the best results, being not much more different from the invasive results than the difference between the two invasive methods in 16 group 1 patients.

Paper VII

Based on the findings that the best level for cardiac output measurements is just below the aortic valve, a method to estimate the valve area in aortic stenosis was designed. Since volume flow must be equal at the annulus and at the stenotic valve, the product of the subvalvular velocity integral and flow area, divided by the jet velocity integral, yields the effective valve area. A simpler method was also developed, where the product of the peak velocity and flow

area at the subvalvular and at the valvular area were assumed to be equal (equation of continuity). The results were compared to invasive estimates of valve area (Gorlins formula) in 30 patients. Good correlations were obtained. The methods were also found to be independent of aortic regurgitation.

GENERAL DISCUSSION

Right heart pressure estimation.

When the right ventricular and pulmonary artery pressure are normal, and in absence of no pulmonary stenosis, a high pressure difference exists between the pulmonic and the systemic circulation in systole, while a relatively low pressure difference developes between the right ventricle (RV) and the right atrium (RA). With increasing SPAP the situation is changed. If an opening exists between two such chambers with different pressures (aortico-pulmonary shunts, VSD, tricuspid regurgitation), the velocity of the blood flow trough it will depend on the pressure difference itself, the viscosity of the blood relative to the size of the opening, and the inertia of the blood (27).

In valve stenosis the valve opening is usually so large that the viscosity has little influence upon the velocity (1-8). The inertia of the blood may cause a delay of the pressure drop curve when it is calculated from the flow velocities, but the effect on the peak velocity during systole is small (27). The relationship between velocity and pressure drop can therefore be described by a simplified formula : $4*Vmax^2$, where Vmax is the maximal velocity in the jet (2).

In shunts and tricuspid regurgitations the situation may be different. There is no lower limit to the size of the opening, and it is to be expected that the viscosity can not always be neglected.

Contrary to valve stenosis, it is the intra-cavital pressure, not the pressure difference, that is of prime interest. To calculate the pressure in one chamber, one has to know the pressure in the other chamber.

Velocities in the tricuspid regurgitant flow.

In 1981, we found it was possible to quantitate the pressure drop across the tricuspid valve in systole with Doppler in some patients with tricuspid regurgitation (I). We also suggested that it might be possible to estimate the RVP by adding the right atrial pressure judged by the level of the neck vein filling.

The problems concerning this method are mainly three: 1) To what extent can the peak velocity in the tricuspid regurgitant jet be recorded and displayed. That is, can the angle between the ultrasound beam and the direction of the flow be made negligible, and is the instrument sensitive enough to record and display the velocities throughout systole in all patients. 2) To what extent can it be assumed that the viscosity does not influence the velocities. 3) How accurately can the right atrial pressure be estimated by the filling of the neck veins?

The first two problems are difficult to judge separately, except that we found that spectral analysis was superior to analog frequency estimation in displaying the velocities (II). The combined effect, however, can be evaluated by calculating the systolic pressure drop across the valve from the velocities in the regurgitant flow, using the simplified Bernoulli equation, and compare to the invasively recorded pressure drop. When the measurements are done simultaneously, the results are very close (I,II,28,29), indicating that angle problems are to a large extent avoided. In patients judged as having a small regurgitation, the correlation to invasive measurements was still good (III), indicating that the viscosity of the blood seems to influence the velocity to a very limited degree.

It is obvious that the estimation of the right atrial pressure by the neck vein filling is the least accurate part of the procedure (II,III,28-31). A fixed figure to add to the calculated pressure drop has therefore been suggested by some (30,31). In practice, however, the

inaccuracy of the right atrial pressure estimation is not a big problem since it generally is much lower than the transtricuspid pressure drop. The absolute error on the estimation of the RVP is therefore relatively small.

The clinical usefulness of the method depends on the incidence of tricuspid regurgitation in patients, and the sensitivity of Doppler in recording the regurgitant flow. Several studies indicate that Doppler is a very sensitive method (32-37), and is very easy to perform (32,37), and also that tricuspid regurgitation is much more frequent than previously thought (38, Hatle L. Unpublished results). The method is thus applicable in a high number of patients.

In summary, the recording of the velocities in the tricuspid regurgitation makes it possible to estimate the RVP with a high accuracy, probably more accurately than by any other noninvasive method.

The pressure estimated by the velocities through a leaking tricuspid valve, is the RVP. In case of pulmonary stenosis, the pressure drop across the pulmonary valve must be subtracted to give the SPAP. It is possible to quantitate this pressure drop with good accuracy (7,8), and the SPAP could be determined in the presence of pulmonary stenosis in our series (III).

Isovolumic relaxation time.

In patients without tricuspid regurgitation, other methods must be used to estimate the SPAP. In 1960 to 1965 the first reports on the usefulness of the isovolumic relaxation period of the right ventricle (Pc-To interval), were presented (39-41). In 1967 Burstin constructed a table where the isovolumic relaxation interval was corrected for heart rate, from which the systolic pulmonary artery pressure could be read (26). Hatle et al. (42) showed that the table was accurate for estimating SPAP also when the isovolumic period was recorded with

Doppler and phonocardiography. The table does not correct for heart rates higher than 125 beats per minute. Based on recordings with M-mode echocardiography, Stevenson et al. (43) modified the table to be used in patients with heart rates from 80 to 170 beats per minute.

The method is based on a low right atrial pressure. It has therefore been stressed that the method is not useful in right ventricular failure and tricuspid regurgitation (26,42-43). Chan et al. (44) found that tricuspid regurgitation did not influence the results, but our own experience (II) indicate that in regurgitations judged as more than mild by Doppler, significant underestimations may occur. In the late phase of isovolumic relaxation, the rate of decrease in pressure diminishes (45). When the right atrial pressure is elevated, the tricuspid valve may therefore open earlier than would be expected if the pressure decrease was constant until the end of relaxation. That is probably the explanation why we were not able to correct the underestimation of SPAP by adding the height of the atrial V-wave to the estimated SPAP (unpublished results).

Gamboa did not find the isovolumic relaxation time useful in ASD (41). Details are not presented, but the reason may be an elevated right atrial pressure, or V-wave, which some of these patients may have. Freund and Wranne (46) questioned the inverse relationship to heart rate in neonates, but no direct correlation to pressure recordings was made.

In applying the same correction table to all patients, similar rates of right ventricular relaxation are assumed. Since the Pc-To interval does not measure the interval from peak SPAP to pulmonary valve closure, a fixed relationship between the pressure decrease before and after pulmonary valve closure is also assumed. A third assumption is that the pulmonary valve closes exactly when the pressure in the right ventricle falls below that in the pulmonary artery. Neither of these assumptions may be completely true. The method therefore needs extensive empirical support. Such support is now

accumulating (41-44,II), indicating that the accuracy is high enough to make the method clinical very useful. According to our own results, however, tricuspid regurgitations may interfer with SPAP estimations with the relaxation time method.

Various ways to quantitate the regurgitation with Doppler has been proposed (32,35,36,47,48,I,II,III), but the problem probably needs further evaluation (36,49). However, our method (I,II,III) allowed us to sort out those regurgitations which are insignificant in relation to the applicability of the Pc-To interval for SPAP estimations.

Cardiac output and valve area estimation.

Cardiac output.

For a detailed discussion on flow and volume flow estimations in the LVOT and the ascending aorta, I refer to paper IV.

The conclusion, that the aortic annulus is the optimal level for measurement of flow area and that the velocities should be made at the same level or just distally, in the proximal aortic root, is supported by the results of Ihlen et al. (9). When they measured the velocities in the proximal ascending aorta and the flow area at the aortic orifice, good results were obtained. When the diameter at the sino-tubular junction was used, significant overestimations resulted. Rein et al. (18) arrived at similar results. This is as expected when the anatomy of the LVOT and the ascending aorta is considered (V). Mathematical modelling of arterial flow, based on the Navier Stoke flow equations, show that the flow pattern distal to obstructions is very irregular (50), and the maximal velocities are clearly higher than the spatial mean. Applying the same mathematical method on diameter measurements made in one patient included in our study on cardiac output estimations (VI), also indicates that volume flow will be significantly overestimated when the diameter and the maximal

velocities are measured at the sinotubular junction (figure 1). Since the sample volume, when positioned centrally in the aorta, only covers the region where the velocity profile is quite regular, even the mean velocity is expected to be higher than the cross sectional mean. This is in accordance with our findings (VI).

Our own results (VI) are similar to those of Ihlen et al. (9), but in a few patients we made unacceptable overestimations when the maximal velocities at the sinotubular junction were combined with the diameter at the annulus. This indicates that factors in addition to those discussed in papers IV and V must be considered when doing velocity recordings from the suprasternal notch.

A common observation is that there is a moderate velocity increase from the distal LVOT and up to the tips of the aortic cusps, indicating a decrease in the cross sectional flow area. There does not seem to be studies on the non-stenotic valve area in relation to the area at the annulus, but in an unpublished study on cusp separation, we found that the maximal separation was from 6 mm smaller to 4 mm larger than the diameter at the annulus. It may be difficult to make any conclusions on corresponding differences in flow area, but since the valve orifice have a shape between a circle and a triangle (the cross sectional area being smaller than that of a circle with the same diameter) (51) the finding suggests that in some patients the valve is restrictive compared to the annulus. The maximal velocity integral, as recorded in the aortic root, will in such patients overestimate cardiac output when combined with the diameter at the annulus. The reason why significant overestimations were only seen in some patients, and that in group 2 patients (VI) mainly underestimations were made, is probably that the angle between the proximal aortic root and the ultrasound beam when the Doppler measurements are made from the suprasternal notch, is large axis symmetric) model of the aortic root. The calculations are based on the Navier-Stokes equations for viscous, incompressible fluid, using the enough to cause underestimation of velocities in many

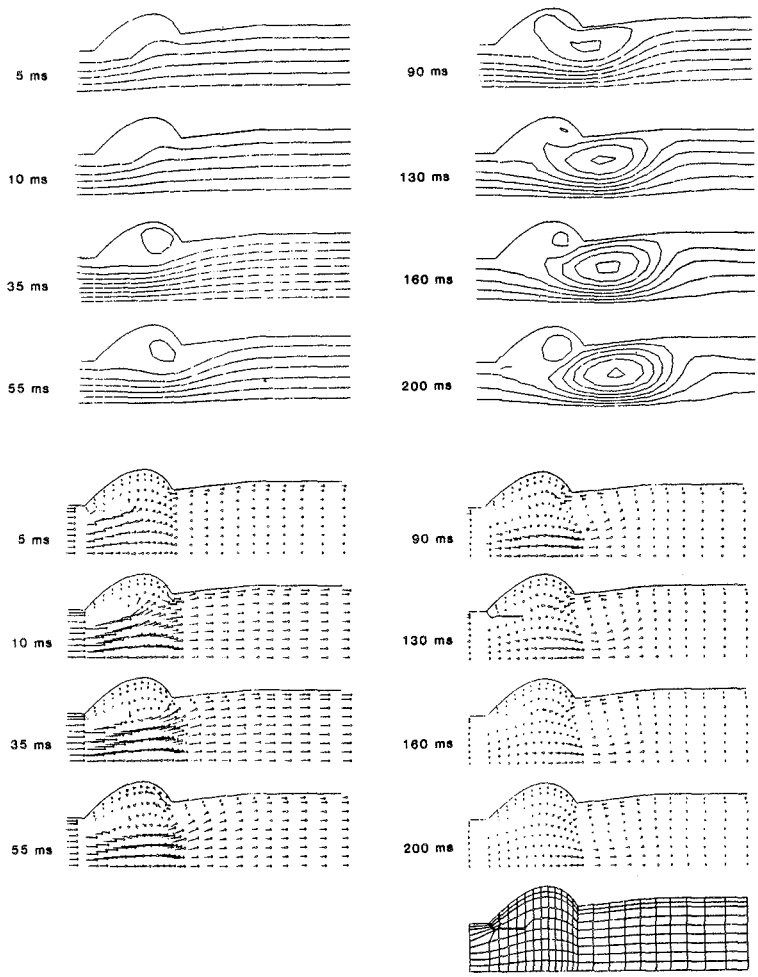

Figure 1.

Calculation of streamlines and velocities in a simplified (straight, finite element method (S. Ø. Wille, H. P. Langtangen, Institute of Informatics, University of Oslo. Reproduced by permission.) The dimensions of the aorta, and the velocities at the annulus is from a patient included in group 1, paper VI. The ejection time was 210 ms., peak velocity 1.1 m/s. In a curved vessel the large eddy will be located at the inner curvature, and give rise to velocity recordings like those in figure 4, paper VI.

patients. This was proved in two group 2 patients (VI) where higher velocities were recorded from the right parasternal border. An angle of 30° between the plane of the aortic cusps and the long axis of the patient is common (52). If this angle also represents the flow direction, the underestimation of velocities is about 16% In fact, this figure is quite representative for the mean per cent underestimation we made in group 2 patients in paper VI. In horizontal hearts the angle probably is even larger. Various combinations of restrictive and nonrestrictive valves, and vertical and horizontal aortas may therefore result in both over- and underestimations.

One important advantage with the apical approach is that the position of the transducer is easier to adjust to reduce the angle of incidence. In practice it was also much easier to do velocity recordings at the annulus from the apex, than at the sinotubular junction from the suprasternal notch. This is in accordance with flow theory as discussed in papers IV and VI. This approach was also the one that gave the best results compared to the invasive measurements (VI), but since the method was used in only group 2 patients in this study, the conclusive power is limited. However, the results of other studies using the same approach are similar to ours, and no clearly conflicting studies are published (14,17). This method therefore emerges as the least controversial one.

The accuracy with which volume flow estimates are made is important. Errors on diameter measurements of up to 2 mm probably are to be expected in some adult patients. Depending on the size of the LVOT, errors of 15-20% on cardiac output will result. The smallest detectable change in velocities seem to be about 10% (53). The combined error can then be up to 25-30%. Usually the error probably is slightly less. If a maximal velocity estimator is used, or if the envelope of a spectral curve is traced, the flow rate is overestimated if the velocity profile is not completely flat. On the other hand, one of the most important errors in measuring the diameter, is probably that the imaging plane does not cross the center of the vessel. The diameter and

consequently the area, is then underestimated, partly compensating the overestimation made by Doppler. Our results suggest that an error of up to about 20% was made by Doppler and echo, being fairly close to the theoretical expected error. The consequence is that when other measurements are added to calculate volume fractions (54-56) or valve areas (VII,57-64), the total sum of potential errors must be considered.

Estimation of valve area in aortic stenosis.

The formula developed by Gorlin and Gorlin (65) is the only available method for the calculation of valve areas. However, because the discharge coefficient varies with the size of the valve relative to the proximal flow area, errors of 20 to 40% may occur (66). When the errors on flow rate estimates are added, it is clear that the formula does not represent the ideal method. Another limitation is that valvular flow rate can not be measured by conventional methods when there is both aortic and mitral regurgitation. This is also the limitation of other semi- or noninvasive methods (58-60,63).

When measurements of flow are made in the distal LVOT, total forward stroke output (net cardiac output plus regurgitant volume in case of aortic regurgitation) is measured, being independent of regurgitations in other valves (VII). The noninvasive methods as described in paper VII, is thus the only method, invasive or noninvasive, by which aortic valve area can be estimated in patients with multiple valve regurgitations. There are, however, limitations to the method to be aware of. In addition to those discussed in paper VII, caution should be exerted in patients with severe dilatation of the left ventricle. In one such patient examined after the discontinuation of the presented series, we significantly overestimated aortic valvular flow rate. The problem with very dilated ventricles is probably that the left ventricular outflow tract is "eliminated", making it very difficult to determine which velocities match the subvalvular diameter.

Also with this method the accuracy presents a limitation. According

to the discussion in paper VII, the summed errors may in some patients be up to about 30-40%. There are, however, indications that some errors partly compensates each other, reducing the total error. Since suboptimal recordings of the maximal velocities in the jet result in too low velocities being recorded, there should be a tendency to overestimate valve area. But, on the contrary, a tendency to underestimate valve area was observed, especially when using the "inner" diameter (trailing to leading edge measurements). Thus, the tendency to overestimate valve area by underestimating the jet velocities, was partially compensated for. This is probably a result of how we selected the level for subvalvular velocity measurements (VII). Even if the errors should not be disregarded, the method holds the potential of being comparable to or better than the Gorlin's formula also in this respect.

There are now four more reports in the literature where the valve area in aortic stenosis is estimated by the same methods we used (57,61,62,64). The results are in accordance with ours.

The final evaluation of the presented method is hampered by the inaccuracy of Gorlin's formula. Nevertheless, the method is promising enough to encourage further research on its clinical value.

CONCLUSIONS.

The papers presented indicate that semiquantitation of tricuspid regurgitation with Doppler is possible. The isovolumic relaxation time of the right ventricle can therefore more safely be used to estimate the systolic pressures in the pulmonary artery, in that patients with significant regurgitation, in whom it may not be applicable, can be sorted out.

In patients with any degree of tricuspid regurgitation the systolic pressure in the right ventricle can be assessed with high accuracy, the

main limitation being the clinical estimation of the right atrial pressure. In case of pulmonary stenosis, the pulmonary artery pressure can be estimated by subtracting the pressure drop across the pulmonary valve.

It is thus possible to estimate the systolic pulmonary artery pressure in almost any patient provided there is ultrasonic access.

Theoretical considerations and the study on cardiac output indicate that the aortic annulus is the optimal level for both diameter and velocity measurements. If care is taken to align the ultrasound beam with the direction of flow, cardiac output can be quantitated with reasonably good accuracy.

The studies represent another step toward the complete noninvasive evaluation of the cardiac patient. Two important complications to a variety of cardiac diseases, pulmonary hypertension and changes of cardiac output, can be estimated with an accuracy that makes the methods clinically very useful.

The method used for cardiac output measurements makes it possible to estimate the valve area in aortic stenosis. Being independent of regurgitations through any valve, the method is in some patients the only one by which the aortic valve area can be calculated.

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34. DIAGNOSIS AND ASSESSMENT OF TRICUSPID REGURGITATION WITH DOPPLER ULTRASOUND

T. SKJÆRPE and L. HATLE

Velocity of blood flow through the tricuspid valve was recorded noninvasively by Doppler ultrasound with a combined pulsed and continuous wave (cw) Doppler (Pedof) [1]. Tricuspid regurgitation (TR) was diagnosed when reverse flow in systole, originating at the tricuspid orifice, could be followed back into the right atrium (RA) (Figure 1). With significant tricuspid regurgitation, forward flow across the valve increases, and assessment of the degree of regurgitation was attempted from the velocity of forward flow across the valve, the intensity of the Doppler signal from the regurgitant jet (increases with increased flow) and the extension of the TR in the right atrium. From these Doppler data the patients were divided in three groups, mild, moderate and severe regurgitation.

Maximal velocity in the regurgitant flow in TR will depend upon the right ventricular (RV) to right atrial pressure difference in systole, the higher this pressure difference the higher the velocity. Even with normal RV pressure

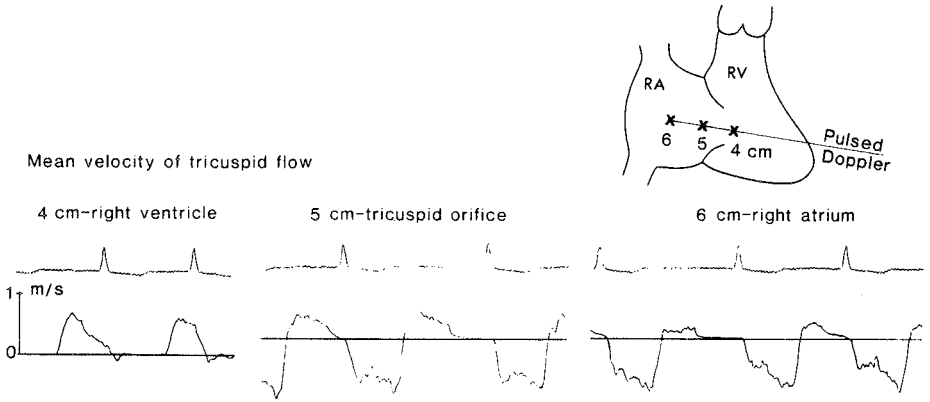

Figure 1. Velocity of tricuspid flow recorded noninvasively with pulsed Doppler. In the right ventricle, flow is towards the transducer (positive) in diastole. Reverse flow during systole is recorded at the valve area and in the right atrium. At the valve, velocity of forward flow in still recorded as in the ventricle, in the atrium it is decreased or no longer recorded. The sound of valve movements, together with flow signals, are used to localise the valve area; tricuspid valve opening is best recorded in the right ventricle, the closing movement best at the orifice. Tricuspid and mitral flow are distinguished by the different location and by the respiratory variation present in tricuspid flow velocity.

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this may be above the limit for the pulsed Doppler. With the cw mode, the audio signal was used to find the highest frequency shift and velocity in the TR in 29 patients. From maximal velocity, RV-RA pressure drop in systole was calculated as in obstructions [2] and compared to the pressure drop recorded at catheterization.

PATIENTS

Tricuspid regurgitation was diagnosed in 85 patients during a 2½ year period. Seventy of the patients were catheterized. In the 31 patients where a right ventricular angiogram or contrast echocardiography (3 cases) was done, the diagnosis was confirmed in all. In another 8 patients, a pathological V-wave in the RA pressure curve indicated TR. Twelve of the patients died and an autopsy was done in all cases. Age was from 6–86 years (mean 52). The diagnoses are seen in Table 1. The patients with secundum atrial septal defects (ASD II) were either elderly with dilated right ventricles (3 cases), or young with combined ASD II and pulmonary stenosis (PS). Among the patients with coronary heart disease, 3 had acute myocardial infarctions. One died and autopsy showed involvement of the right ventricle and papillary muscles. Pulmonary heart disease included 2 patients with acute pulmonary emboli and 4 with primary pulmonary hypertension. A primary lesion of the tricuspid valve or valve apparatus was present in 8 (Ebsteins malformation in 2, a malformed tricuspid valve in one with ASD II and PS, combined tricuspid stenosis and regurgitation in 3 and ASD I in 2) and possibly in another 8 (repair of congenital heart disease in 7 and papillary muscle infarction in one). In the 69 others, the tricuspid regurgitation was most likely secondary to pulmonary hypertension or right ventricular dilatation. Forty-nine patients were in RV failure. A clinical diagnosis of TR was based on one or more of the following: visible systolic pulsations of neck veins, palpable systolic pulsations of the liver and a systolic murmur over the lower

Table 1. Cause of tricuspid regurgitation in 85 patients.

Valvular heart disease		37
mitral	29	
aortic	4	
combined	4	
Congenital heart disease		22
ASD II, ASD II+PS	7	
ASD I	2	
PS, Fallot	5	
Other	8	
Coronary heart disease/cardiomyopathy		17
Pulmonary heart disease		7
Constrictive pericarditis		2

third of the sternum, or at the lower left sternal border, with increase during inspiration.

RESULTS

A clinical diagnosis was made in 32 of the 85 patients (38%), the frequency of a clinical diagnosis was not influenced by the presence of RV failure. Table 2 shows the frequency of clinical signs related to severity assessed by Doppler and to presence of abnormal V-waves in the RA pressure curve. A clinical diagnosis was made in only 1 of the 35 patients with a mild lesion, it also failed in nearly half of those with moderate TR and in 2 of 11 with severe TR. In the 2 with severe TR, contrast echocardiography also indicated pronounced regurgitation. Both had large right atria, which might have decreased the effect of TR on the peripheral veins. Pathological V-waves were seen in 82% of patients assessed as having severe TR and in 59% of those with moderate TR. The clinical diagnosis of systolic pulsations in neck veins and/or liver correlated well with the presence of V-waves in the RA pressure tracing; pulsations were noted in 22 out of 27 cases.

The murmur of TR was in most cases soft and low grade and, since many patients had louder murmurs from other lesions, the respiratory variations of the former might not have been possible to detect. In several patients no systolic murmur could be heard despite auscultation following the Doppler diagnosis. Five patients had a harsher murmur, grade III-IV, 3 of these had severe pulmonary hypertension with hypertrophic non-dilated right ventricles, the other two had Ebstein's malformation. A systolic murmur with inspiratory increase was the only sign of TR in 6 patients.

Table 2. Tricuspid regurgitation (TR), diagnosis and assessment by Doppler compared to clinical diagnosis.

		Severity of TR		
	Total	mild	moderate	severe
No. of patients	85	35	39	11
Clinical diagnosis	32 (38%)	1	22	9
systolic venous pulsations	23	1	15	7
systolic hepatic pulsations	20	0	13	7
murmur of TR	18	0	12	6
No. catheterized *	62	22	29	11
Abnormal V-waves	27 (44%)	1	17	9
Systolic venous pulsations	22	0	15	7

* In close relation to the clinical and Doppler examination.

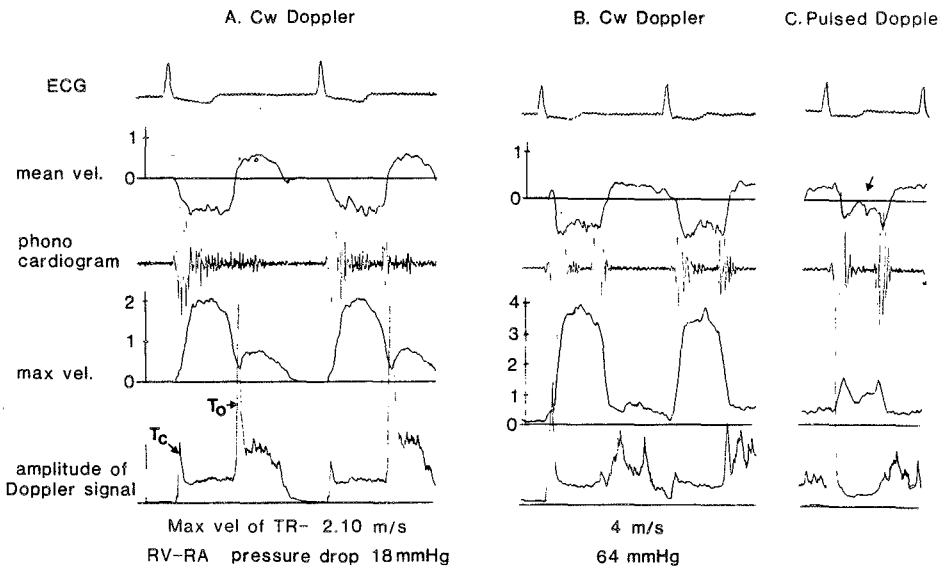

Figure 2. With cw Doppler, high velocities in the tricuspid regurgitation can be recorded and used to calculate the right ventricular (RV) – right atrial (RA) pressure difference in systole. A, maximal velocity (non-directional) indicates a RV pressure within the normal range. In B, maximal velocity indicates a RV pressure about 70 mm Hg (with normal RA pressure) or higher (raised RA pressure). C = pulsed Doppler at the valve area in the same patient. With the pulsed mode, high velocities cannot be recorded and the mean velocity curves shows a midsystolic artefact due to velocities above the limit for the pulsed mode. In the amplitude curve valve movements are clearly seen. T_c = tricuspid valve closure. T_o = tricuspid valve opening.

Maximal velocity of forward tricuspid flow was 0.61 m/s (range 0.34–0.90) in mild TR, 0.79 m/s (0.46–1.25) in moderate and 1.02 m/s (0.64–1.50) in severe TR (normal range 0.35–0.80 m/s, unpublished data). With reduced cardiac output, as in heart failure, lower velocities are found and may then be within the normal range, even in the presence of severe TR. With normal cardiac output, velocities up to 1.5 m/s were found indicating a peak pressure drop from RA to RV of 9 mmHg in early diastole in the absence of tricuspid stenosis.

Figure 2 shows maximal velocity in the TR in one patient with normal and one with raised RV pressure. Panel C shows the limitations of the pulsed mode when high velocities are present. The amplitude curve indicates the intensity of the Doppler signal. Maximal velocity in TR ranged from 1.32–4.04 m/s – with calculated RV-RA pressure drops of 7–65 mmHg. In Figure 3 the calculated and recorded RV-RA pressure drops are compared. In patients with raised RV pressures, the high velocities in the TR were easy to find and the good correlation with recorded pressures indicates that small angles to the regurgitant jet could be obtained in most. With a noninvasive estimate of the RV-RA pressure difference in systole available, RV systolic

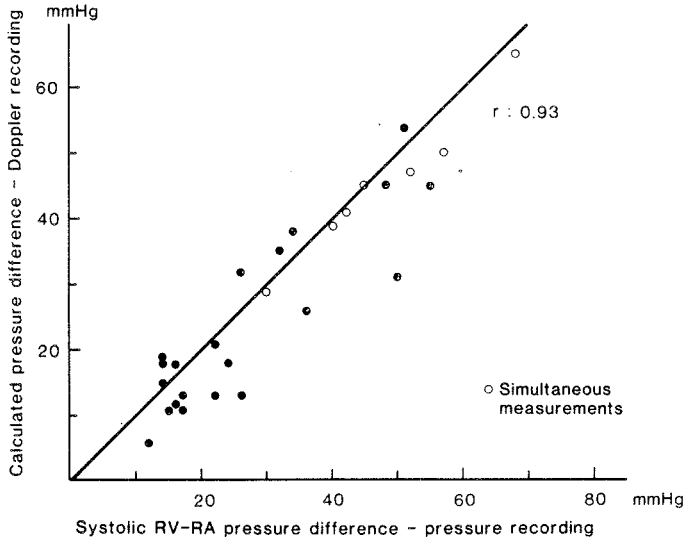

Figure 3. Right ventricular – right atrial pressure difference in systole calculated from maximal velocity in tricuspid regurgitation recorded noninvasively with Doppler and compared to the pressure difference recorded at catheterization.

pressure can also be estimated if the level of RA pressure can be judged from the neck veins.

CONCLUSION

Diagnosis of TR with Doppler is an easy and sensitive method as compared to the clinical diagnosis, which could be missed even in significant regurgitation. The lack of clinical signs in many of the patients with TR could mostly be explained by lack of abnormal RA pressure curves and abnormal venous pulsations in many of the patients with mild to moderate TR, and by the presence of systolic murmurs from other lesions. Comparing the attempted grading of the TR to the clinical and hemodynamic data indicates that semiquantitation of the TR may be possible. RV-RA pressure drop in systole can be calculated from cw Doppler recording of maximal velocity in the regurgitant jet.

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NONINVASIVE ESTIMATION OF PULMONARY ARTERY PRESSURE BY DOPPLER ULTRASOUND IN TRICUSPID REGURGITATION.

T. SKJÆRPE, L. HATLE

INTRODUCTION

Systolic pulmonary artery pressure (SPAP) can be estimated noninvasively from the pulmonary valve closure (Pc) - tricuspid valve opening (To) interval (1,2). In tricuspid regurgitation, however, this may be unreliable. SPAP is underestimated in some patients especially with severe regurgitation, even if a raised right atrial pressure is added.

The maximal velocities in tricuspid regurgitation depends on the pressure drop from the right ventricle (RV) to the right atrium (RA) in systole. The higher the pressure drop, the higher the velocities. Therefore, another way to estimate SPAP noninvasively in tricuspid regurgitation is to record the maximal velocities in the regurgitant jet by continuous wave doppler and calculate the RV-RA pressure drop from the Bernoulli equation (3). RA pressure jugded by neck vein congestion is added to obtain an estimate of systolic RV pressure. This should equal SPAP when pulmonary stenosis is excluded by doppler.

This work was aimed at the evaluation of both methods in estimating SPAP in tricuspid regurgitation.

METHODS

A combined pulsed and continuous wave doppler (Pedof) was used. This displays maximum and mean velocities by frequency estimation. These were recorded together with the amplitude of the doppler signal, ECG and phono cardiogram. In some patients a combined 2D-echo, pulsed and continuous wave doppler with spectral display (Irex III B) was used.

Tricuspid regurgitation was diagnosed when reverse flow in

systole, originating at the tricuspid orifice, could be followed back into the RA. The audio signal was used to find the highest frequency shift to obtain a small angle between the ultrasound beam and the regurgitant jet. The maximal velocity was recorded with continuous wave doppler and RV-RA pressure drop calculated. Semiquantitation of tricuspid regurgitation with doppler seems possible (4,5). Based on the extension of the regurgitant jet into the RA, the intensity of the signal from the jet and the velocity of forward flow, the patients were divided in groups of mild, moderate and severe regurgitation.

Ra, or central venous pressure (CVP), was roughly estimated from neck vein congestion. No congestion was taken as CVP of 0 mm Hg. Congestion to the level of the clavicle was taken as CVP of 5 mm Hg. For each additional cm of congestion 1 mm Hg was added to the CVP. The level of congestion was measured along a vertical line above the clavicle.

The Pc-To interval was recorded as described by Hatle (2). Correcting the interval for frequency, the SPAP is read from a table constructed by Burstin (1).

A right heart catheterization was done in all. In most patients there was a time interval of less than a few days between the catheterization and the doppler examination. The measurements were done simultaneously in seven.

PATIENTS

Tricuspid regurgitation was diagnosed with doppler in 92 catheterized patients. In 18 a RV angiogram was done and the diagnosis confirmed in all. Contrast echocardiography was performed in 27 patients. It was positive in 3 of 8 with mild tricuspid regurgitation, in 8 of 10 with moderate regurgitation and in all with severe regurgitation. In 21 patients examined with the Irex III B origination of the reverse flow could be located to the closure level of the tricuspid valve in all (figure 1).

Pathological v-waves in the RA pressure curve were found in 10 patients in sinus rythm (v-wave higher than a-wave) and in 25 patients in atrial fibrillation. All except 5 were found in patients with moderate or severe regurgitation.

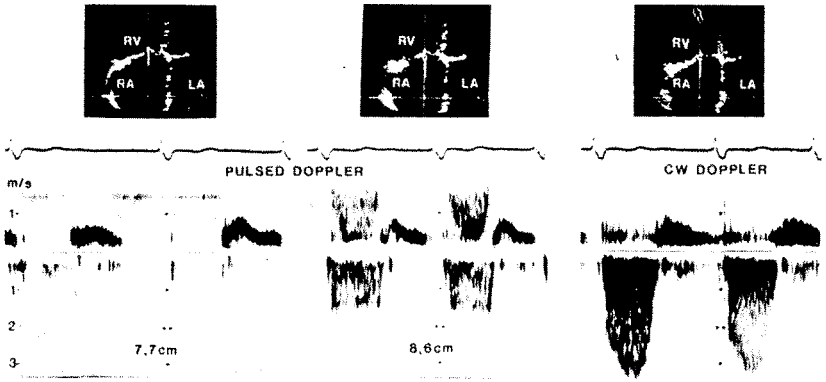

FIGURE 1. With pulsed doppler the regurgitant flow can be located to the closure level of the tricuspid valve. To the left the sample volume is placed in front of the valve and only forward flow is recorded. In the middle the sample volume is placed behind the valve. Reverse flow is now recorded. Switching to continuous wave doppler makes recording of maximal velocity possible (right).

The clinical diagnosis was based on one or more of the following signs: Visible systolic neck vein pulsations, palpable systolic pulsations of the liver and a murmur with inspiratory increase located over the lower part of the sternum or at the left sternal border. In patients with mild tricuspid regurgitation clinical diagnosis was made in only 4 of 40, in those with moderate regurgitation in 26 of 38 and failed even in 2 of 14 patients with severe regurgitation.

The diagnoses are shown in table 1.

Table 1.

Valvular heart disease		37
Mitral	24	
Aorta	5	
Combined	8	
Ischemic heart disease/cardiomyophatia		18
Pulmonary heart disease		5
Congenital heart disease		24
Constrictiv pericarditis		2
Other		6

Except patients with congenital lesions involving the right heart, the regurgitation in the remaining 70 patients most likely was secondary to impaired left heart function or pulmonary vascular disease. 15 % of the patients with mild regurgitation, 49 % of those with moderate, and 50 % of those with severe regurgitation were in RV failure.

RESULTS

Maximal velocity in the tricuspid regurgitation was obtained in 67 of the 76 patients where continuous wave doppler was used. It was missed in 6 with mild regurgitation, in 3 with moderate and in none with severe regurgitation. The main problem in recording these velocities was a very low intensity of the doppler signal, especially when problems of impaired ultrasound transmission were added (emphysema etc.). Ideally a velocity curve should have a smooth, rounded top. The rejected curves showed uneven, jagged tops. Curves showing the first part of the rounded top, but where the top itself was lost, were accepted

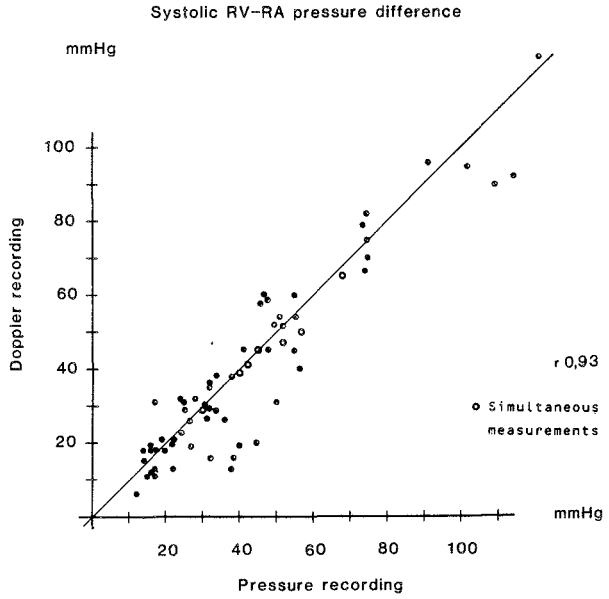

FIGURE 2. RV-RA pressure drop calculated from the maximal velocities in tricuspid regurgitation compared to pressure drop recorded at catheterization.

despite some underestimation of velocity would occur.

The velocities ranged from 1.2 to 5.6 m/s giving calculated RV-RA pressure drops of 6 to 125 mm Hg. In figure 2 calculated pressure drops are compared to pressure drops recorded at catheterization. The good correlation indicates that a small angle to the regurgitant jet is obtained in most. Twenty-one patients were examined with spectral analysis of the doppler signal. The maximal velocities were recorded in all, and correlation of calculated to recorded pressure drop was very good (figure 3). This

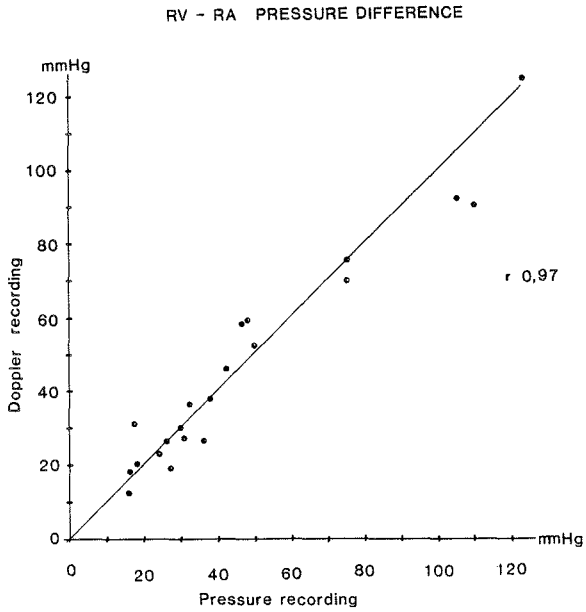

FIGURE 3. RV-RA pressure drop calculated from maximal velocities in tricuspid regurgitation obtained by spectral analysis of the doppler signal. Correlation to pressure drop recorded at catheterization.

indicates that the maximal velocity in tricuspid regurgitation is better displayed by spectral analysis than by frequency estimation. When CVP jugded by neck vein congestion is added to calculated pressure drop, correlation to invasively recorded SPAP as shown in figure 4. Compared to RA pressure recordings, CVP is generally underestimated by a few mm Hg. In patients with very high CVP, however, underestimation was up to 15 mm Hg in some.

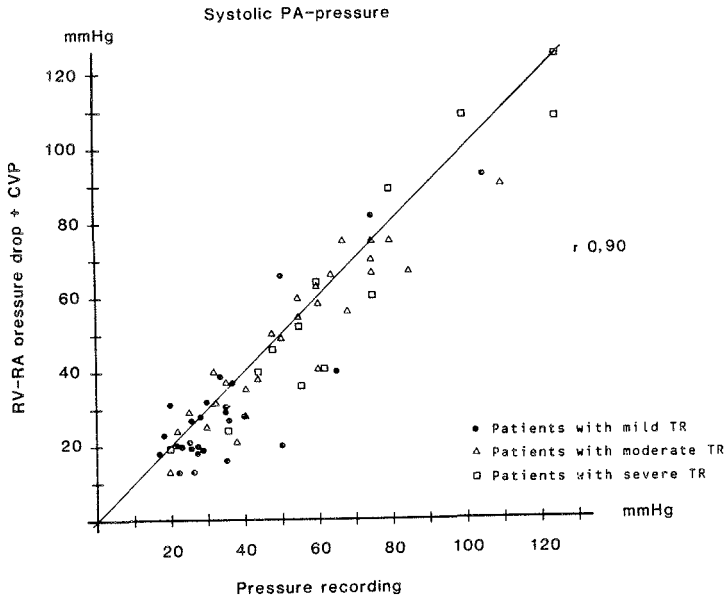

FIGURE 4. Estimation of SPAP by adding clinically jugded CVP to calculated RV-RA pressure drop compared to invasively recorded pressure.

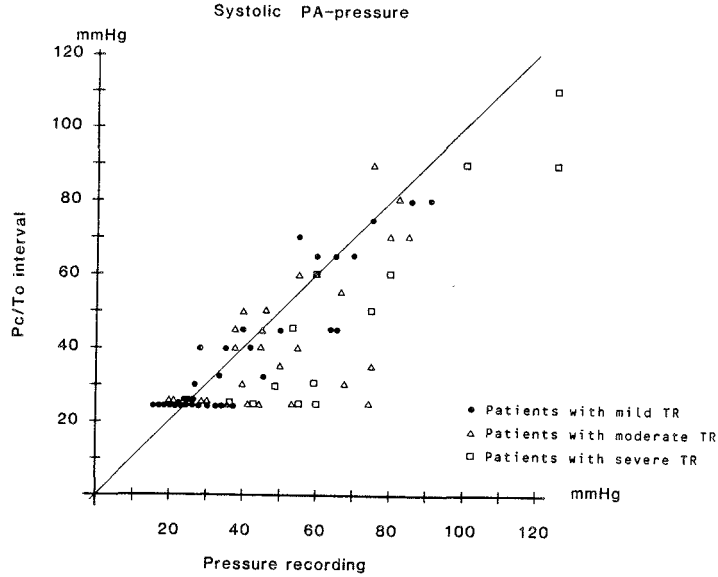

FIGURE 5. SPAP estimated from the Pc-To interval compared to invasively recorded pressure. SPAP below 30 mm Hg is not specified in Burstin's table. In this figure 25 mm Hg is choosen.

In 83 patients where we tried to record the Pc-To interval, we succeeded in 75. The main reasons for not being able to record the interval were difficulties in recording the pulmonary valve closure and locating P2 in the phono registration. In a few patients the early diastolic opening of the tricuspid valve was difficult to record. At rapid heart rates care should be taken not to mix the early opening of the valve with the re-opening following atrial contraction. Figure 5 shows how SPAP estimated by this method correlates to catheterization pressure. SPAP is underestimated in some with severe and moderate regurgitation, but correlation in mild regurgitation is good.

Clinically jugded CVP added to calculated RV-RA pressure drop gives a good estimate of SPAP. However, as no overestimation of SPAP occurs with the two methods described, it seems adequate to choose the highest value obtained when both methods are used. When only the Pc-To interval is recorded, SPAP can be estimated from this if the tricuspid regurgitation is jugded by doppler to

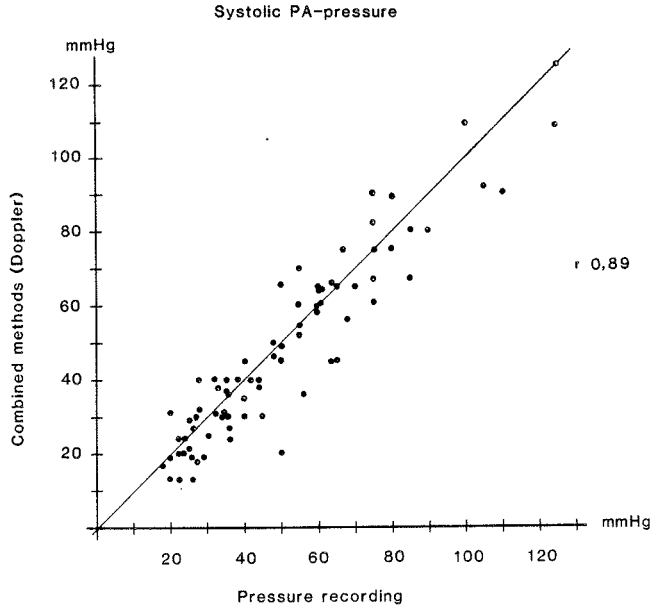

FIGURE 6. Noninvasive estimation of SPAP by doppler in tricuspid regurgitation. The two methods are combined as described in the text and compared to pressure recorded at catheterization.

be mild. If the results are combined in this way, a good estimate of SPAP is found in 79 of 92 patients (figure 6).

CONCLUSION

Systolic pulmonary artery pressure in patients with tricuspid regurgitation can be estimated noninvasively by doppler ultrasound in most. Semiquantitation of the regurgitation is necessary to get reliable estimates from the Pc-To interval. There remains a problem of locating Pc in some patients. The problem of recording the maximal velocities with continuous wave doppler in difficult patients, and when the intensity of the signal is very low, seems to a large extent solved by spectral analysis of the signal.

The two methods supplement each other as the Pc-To interval can be used in mild tricuspid regurgitation where the maximal velocities sometimes is difficult to obtain. In moderate and severe regurgitation, where the Pc-To interval can not be used alone, the maximal velocities are seldom missed.

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Noninvasive estimation of systolic pressure in the right ventricle in patients with tricuspid regurgitation

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EY WORDS: Right ventricular systolic pressure, Doppler ultrasound, tricuspid regurgitation.

70 patients, tricuspid regurgitation was diagnosed with Doppler by recording reverse flow in systole, iginating at the tricuspid orifice, directed into the right atrium. The peak velocities were recorded, and the ak pressure drop from the right ventricle to the right atrium in systole was calculated from the simplified rnoulli equation (pressure drop = $4 \times 4 \times Vmax^2$), and was found to correlate well with invasive measure-nts ($r = 0{\cdot}97$, SEE $\pm 6{\cdot}1$ mmHg). Central venous pressure was judged by neck vein congestion and added the pressure drop to obtain a noninvasive estimation of systolic right ventricular pressure. Correlation with theterization measurements was good ($r = 0{\cdot}96$, SEE $\pm 7{\cdot}1$ mmHg). In patients in whom the tricuspid gurgitation was judged as mild with Doppler, the correlation between noninvasive and invasive measure-nts of the transtricuspid pressure drop was still good ($r = 0{\cdot}95$, SEE $\pm 5{\cdot}1$ mmHg), indicating that the cosity of the blood does not invalidate the use of the simplified Bernoulli equation when the regurgitant ea of the valve is small.

troduction

In earlier reports[1,2] we evaluated a new non-vasive method for the estimation of the systolic lmonary artery pressure (SPAP) in patients with cuspid regurgitation. The maximal velocities in e regurgitant jet were recorded with continuous ave Doppler and the pressure drop from the right ntricle to the right atrium in systole (RV–RA essure drop) was calculated from the simplified rnoulli equation.[3,4] Central venous pressure VP) judged by neck vein congestion was added obtain an estimate of systolic right ventricular essure. In the absence of right ventricular out-w obstruction, this should equal the pulmonary tery systolic pressure.

Underestimation of the RV–RA pressure drop as seen in some patients with mild tricuspid gurgitation when the maximal velocity was dis-ayed by an analog frequency estimator. When e signal to noise ratio was poor, the frequency timator was not able to track the maximal velo-y at all. These limitations seemed to be elimi-nated in a subgroup of patients where the velocities were displayed by spectral analysis.[2] This is in accordance with the results of others.[5–8] The purpose of this study was to evaluate the usefulness of the method in a larger group of patients when a spectral analyzer was used to display the velocities in the tricuspid regurgitation.

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Methods

PATIENTS

The study group consisted of 70 patients who has tricuspid regurgitation diagnosed by Doppler according to the diagnostic criteria described earlier.[1,2,9–12]

Table 1 Diagnoses

Mitral stenosis and/or regurgitation	24
Aortic stenosis and/or regurgitation	11
Combined aortic and mitral valve disease	6
Mitral valve prosthesis	2
Combined mitral and tricuspid stenosis and regurg.	1
ASD	9
VSD	6
Functional single ventricle	7
Others	4

Others include 1 patient with constrictive pericarditis, 1 with hypertrophic cardiomyopathy, 1 with chronic lung disease, and 1 patient with lymphoma and obstruction of pulmonary vein drainage.

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There were 45 men and 25 women. Age was 2 to 2 years (mean 51 years). The diagnoses are listed n Table 1.

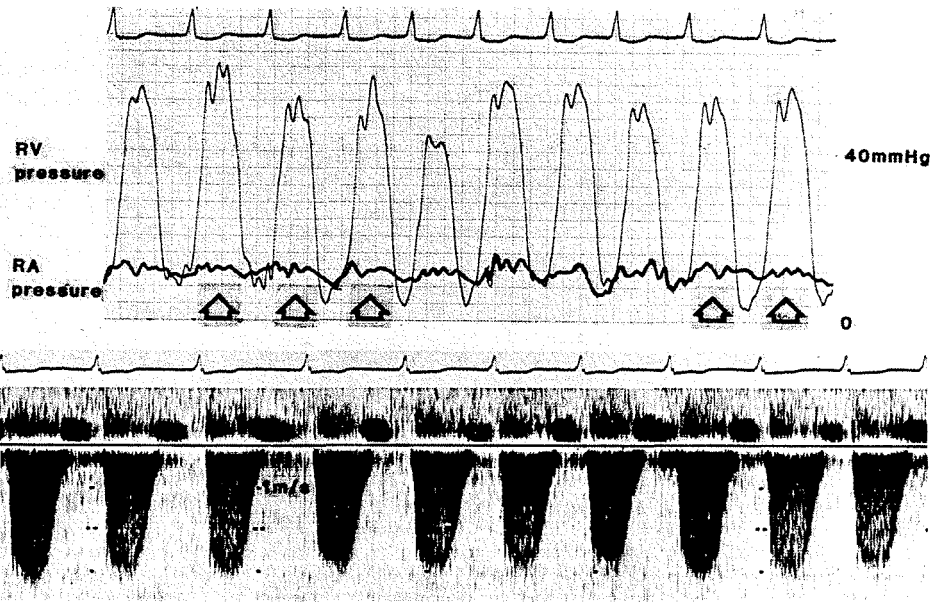
ELOCITY RECORDINGS AND PRESSURE DROP ALCULATIONS

The velocity recordings were made with a cross-ectional echocardiograph machine (Irex III B, rex Corporation) which includes pulsed and con-nuous wave Doppler (Pedof, Vingmed). With this nstrument, pulsed and continuous wave Doppler neasurements are possible simultaneously with naging. For optimal sensitivity, velocity record-ngs with a separate Doppler transducer are also ossible. The velocities in the tricuspid regurgitant t were recorded both from the apex and from ne lower left parasternal position to obtain the nallest possible angle to the direction of flow. The elocities were always related to the opening and osure of the tricuspid valve to exclude aortic flow elocities which might be picked up by sidelobes of ne ultrasound beam. When necessary, the separate oppler transducer was used to obtain velocity ecordings with a continuous envelope. The RV–RA pressure drop was calculated from the peak velocity using the simplified Bernoulli equation (Fig. 1), and averaged over 5 consecutive beats in sinus rhythm, and over 10 beats in atrial fibrillation. In patients in whom only a few beats with a continuous envelope were recorded, these beats were used for the pressure drop calculation. Patients in whom regurgitant flow was recorded in early or late systole only, were not included in the study.

CVP was estimated by the level of neck vein congestion, measured vertically above the clavicle at 30 degrees elevation of the thorax and the head of the patient. Further elevation was made if the level of congestion was very high. The atrial pressure was assumed to be zero if no vein congestion was seen, and 5 mmHg if the congestion was up to the level of the clavicle. For each cm of vein congestion above the clavicle, 1 mmHg was added. The highest level during systole was measured.

Right ventricular outflow obstruction was excluded or quantitated by recording the flow velocities below and above the pulmonary valve.[11]

Since Doppler measurements may underestimate



igure 1 Above: The invasive right ventricular pressure curve with the right atrial pressure curve superimposed. ive beats with preceding R–R intervals representative for the mean heart rate are marked. Averaged peak ressure drop is 43 mmHg (RVP = 54 mmHg). Below: Recording of the velocities in the tricuspid regurgitant jet. veraged peak pressure drop is 36 mmHg. Two cm of neck vein congestion gives an estimated RVP of 43 mmHg.

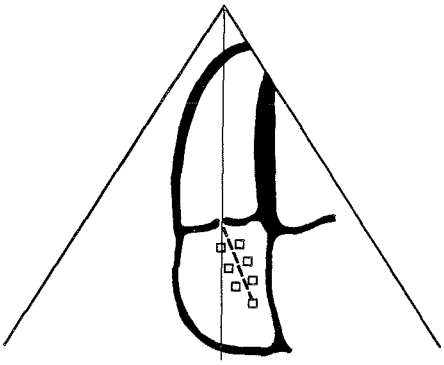


Figure 2 Mapping of the tricuspid regurgitant jet into the right atrium. The squares indicate the positions of the sample volume when the velocities in the jet fell below the cut-off frequency of the high pass filter of the Doppler instrument. The broken line indicates the assumed direction of the jet. A second mapping was made in the orthogonal plane by rotating the transducer 90 degrees.

the velocities in the regurgitant jet if the angle between the ultrasound beam and the direction of flow is too large, in some patients we mapped the axial and the lateral extension of the regurgitant jet into the right atrium in two orthogonal planes with pulsed Doppler. The measurements were made from the position where the highest velocities were recorded. The midline between the lateral borders was assumed to represent the direction of the jet (Fig. 2). The largest angle to the ultrasound beam as indicated by the Doppler line on the cross-sectional image, in any of the two planes, was measured.

When the tricuspid regurgitant area is so small that the viscosity of the blood can no longer be neglected, the pressure drop calculated by the simplified Bernoulli equation will be underestimated.[3] To see if this was a problem in tricuspid regurgitation judged as mild by Doppler, the regurgitation was semiquantitated according to criteria developed earlier,[1,12] which are partly based upon the findings of Miyatake *et al.*[13] The patients were divided into groups of mild, and more than mild regurgitation.

CATHETERIZATION

The pressures in the pulmonary artery, the right ventricle and the right atrium were recorded with a 7F fluid filled, end hole catheter connected to an Elema 746 pressure transducer, or an Elecath Baltherm 7F thermal dilution catheter (Electrocatheter Corp.) connected to an AE 840 (Mikro elektronikk A/S) pressure transducer. The RV–RA pressure drop was measured by superimposing the pressure curves recorded on withdrawal of the catheter from the ventricle to the atrium. The peak pressure difference between the curves was measured and averaged over 5 beats in sinus rhythm. In atrial fibrillation beats with R–R intervals representative for the mean heart rate were chosen.

Since the right ventricular pressure at any moment equals the pressure difference from the right atrium plus the right atrial pressure at the same moment, the right atrial pressure was measured at the time of peak pressure difference during systole. The pressure, which will be referred to as 'the invasive RA pressure' was compared with the CVP judged by the neck vein congestion.

The patients were catheterized within two days of the Doppler examination, except for seven with functional single ventricles where an earlier catheterization had shown a ventricular pressure within 5 mmHg of the systolic blood pressure. In these, estimated right ventricular pressure was compared with the systolic blood pressure at the time of the Doppler examination. For pressure drop comparisons, an invasive RA pressure of 5 mmHg was assumed. At the time of catheterization all had invasive RA pressures between zero and 5 mmHg, and in none were significant clinical changes observed.

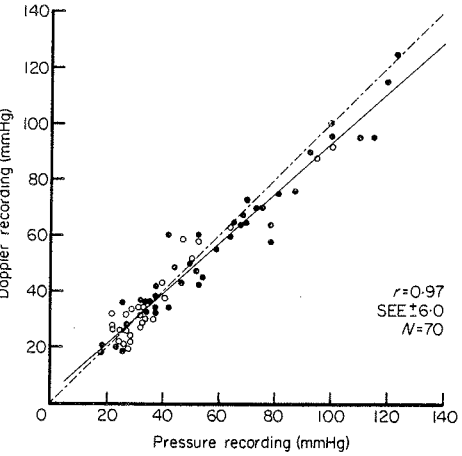
There was no change in medication between the invasive and the noninvasive measurements. Both were made 30–60 min after the patient had been placed in the supine position. Care was taken not to exercise the patients the last few hours before the examinations.

The invasive pressure recordings in the right ventricle and the pulmonary artery, and the Doppler velocity recordings were analyzed with no knowledge of the result of the other measurement. The superimposition of the right ventricular and right atrial pressure curves, and the calculation of the invasive RV–RA pressure drop was made at the end of the study when some knowledge of the Doppler results existed.

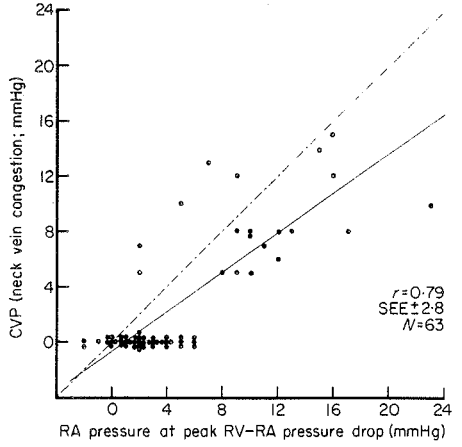
Results

In Fig. 3 the pressure differences between the right ventricle and right atrium as calculated from the velocity recordings and measured during catheterization, are compared. The good correlation

ndicates that a small angle between the ultrasound eam and the direction of flow could be obtained n most patients. In the 10 patients in whom angle neasurements were attempted, the result was etween 2 and 12 degrees in 9 patients, and 18 egrees in one (mean 8 degrees). The jet tended to e directed towards the atrial septum in most atients.



igure 3 Peak RV–RA pressure drops as calculated from the oppler recordings, compared with invasive measurements. pen circles: Patients with mild tricuspid regurgitation as dged by Doppler.



igure 4 CVP judged by neck vein congestion compared ith invasively recorded RA pressure at peak RV–RA pres- re drop. Seven patients with single ventricle not included ee text).

In Fig. 4, the invasive and noninvasive measurements of RA-pressures are compared. When the right atrial pressure was normal, or moderately elevated, the estimated pressure was within 6 mmHg of the recorded pressure. When the pressure was high, underestimations of up to 13 mmHg were made. Fig. 5 shows how the RA pressure at peak pressure difference to the right ventricle compared to the mean right atrial pressure. There was no constant relationship, the largest difference being 7 mmHg. In some patients, especially those with high pressures in the right atrium and ventricle, the peak RV–RA pressure difference often occurred at, or just before the highest systolic level of atrial pressure (Fig. 6, left), while in others, usually patients with moderately elevated or normal pressures, the peak RV–RA pressure difference was measured at the X-depression which could be significantly lower than the mean pressure (Fig. 6, right).

The correlation between noninvasive and invasive right ventricular pressures is shown in Fig. 7. The standard error of the estimate (SEE) was somewhat higher in patients with pressure higher than 35 mmHg (±7·6 mmHg) than in patients with lower pressures (±5·1 mmHg).

In patients without pulmonary stenosis according to invasive pressure recordings, the pressure drop across the pulmonary valve calculated from Doppler recordings was less than 4 mmHg. In four patients with pulmonary stenosis, or with pulmonary artery banding, the velocities in the stenotic

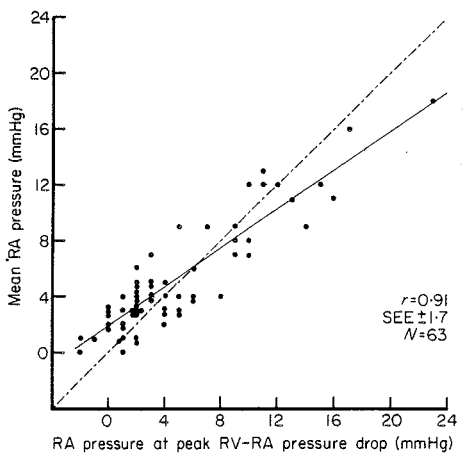


Figure 5 Comparison of the mean right atrial pressure with the right atrial pressure at peak RV–RA pressure drop. Seven patients with single ventricle not included (see text).

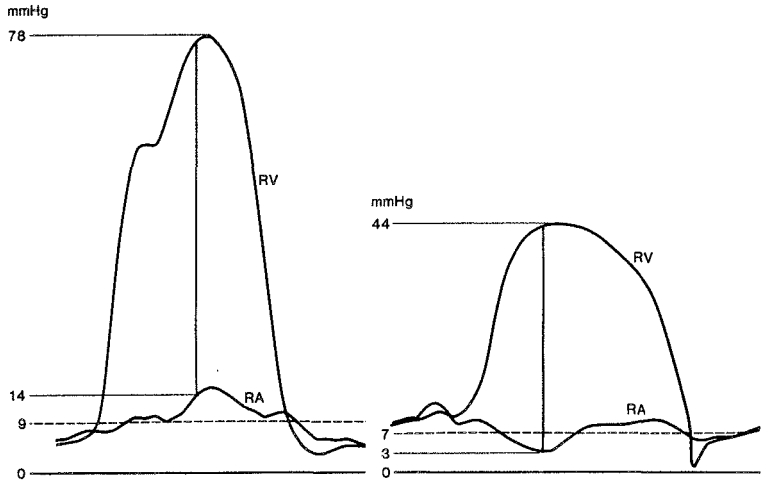


Figure 6 Invasive recordings of right ventricular and right atrial pressures in two patients. Timing of peak RV–RA pressure drop (vertical line) in relation to the shape of the pressure curves. Broken line: Mean right atrial pressure.

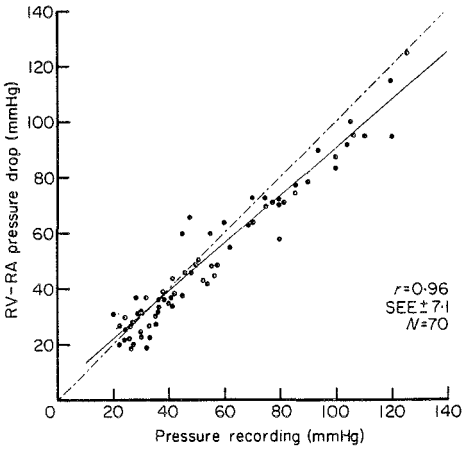


Figure 7 Noninvasive estimation of right ventricular (RV) pressure compared to invasive recordings.

jet were recorded. The pressure drop across the obstruction at the time of peak velocity in the tricuspid regurgitant jet was calculated, and when subtracted from the right ventricular pressure, a normal systolic pulmonary artery pressure was obtained. This was also found at catheterization.

Discussion

The results when noninvasive and invasive RV–RA pressure drops are compared (Fig. 3), are in close agreement with our first experience with the method when spectral analysis of the Doppler signal was applied,[2] and with the results of others.[5–8] Some errors may have been made by assuming an invasive RA pressure of 5 mmHg in 7 patients. However, none of these had neck vein congestion, and in other patients without vein congestion, the highest invasive RA pressure was 6 mmHg. The error is therefore probably small.

The somewhat better correlation obtained in this series when RV pressures are compared (Fig. 7), may partly be explained by the larger number of patients with high pressures, giving more data points at the upper end of the pressure range, and partly by differences in how the CVP was estimated clinically. Since we in our first experience with the method[2] tended to underestimate the true RA-pressure by the neck vein congestion, we did not consider it necessary to correct completely for the differences in density between blood and mercury (i.e. dividing cm vein congestion by 13 instead of 10). For the same reason we also measured the highest level of vein congestion during systole. This procedure seemed justified by observing that the mean right atrial pressure tended to underestimate the right atrial pressure at peak RV–RA pressure drop at the high end of the pressure range (Fig. 5 and Fig. 6, left). To assume a CVP of 0 mmHg when no neck vein congestion is seen, is also supported by Fig. 5. The majority of these patients had invasive RA pressures in the range of 0–2 mmHg.

Despite this, the noninvasive estimation of RA-pressure showed relatively larger errors than RV–RA pressure drop estimations. But, as pointed out by Yock and Popp[5], the absolute deviations from true RA pressure are relatively small compared to the higher RV–RA pressure drop.

The calculation of invasive RV–RA pressure drop was not completely blinded with regard to the Doppler results. There is little reason to believe that this has influenced the results. The measurements of noninvasive and invasive RVP were blinded, and correlated only slightly less well than the measurements of pressure drops. This is as expected since the noninvasive RVP is obtained by adding the less accurate estimate of CVP to the pressure drop.

A source of error may be inadequate recording of the whole velocity curve, making it difficult to measure the peak velocity. This may be a problem especially when there is little regurgitation, when the Doppler signal is of very low intensity, and in patients in whom the transmission of ultrasound is impaired (emphysema etc.). The problem was to a large extent solved by using the separate Doppler transducer. It was also considered necessary to adjust the greyscale of the hard copy so that the background noise was visualized, partly by applying a high degree of compression and minimal reject, partly by applying maximal high pass filtering to reduce the effect of the automatic gain control. In this way, velocity components with an intensity down to the noise level, was displayed. Despite these precautions, it was possible to display the whole velocity curve only in a few beats in 7 patients. These beats were recorded mainly during inspiration, probably producing a higher noninvasive pressure drop than if beats from a complete respiratory cycle had been averaged. However, in no patient with such recordings was the noninvasive pressure drop higher than the invasive. This is partly explained by the moderate respiratory variations in RVP observed at catheterization in these patients. One should note, however, that underestimation of velocities caused by nonalignment between the regurgitant jet and the ultrasound beam will to some extent be compensated for, improving the correlation with invasive measurements.

Since significant variations in right ventricular and pulmonary artery pressure may be seen even during catheterization, the time interval of 1 to days between the noninvasive and invasive measurements, might account for some of the differences in estimated pressures, particularly when close correlation can be obtained with simultaneous measurements[2,5,6,7]. Near alignment between the flow direction in the regurgitant jet and the ultrasound beam can be obtained in most patients. This is supported by the results in 10 patients in the present series where the angle between the direction of flow and the ultrasound beam was found to be 18 degrees or smaller. The corresponding underestimation of pressure drop is up to 11%.

In mild tricuspid regurgitation (Fig. 3), the correlation when RV–RA pressure drops are compared, is still good ($r=0{\cdot}95$, SEE $\pm 5{\cdot}1$ mmHg). This indicates that viscosity does not invalidate the use of the simplified Bernoulli equation in patients with mild regurgitation.

Our results show that when pulmonary stenosis is excluded by Doppler, the estimated right ventricular pressure can be assumed to represent systolic pulmonary artery pressure. When pulmonary stenosis is present, the pulmonary artery pressure can be calculated by subtracting the pressure drop across the obstruction from the systolic right ventricular pressure. It should be noted that the peak pressure drop across the tricuspid and the pulmonary valve may occur at different times, and that the sum of the peaks may be larger than the peak right ventricular pressure. It is important to use pressure drops with identical timings during systole.

In our laboratory the noninvasive method has proved very useful in monitoring the pulmonary artery pressure both in congenital malformations and in acquired heart disease, and in all age groups. The usefulness is enhanced by the fact that Doppler is a very sensitive method for diagnosing tricuspid regurgitation[9,10,12] — probably the most sensitive available. Tricuspid regurgitation can be recorded with Doppler in the majority of cardiac patients both with and without pulmonary hypertension[5,6,14]. The method is also completely noninvasive.

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7 Cardiac Output

Terje Skjaerpe, Lars Hegrenaes, and Halfdan Ihlen

7.1 INTRODUCTION

The ability to deliver a sufficient volume of blood to meet the demands of the body is an important measure of left ventricular function. A simple, non-invasive method to estimate cardiac output and changes in cardiac output is therefore of considerable clinical importance.

Doppler ultrasound has the possibility of being such a method, and much research is being done to evaluate the reliability of this technique. The method is based on measurements of the velocity of blood flow in the aorta. The area under the velocity curve is integrated manually or automatically. The diameter of the aorta is measured by echo, and the cross-sectional area is calculated. The product of the velocity curve integral and the cross-sectional area gives the stroke volume. Cardiac output is calculated by multiplying stroke volume by heart rate. When the heart rate varies, as in atrial fibrillation, averaging stroke volume over several cardiac cycles is necessary. If only changes in stroke volume and cardiac output are of interest, the areas under the velocity curves can be compared directly.[1-9]

In theory, flow in the pulmonary artery and through the mitral and tricuspid orifice could also be used to quantitate cardiac output, provided an accurate estimate of the area through which the blood flows can be obtained. In animal models and in children,[10-19] this method has worked well, but more data are needed to establish these methods in an unselected group of patients.

Because most of the research conducted is on aortic blood flow, we concentrate on cardiac output based on measurements of the blood velocity and the cross-sectional area in the aorta.

7.2 AORTIC FLOW

A. Theoretical Considerations

In Section 2.3A, the theoretical conditions for a flat velocity profile in a vessel are discussed. These conditions apply to some extent to the ascending

aorta because the blood is accelerated and the diameter is large, so viscous resistance is low. Nonetheless, the ascending aorta has important geometrical features that obviously influence the velocity profile in a nonflat way.

First, the theory of flow in a uniformly curved tube anticipates higher velocities along the inner curvature during the first part of the bend.[20-22] During the course of the curvature, the higher velocities may be redistributed to the outer bend (Fig. 7-1).[21, 22] Because the aorta is curved both in the frontal and anteroposterior planes, the actual skew may be complex. In the arch, the branching of the arteries to the arms and the head may influence the profile. Wave reflections from these branches may even affect the velocity profile in the ascending aorta. The skew is expected to change during systole, owing to the different effects of acceleration and deceleration on the velocity profile.

Second, as the aorta expands during systole, some of the ejected blood has to fill this space. The direction of this blood flow deviates from the axial

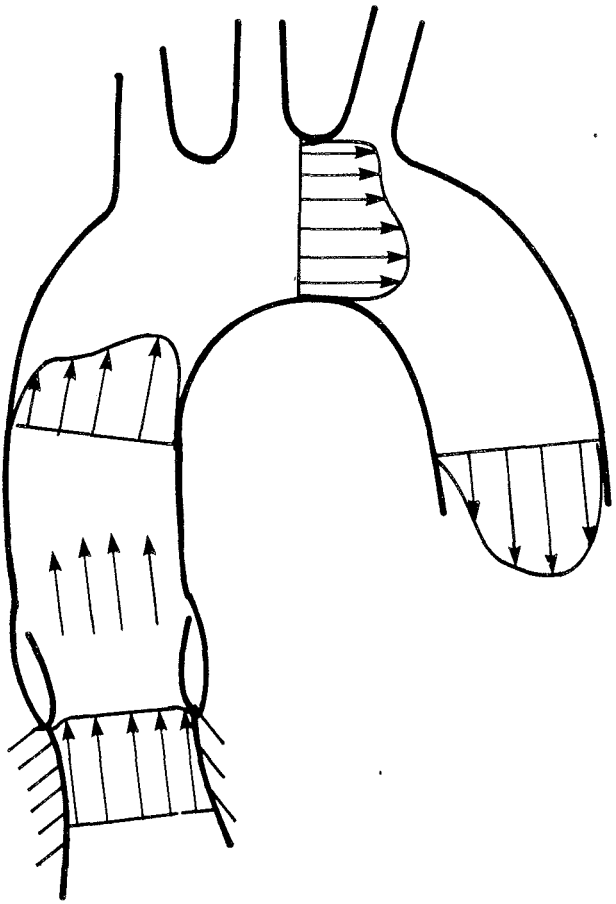

Fig. 7-1. Theoretically expected velocity profiles in the left ventricular outflow tract and at various levels of the aorta when no dilation of the ascending aorta is present.

direction and deforms the profile. As the increase in area during systole is only about 10%,[23] the effect on the velocity profile is probably small.

The third point is the most important in our experience. The aorta, except for the sinus of Valsalva, is often thought of as a curved tube with unchanging dimensions from the inlet to the arch. Such may be true in children and perhaps also in dogs, on which most of the experimental work is performed. In older people, the geometry of the aorta is usually different. The aorta has its smallest diameter at the inlet, the aortic orifice (Fig. 7-2). The diameter at the sinus of Valsalva is much greater. Superior to the sinus of Valsalva, the diameter again becomes smaller, but it is still larger than the diameter at the orifice. The left ventricular outflow tract is a converging channel, and because the blood is accelerated at this level, a flat velocity profile is expected at the aortic orifice (see Fig. 7-1). As the cross section of the aorta expands, a more peaked profile develops. Because inertia prevails on the velocities for some distance, a blunt central core with approximately the same velocities as at the orifice is expected, as indicated in Figure 7-2. Outside the core, the profile can be complicated, depending on the degree of expansion. With large dilations of the aorta, even eddies around the core can occur. Further downstream, the velocities reorganize into a more regular profile with lower peaks. This phenomenon is equivalent to the distal process in obstructions.[24]

B. Experimental Evidence

The experimental evidence on the velocity profile in the ascending aorta is conflicting. Schultz and colleagues describe a flat profile that persists throughout the cardiac cycle.[25] This finding is in contrast to a later article of Clark

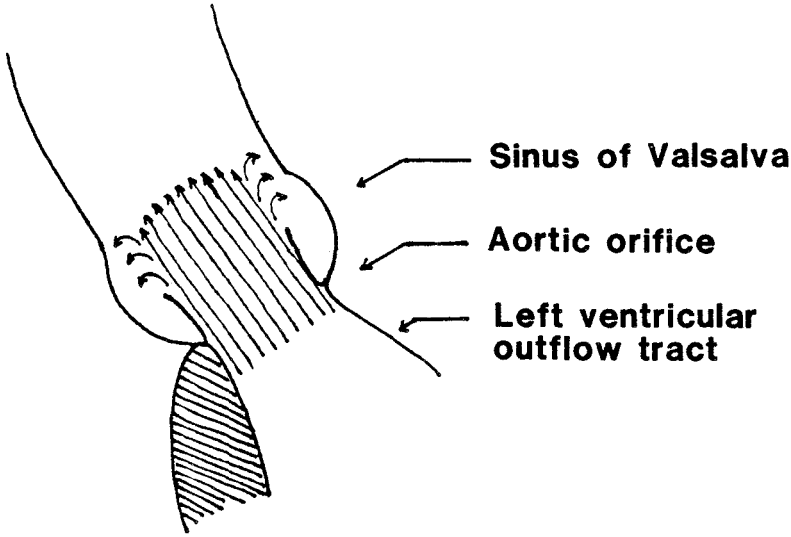

Fig. 7-2. Schematic drawing of the ascending aorta as found in some older people. The expected velocity distribution in the first part of the aorta is indicated.

and Schultz, who observe a skew.[26] Falsetti and co-workers also found a fairly flat profile in the baseline state.[27] During isoproterenol infusion, however, the profile was much more peaked, with a skew toward the posterior wall. Seed and Wood found a flat profile in early systole, but with a skew developing in late systole with a peaking of velocities toward the anterior wall.[20] The same is observed by Amyot and associates.[28] The presence of a flat velocity profile with a skew to the inner bend was shown by Tunstall-Pedoe,[29] Farthing and Peronneau[21] and Paulsen and Hasenkam.[30] The skew was found to vary with the timing within the cardiac cycle and with the distance from the valve. This variation complicates the full evaluation of the velocity profile. Falsetti's findings also show that the profile changes with the contractile state of the myocardium. When the velocities are integrated during the whole systole, the effect of temporal skewing is much less obvious, according to the work of Fisher and co-workers.[31] When a mean velocity was used, the localization of the same volume at various levels in the aorta was not critical. In some patients the skew can be pronounced, however, and small changes in sample site can cause large variations in recorded velocities. This situation is shown in Figure 4-1, which is from a patient with a low heart rate due to complete atrioventricular block. With increased left ventricular filling, enhancement of contractile force may give a situation similar to that observed by Falsetti in his dogs during isoproterenol infusion. (See also Figures 4-13 and 4-14.)

Little experimental evidence and little discussion exist on the way in which the changing dimensions of the aorta influence the velocity profile. Wille applied the finite element method for mathematical modeling of arterial flow.[24] The expected velocity pattern (see Section 7.2A) when blood is ejected into a wide aorta through a narrow orifice is supported by his results.

In an unselected group of patients, evidence exists of significant variations of the aortic velocity profile in which neither maximal nor mean velocity within the Doppler sample volume represents the cross-sectional mean velocity in the ascending aorta.

C. Volume Flow

To obtain volume flow, one should spatially integrate the velocity profile over the whole aortic cross section (see Section 2.2B). The detailed velocity profile is difficult to obtain because the ultrasonic access to the aorta is limited and the Doppler instrument has a limited resolution. If it were possible to insonify the whole cross-sectional area with a uniform beam, the mean frequency of the signal could be used to obtain the volume flow from Equation 2.17. It would still be difficult to avoid interference from flow outside the aorta. A more practical approach is to measure the velocities at a level at which the profile is practically flat. Then the spatial average velocity is close to the maximal velocity found. When the cross-sectional area at this level is measured, the volume flow is obtained as the product of this area and the velocity (Equation 2.17).

This reasoning suggests that the velocities should be measured at the level of the aortic orifice. Because the signal here is disturbed by the valve movements, however, these velocities can be measured superior to the valve level,

according to the arguments in Section 7.2A and B. It is then necessary to record the highest velocities because these represent the velocities at the aortic orifice.

Seed and Wood found the centerline velocities in the ascending aorta to approximate the cross-sectional mean accurately.[20] Fisher and associates showed that this method worked well in an animal model.[31] These workers also applied some angle correction. This technique requires a combined two-dimensional echo-Doppler transducer. The size of this transducer limits adjustments of transducer position in the suprasternal notch, even with transducers specially designed for this purpose. In addition, angle corrections can only be made in one plane, not the azimuthal plane. On the other hand, a specially designed single Doppler probe is small enough to permit considerable movement in the suprasternal notch. Preliminary evidence in our laboratory also indicates that the centerline velocity in some patients is higher than the cross-sectional mean velocity. Such patients have dilation of the ascending aorta, when compared to the orifice. The assumption of a jetlike profile in those persons thus seems to be confirmed. This problem is not avoided by using a mean frequency estimation. Fisher found that the sampling site was not critical. One should remember that he used an open-chest dog model. When the probe's position is fixed to the suprasternal notch, the angle to the direction of flow changes as the sample volume is moved to the arch, and the velocities are underestimated (Fig. 7-3). This problem could be solved by angle correction. The foregoing limitations should be borne in mind, however.

7.3 DOPPLER MEASUREMENTS

Although adequate signals in many patients can be obtained from the aortic arch or the first part of the descending aorta, we prefer to record signals from the ascending aorta, for reasons described in the previous section. In addition, the quantitation of cardiac output from flow velocities in the descending aorta is difficult, owing to problems in diameter measurements at this level. Part of the cardiac output has also left the aorta through the arteries to the arms and head. Coronary flow is greatest during diastole, and any influence on the quantitation of flow in the ascending aorta during systole should be small.

As always, when Doppler ultrasound is used to quantitate velocities, it is essential to try to align the ultrasound beam to the direction of flow. This alignment is possible in most patients when the aortic flow velocity is recorded from the suprasternal notch.[32, 33] To direct the ultrasound beam down the ascending aorta, some dorsal pressure must be applied to overcome the shadowing effect of the upper sternum. This technique is easier when the transducer is not too large. In some patients, obtaining adequate signals can be difficult. In our experience, such is especially the case with elderly, obese patients with short necks and an emphysematous thorax. Better signals may be recorded from the apex. Higher velocities may even be found from this position owing to a smaller angle to the direction of aortic flow (Fig. 7-4).

Flow velocity in the ascending aorta is usually best recorded in the supine position. It is also easier to maintain a stable signal because the patient is more relaxed. When the patient is sitting, better signals may be obtained if he leans slightly forward. When aortic flow is recorded from the apex, the best

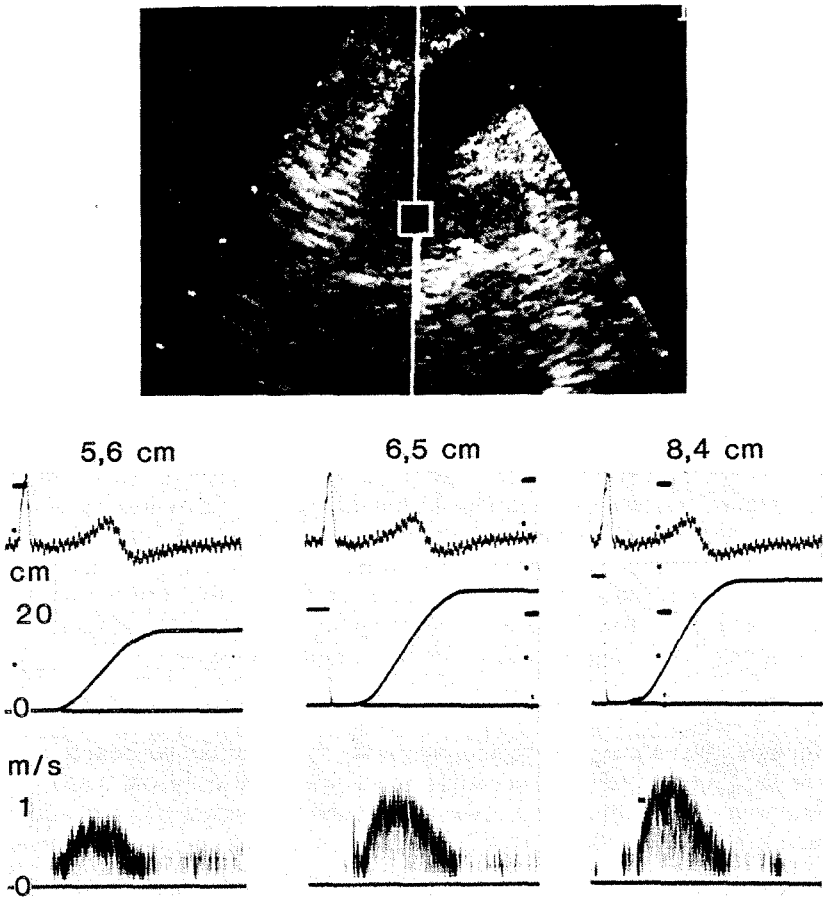

Fig. 7-3. When the sample volume is moved down the ascending aorta, higher velocities are recorded because of a decreasing angle to the direction of flow. This change is expected because of the curved course of the aorta (two-dimensional picture). A corresponding increase in integral size is seen.

signals are obtained when the patient is partly turned over to the left and the transducer is placed over the apex.

To find the highest velocities, the transducer should be directed to where the highest pitch of the received Doppler signal is heard. Both continuous wave (CW) and pulsed doppler can be used. In CW Doppler, all velocities along the sound beam are recorded. In this way, the highest velocities are more easily found. Because more noise is also picked up, however, the velocity curve may be more difficult to trace and to integrate by an automatic integrator. This drawback can cause some underestimation of the maximal velocity integral; the mean velocity is likely to be underestimated because of interference from low-frequency signals outside the aorta (Fig. 7-5).

In the pulsed mode, it is usually easy to record a satisfactory signal, but in

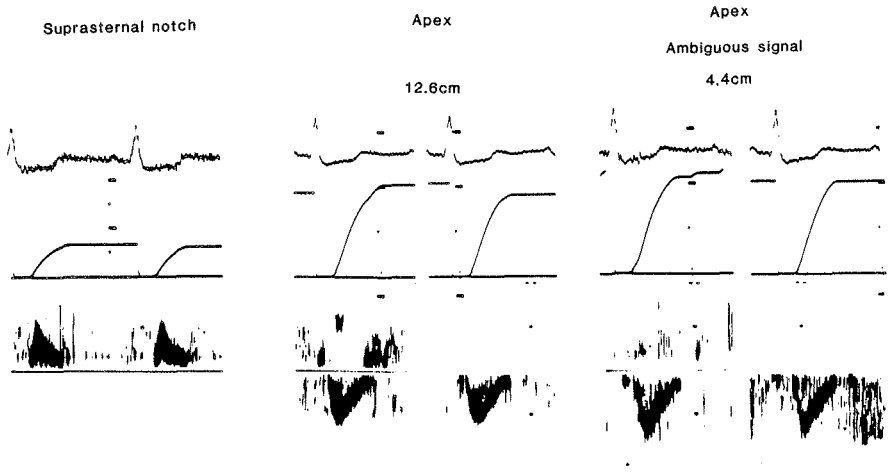

Fig. 7-4. To the left, the highest velocities recorded from the suprasternal notch in a patient are shown. In the middle, the velocities are recorded from the apex in the same patient. To the right, the ambiguous signal 8-cm closer to the transducer is recorded. The recordings from the apex are also shown, with rejection of positive velocities.

addition to directing the beam inside the aorta, it is also necessary to vary the depth of the sample volume to find the highest velocities. These velocities are usually found from 5 to 12 cm from the transducer. The sample volume should be moved to the aortic valve, to ensure that the whole length of the ascending aorta is scanned. The valve movements are heard as sharp clicks in the audio signal. In some patients, the distance from the transducer to the valve is so great that the depth limit of the pulsed mode is exceeded. In these patients, the recorded velocities should be compared to those obtained with CW Doppler to prevent underestimation of flow (Fig. 7-6). Because blood velocities in the

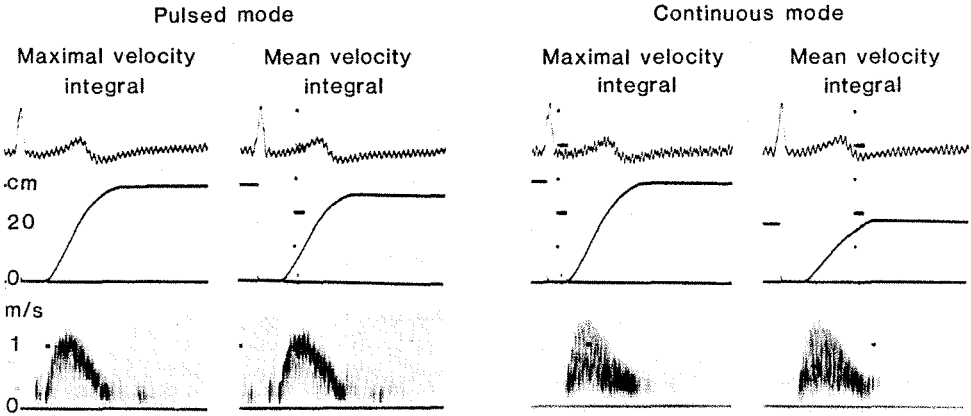

Fig. 7-5. The two panels to the left show the difference between the maximal and mean integral in a pulsed Doppler recording. To the right, the same difference is shown with a continuous wave recording. Because velocities all along the ultrasound beam are recorded in continuous mode, low-frequency components produce an underestimation of the mean velocity curve and integral size.

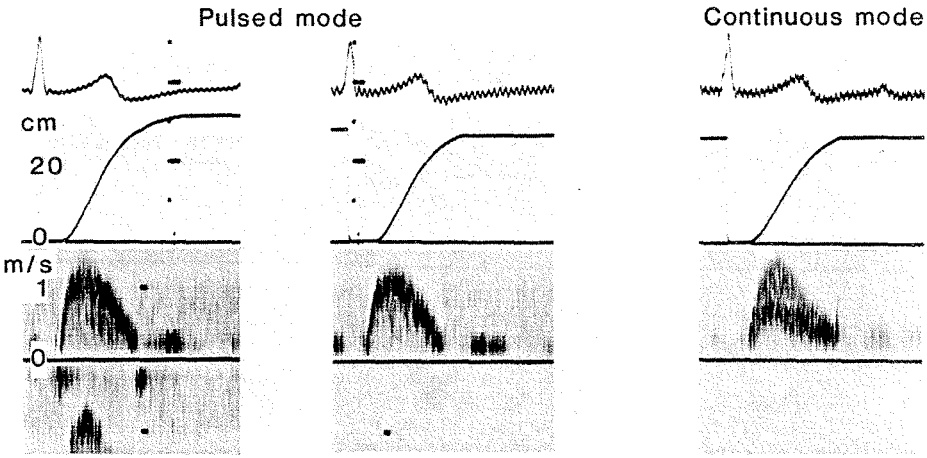

Fig. 7-6. To the left, the highest velocities obtained in pulsed mode are shown. Slight aliasing of the velocities occurs, as well as some negative velocities in early diastole. In the middle, the integrals are corrected by rejecting the negative velocities. To the right, the velocities are recorded with continuous wave Doppler to ensure that no underestimation of integral size occurs from the slight aliasing in the pulsed mode. The same procedure can be applied to ensure that the highest velocities are not missed when the whole length of the ascending aorta cannot be scanned in pulsed mode.

aorta are often higher than 1 m/s, the velocity limit of the pulsed Doppler may cause underestimation if these velocities are found deeper than 8 cm. The instrument we use* to this depth can measure velocities up to 1.7 m/s, which is sufficient in most patients. The velocity limit of the pulsed mode at various depths may be different in other instruments. If the velocity limit is exceeded, the ambiguous signal closer to the transducer can be recorded (see Fig. 7-4 and Section 3.3B). Alternatively, using a multifrequency Doppler instrument†, higher velocities can be recorded at a certain depth by changing from a 2-MHz to a 1-MHz transducer. The extra time spent in making a proper pulsed Doppler recording is usually worthwhile because the signal is more sharply defined than in the CW mode.

In Section 7.2, we suggest recording the highest velocities in the ascending aorta because they are assumed to represent the velocities at the aortic orifice, where the velocity profile is expected to be flat. It is difficult to exclude any variation of the velocity distribution even at this level, so a mean velocity estimation might in theory be the best choice. We prefer, however, to use the maximum velocity for the following reasons: The maximum velocity is less affected by errors in aiming, as shown in Figure 7-7. If the sample volume is placed partly outside the aorta, lower velocities are picked up, and the mean velocity is reduced. Another point is that the spectral mean velocity is not the same as the spatial mean velocity because we have no uniform insonification of the lumen. Sidelobes of the beam can also allow interference from flow outside the aorta and may change the spectral mean.

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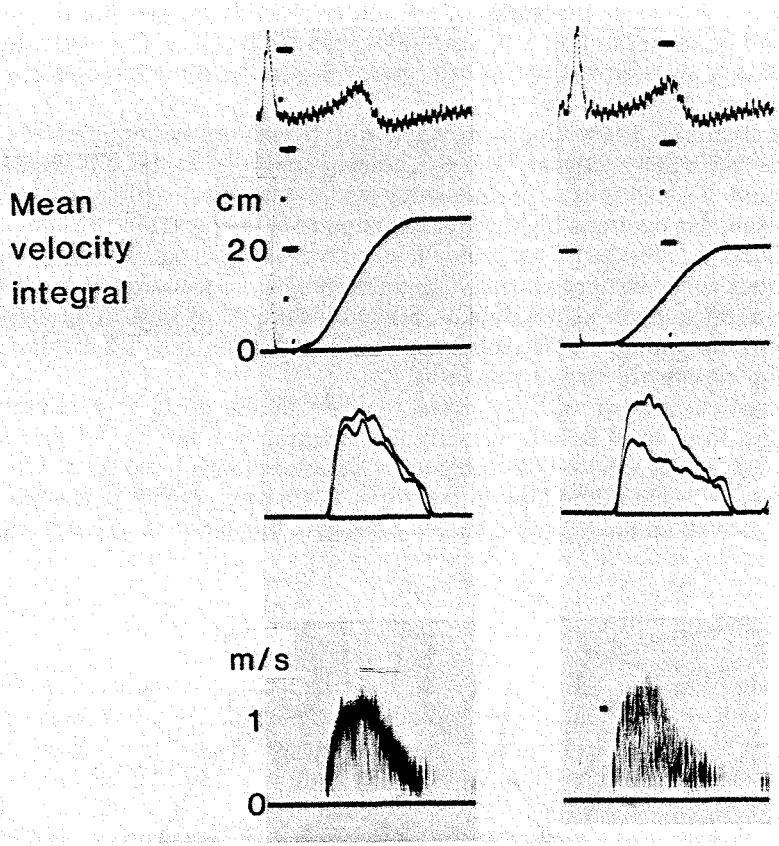

Fig. 7-7. To the left, a good Doppler signal in pulsed mode is shown. It is characterized by a narrow band of frequencies on the spectral display. The maximal and mean frequency curves are close. To the right, the sample volume is placed partly outside the aorta. The mean velocities are underestimated, with a corresponding decrease in integral size. As can be seen, the maximal velocity curve is not affected by this aiming error.

The criteria for an adequate Doppler signal, and the way to obtain it, are to some extent matters of experience; however, the following rules can help: First, the highest velocities are found. The direction of the beam is then adjusted to obtain a "clean" signal, which has a musical or whistling character. On a spectral display, this signal, is characterized by a narrow band of frequencies with few low ones. Such a signal is not always possible to obtain in the CW mode because of the recording of velocities all along the beam. If frequency estimators are used, the maximal and mean velocity curves in pulsed mode are close (Fig. 7-7).

If the sample volume is placed too close to the aortic wall, the movement of the aorta during ventricular contraction can give rise to velocities opposite to the direction of flow. This situation occurs chiefly at the start of systole and can influence the integral of both the maximum and mean velocity curves. At

and after closure of the aortic valve, eddies may form, and the reverse flow that can be recorded (Fig. 7-8) also influences the integral. These problems are solved to a great extent by rejecting velocities of the direction opposite to the main aortic flow (Fig. 7-9).

In patients in whom the transmission of ultrasound is impaired, the signal can be difficult to extract from the noise, especially in the CW mode. With too great a sensitivity, the frequency estimators start estimating on noise. Because noise contains high-frequency components, the velocities can be overestimated (Section 3.6). This problem is much less important with normal blood velocities than when recording high-velocity jets, such as found in aortic stenoses or ventricular septal defects. Some adjustment of sensitivity allows one to avoid the problem. This problem is also avoided if a spectral display is used and the curves are traced manually.

Reproducibility of velocity measurements in the aorta is satisfactory.[33-36] Changes from 10 to 20% in velocity and integral size can be detected.[33, 34]

As can be seen, both CW and pulsed Doppler modes have their virtues and drawbacks in recording flow in the aorta. We usually prefer to use the pulsed mode as well as the maximal velocity because this mode is less influenced by

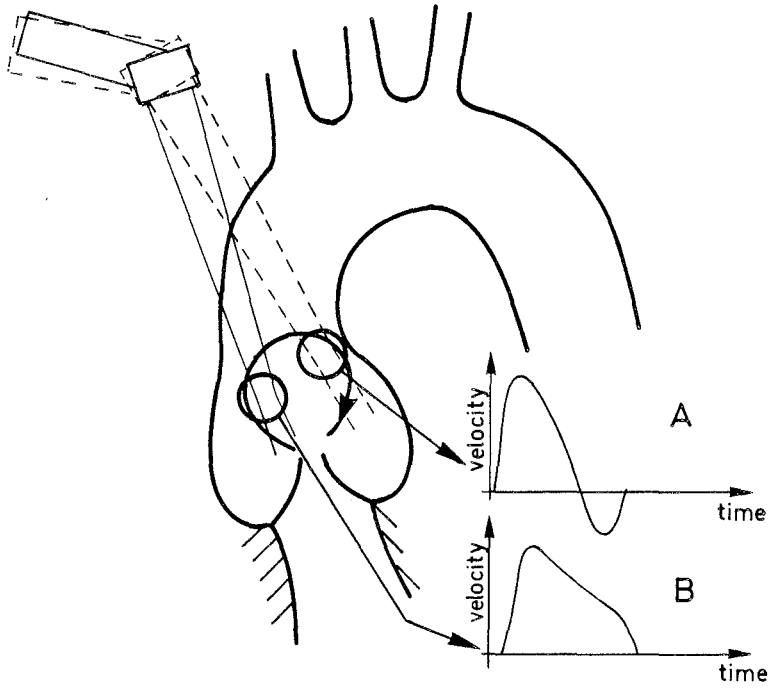

Fig. 7-8. At and shortly after closure of the aortic valves, eddies may form. Depending on the position of the sample volume, negative velocities can be recorded (A). The formation of eddies might in theory also give rise to positive velocities during the same phase of the cardiac cycle (B), but in practice this problem is minor. The reason is probably that the net flow in the aortic root during this phase has to be negative to close the aortic valve.

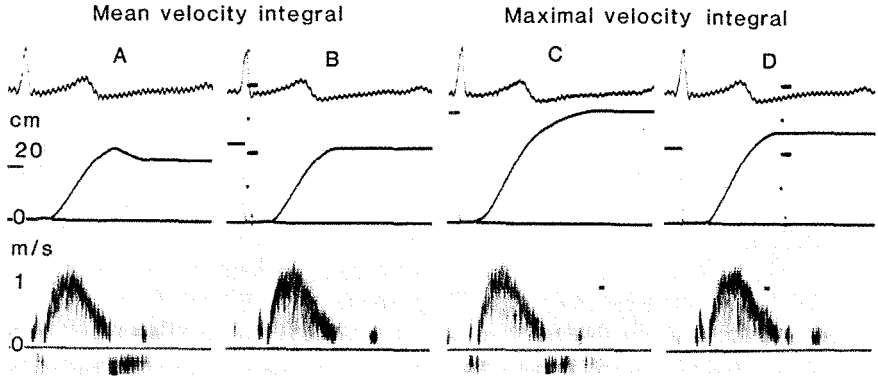

Fig. 7-9. A, the influence on the mean velocity integral size when reverse flow in early diastole is recorded. In B, these velocities are rejected. C and D show how the maximal velocity integral is influenced by the reverse flow and the effect of rejecting the negative velocities. The different effect of negative velocities on the mean and the maximal velocity integral is due to the estimated mean velocity being directional and the estimated maximal velocity being nondirectional.

recording problems. In high-output states, the CW mode might be the best choice. Both pulsed and CW Doppler modes can be used in the noninvasive estimation of cardiac output, provided care is taken to obtain a good signal.

7.4 DIAMETER MEASUREMENTS

A basic assumption in Doppler and echocardiographic estimation of cardiac output up to now has been that the velocity profile in the aorta is flat. The recorded velocities are then representative of the velocities over the whole cross-sectional area. With a negligible, thin boundary layer, the effective diameter is the same as the anatomical diameter. Stroke volume is then simply obtained by multiplying the velocity curve integral by the cross-sectional area measured at the same level. Although echocardiographic diameter measurements agree with perioperative and postmortem findings,[37,38] most workers think that measuring the diameter is the most difficult part of this procedure. Several reasons exist for this difficulty. The calculation of the cross-sectional area assumes a circular aorta, which may not be found in all patients. The area of the aorta also varies by about 10% during systole. Because of the squaring of the radius, errors in diameter measurements are doubled in area calculations. For example, if a diameter of 30 mm is measured to 32 mm, cardiac output will be overestimated by about 14%. Because 2 mm is typical of the width of the aortic wall echo, it is important to decide whether the diameter should be measured from the trailing edge or the leading edge of the anterior wall echo. Theoretically, measurements should be done with the leading echoes.[39] Most workers prefer to measure from the trailing edge of the anterior wall to the leading edge of the posterior wall, however. This method requires one to lower the gain sufficiently so that no "blooming" of echoes occurs. Otherwise, the diameter is underestimated. When the level of the aorta is chosen for diameter measurements, the aorta should be scanned in the short-

axis plane to obtain the largest, and therefore the true, diameter at this particular level.

A-mode, M-mode, and two-dimensional echocardiography can be used for diameter measurements. The last is apparently the best choice because the diameter can always be measured perpendicular to the long axis of the aorta. Such is not the case with A-mode and M-mode. When measurements are done superior to the valve, a fixed site is difficult to define without two-dimensional echocardiography, and reproducibility is thereby made more difficult.[40]

Which level should be chosen for diameter measurements? The diameter of the aortic root is used by some.[5, 10, 11, 41] An aortic root defined as the sinus of Valsalva is too large in diameter in our experience. This problem could be anticipated because the aortic valve leaflets in the open position occupy part of the cross-sectional area at this level. Others have used the smallest diameter superior to the sinus of Valsalva.[42] The discussion in Section 7.2 forms the basis for our choice of measurement level. Measuring the diameter of the aortic orifice was suggested by Ihlen and associates.[43] This diameter was found to be smaller than the diameter of the proximal and distal aortic root, and it did not change during systole. It was also more reproducible than the diameters of the aortic root. Others have reported similar findings.[37, 44]

The measurement of the diameter of the aortic orifice seems so far possible only with two-dimensional echocardiography. In the parasternal long-axis view, the echoes of the orifice appear as two bright spots at or just inferior to the level of attachment of the valve cusps. A-mode and M-mode echocardiography cannot be used because a beam direction for simultaneous recording of these two spots cannot usually be obtained.

7.5 CARDIAC OUTPUT

Most groups report a good correlation of cardiac output estimated by Doppler and echocardiography with that estimated by the thermodilution or Fick method.[5, 10-17, 31, 34, 36, 41, 42, 45-47] Reports do exist of poor correlation and problems of measuring velocities in the ascending aorta, however.[48,49] These conflicting reports are not readily explained. The differences may be due to a variety of reasons, such as selection of patients, method of diameter measurement, size of Doppler transducer, quality of the Doppler signals accepted, and sensitivity of the Doppler instrument. In our experience, considerable overestimation, up to 200%, of cardiac output occurred when the highest velocities in the ascending aorta were recorded and the smallest diameter superior to the sinus of Valsalva was measured.[43, 44] This overestimation was only slightly reduced when a mean frequency estimation was used. Only the diameter of the orifice was useful. A good correlation to the invasively measured cardiac output was then obtained. This finding fits well with the assumption of a flat velocity profile at this level in most patients and a nonflat profile in the ascending aorta in a large group of patients.

By choosing the aortic orifice for diameter measurements, the following advantages are gained:

1. The site for measurements is sharply defined.

2. The smallest diameter is measured.
3. The highest velocities are found at the same level or in the jet downstream.
4. A flat velocity profile is more likely at this level than at any other level in the aorta.

The risk of overestimating cardiac output can thus be minimized.

We hope that in the future it will be possible to standardize measurement procedures and to improve the accuracy of the method.

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5. Influence of the geometry of the ascending aorta upon the velocity profile

T. SKJAERPE

INTRODUCTION

When the cardiac output (CO) is estimated noninvasively by echo and Doppler measurements in the ascending aorta, the velocity profile is generally assumed to be flat [1–4]. The recorded velocities will then represent the spatial mean velocity, and the stroke volume is obtained as the product of the integral of the velocity curve and the cross sectional area of the aorta at the same level.

Our first experience with this method did not fit the assumtion of a flat velocity profile. In some patients severe overestimation of CO, up to more than 200%, were made compared to thermodilution measurements. We therefore decided to have a closer look upon geometrical features of the ascending aorta that might influence the velocity profile.

PROCEDURE

Theoretical considerations

When the outlet from a flow channel with a large diameter is through a channel with a much smaller diameter (Figure 1), the velocity profile in the entrance region will be flat with a very thin boundary layer [5]. However, if the entrance region is curved, as the aorta, flow theory says that the profile is skewed with the highest velocities at the inner curvature during the first part of the bend, with a redistribution of the highest velocities to the outer curvature after a distance of 1–2 radii from the inlet [6].

Sometimes a vena contracta may form in the proximal part of the entrance region, where the effective flow area is smaller than the physical area (Figure 2A).

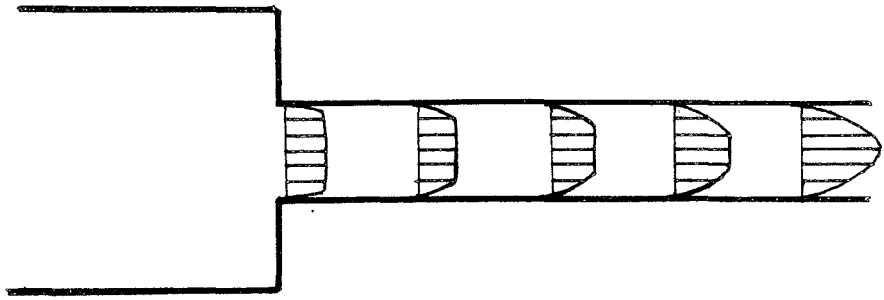

Figure 1. Flow pattern in a straight channel branching of from a much larger channel. In the entrance region the velocity profile is flat. Downstream, viscous friction gradually changes the shape of the profile into a parabola.

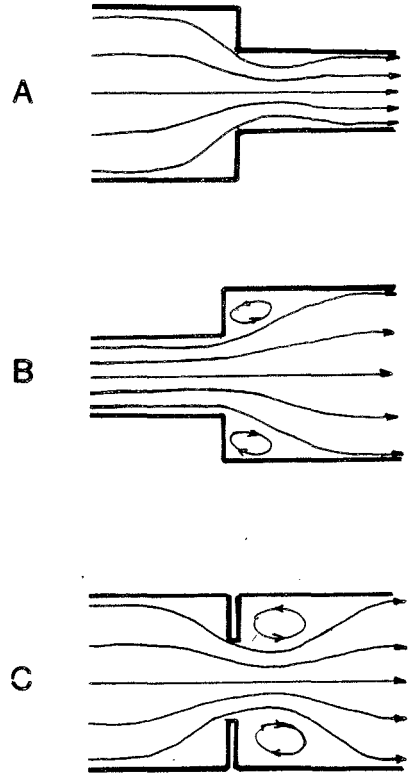

Figure 2. (A) In the entrance region a vena contracta may form, reducing the effective flow area. (B) The flow pattern when there is a sudden expansion of a flow channel. (C) The flow pattern in a channel which at one level is partly obstructed.

When there is a sudden expansion of a flow channel, a central, expanding core of flow is found where the velocity is higher than the cross sectional mean velocity (Figure 2B). Eddies are surrounding the core, giving rise to negative velocities close to the walls. Further downstream the expanding core will fill the channel, with a corresponding decrease in velocity.

If the models in Figure 2A and 2B are combined to form a channel which at one level is partly obstructed, the flow pattern will be as indicated in Figure 2C. Just proximal to the obstruction the velocity will increase, and the highest velocity is found at the obstruction or in the first part of the downstream flow, depending on if, or to what extent a vena contracta is formed. The important point to realize is that, when the highest velocities are measured, only the area of the obstruction can be used to calculate a sensible estimate of volume flow.

Patients

Fifty-five patients were selected from out- and inpatients referred for an echocardiographic study. The only criterion for inclusion was a normal aortic valve as judged by echocardiographic and Doppler examinations. There were 32 men and 23 women, aged 3 to 74 years (mean 35.8 years). Twelve patients had simple shunts, 8 had ischemic heart disease, 10 had mitral and pulmonary valve disease, and in 25 no cardiac disease were found (most of them had innocent murmurs).

Diameter measurements

The ascending aorta was visualized in the parasternal long axis view with two-dimensional echocardiography, and diameters were measured at four different levels (Figure 3). An ATL mark III (Advanced Technology Laboratories) with a 3 mHz mechanical sector tranducer, or an Irex III B (Irex Corporation) with a 2.5 mHz phased array transducer was used. The diameters were measured in systole using leading edge measurements.

Velocity measurements

To evaluate the importance of the curved course of the ascending aorta on the velocity profile, it was attempted to record flow velocities at the inner curvature, centerline velocities, and velocities at the outer curvature in patients where the diameter measurements indicated a fairly even calibred aorta (diameters at levels 1, 3 and 4 differing no more than 3 mm), and where the ascending aorta could be visualized from the suprasternal notch. The sample volume was placed at a level where the angle between the

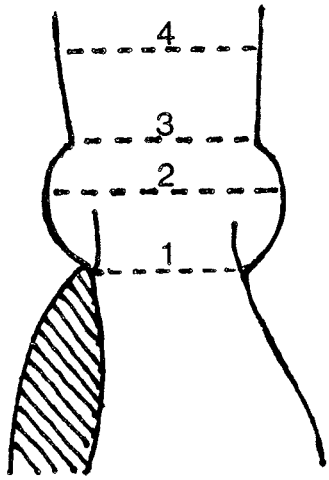

Figure 3. The levels chosen for diameter measurements. Level 1: Just below the aortic valve (the annulus). Level 2: The largest diameter of the Sinus of Valsalva. Level 3: Just distal to the sinus of Valsalva. Level 3: 2 cm distal to the sinus of Valsalva.

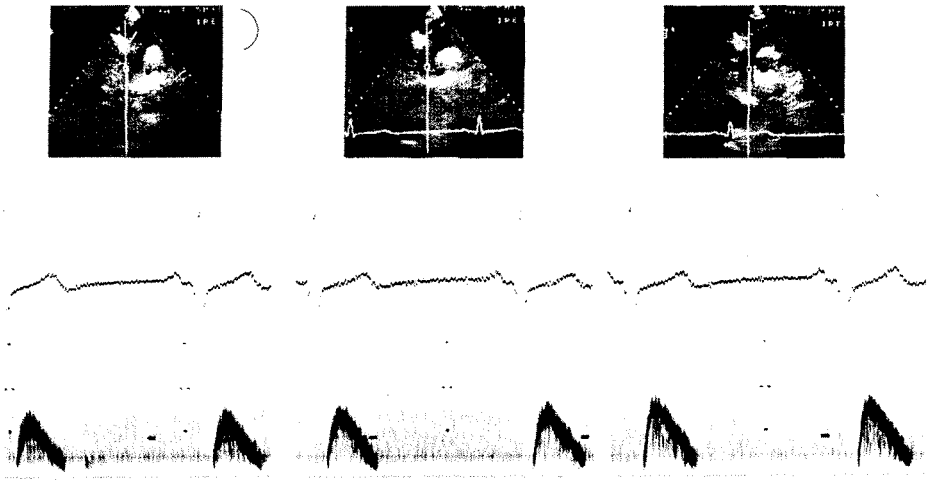

Figure 4. Velocity measurements in the ascending aorta. Left: Velocity recording at the outer curvature. Middle: Centerline velocity recording. Right: Velocity recording at the inner curvature.

ultrasound beam and the direction of flow was close to zero (Figure 4). The Irex III B instrument was used which allows pulsed and continuous wave Doppler recordings to be made simultaneously with imaging.

RESULTS

The smallest diameter was in all patients found at level 1 (mean 2.09 cm, $\pm$0.36 cm ($\pm$1 SD)). The next smallest diameter, found at level 3, was 0 to 18 mm larger (mean 2.61 cm, $\pm$0.58 cm ($\pm$1 SD)), giving up to 3 times larger cross sectional area than the area at level 1. The largest diameter was found at level 2 (mean 3.02 cm, $\pm$0.69 cm ($\pm$1 SD)), while the diameter at level 4 was somewhat larger than the diameter at level 3 (mean 2.76 cm, $\pm$0.65 cm ($\pm$1 SD)). Table I shows per cent increase in diameters from level 1 to levels 2, 3 and 4.

The criteria for making velocity measurements in the ascending aorta were met in 6 patients. The highest velocity was in all recorded at the inner curvature, and the integral of the velocity curve was 8–22% higher than the integral of the centerline velocity curve. The lowest velocity was measured at the outer curvature.

Table I. Per cent increase in diameters at levels 2, 3, and 4 compared to level 1.

	Level 2	Level 3	Level 4
Range	14-86 %	0-75 %	10-88 %
Mean	45 %	25 %	34 %
	$p<0.001$	$p<0.001$	$p<0.001$

DISCUSSION

Most experimental studies on the velocity profile in the ascending aorta conclude on a 'flat, but skewed' profile [6–8]. This means that the velocities increase steadily from one side of the aorta to the other. This was also found in 6 patients in this study. Seed and Wood [7] found that the centerline velocity was representative for the cross sectional mean velocity. In our patients, calculated CO was from just slightly higher (about 8% as in the paitent shown in Figure 4), and up to 22% higher when the highest velocities were integrated compared to when centerline velocities were integrated. Since the degree of skewing is different in different patients, and experimental findings indicate a variable skew when the contractility of the heart

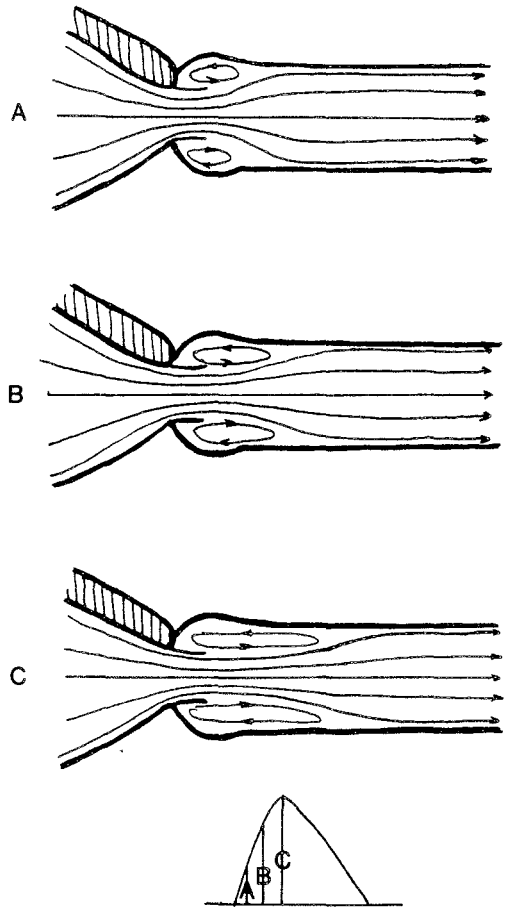

Figure 5. Expected flow patterns in the aortic root when there is a dilation of the aorta compared to the annulus. The patterns are indicated for three different parts of systole.

changes [8], this overestimation is impossible to correct for, except to use a combined echo/Doppler instrument and record centerline velocities. However, in our experience it was very difficult to adequately visualize the ascending aorta in adults.

Since the presence of a skewed profile explains only a fraction of the overestimation of CO we made when we first tried the method, we had to seek other explanations.

According to our diameter measurements, which showed that the smallest diameter was found at the aortic annulus and the largest at the Sius of Valsalva, there is in many patients a sudden expansion of the cross sectional flow area distal to the annulus. Therefore, flow patterns as indicated in Figure 2B and 2C is expected in these patients. The downstream extension

of the central core of flow depends on the velocity of flow, and, accordingly, varies in pulsatile flow. In Figure 5 the expected flow patterns in a simplified (straight) model of the ascending aorta is shown. In a curved model the flow pattern is complicated by the skewing of the velocity profile. The highest velocities are found in the proximal aortic root, and will be similar to the velocities at the annulus. Theoretically then, if the highest velocities in the ascending aorta are recorded, only the cross sectional area of the annulus can be expected to give a good estimate of CO. This suggestion is supported by the findings of Ihlen [9]. The overestimation we experienced in estimating CO noninvasively, can now be explained. Since we originally used the diameter at level 3 to calculate CO, the overestimation should be proportional to the increase in cross sectional flow area from level 1 to level 3, which in this series was up to 206%.

One should note that these findings do not preclude CO estimations by velocity and diameter recordings distal to the aortic annulus. However, it then appears necessary to make the measurements at a level distal to the jet-like flow in the aortic root.

It can be concluded that the ascending aorta exhibits geometrical features that will have significant influence on the velocity profile. The velocity profile at all levels in the ascending aorta can therefore not be expected to be flat in an unselected group of patients.

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EVALUATION OF THE OPTIMAL LEVEL FOR NONINVASIVE MEASUREMENTS OF CARDIACOUTPUT IN THE LEFT VENTRICULAR OUTFLOW TRACT AND THE ASCENDING AORTA. COMPARISON WITH TWO INVASIVE METHODS.

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Key words:
Doppler Ultrasound, Fick, Thermodilution.

Abstract.

In a group of 40 patients (group 1) undergoing right heart catheterization, cardiac output (CO) was measured by the Fick or the thermodilution method; by both in 16 patients. With two-dimensional echocardiography diameters were measured at the aortic annulus (level 1), just distal to the Sinus of Valsalva (level 2), and 2 cm distal to the Sinus of Valsalva (level 3). Max and mean velocities were recorded with Doppler from the suprasternal notch at level 2, simultaneously with invasive measurements. The velocity curve integrals and diameters at levels 2 and 3 significantly overestimated CO. The max velocity integral combined with the diameter at level 1 also produced overestimations in some patients, while the mean velocity integral gave a good correlations to invasive measurements.

In a second patient group consisting of 18 patients (group 2), max velocity measurements were also made at level 1. CO calculated from velocity and diameter recordings at level 1, correlated well to invasive measurements. CO calculated from max velocity integrals at level 2 and diameters at level 1 tended to be lower than in group 1, being closer to the results obtained with the mean velocity integral in that group. The differences may be explained by the groups not being equal with regard to the angle between the aortic root and the ultrasound beam, and that the aortic valve in some patients may be restrictive.

Our results indicate that severe overestimation of CO may occur when both velocity and diameter measurements are made in the ascending aorta. Diameter and velocity recordings at the aortic annulus provided the most accurate estimates. The correlation between the invasive methods indicate that they are only moderately more accurate than the best noninvasive method.

INTRODUCTION

Despite numerous publications on the noninvasive quantitation of cardiac output with Doppler and echocardiography in the left ventricular outflow tract (LVOT) and the ascending aorta, there is still no general agreement on how to do the measurements This is largely because different sites for diameter and velocity measurements have been used, and sometimes the exact level for the measurements are not described. There seems, however, to be a tendency to record the highest velocities in the ascending aorta (1,2), in which case it is reasonable to measure the smallest diameter of the ascending aorta, which is regularely found just distal to the Sinus of Valsalva (1,3). Another trend is to make centerline velocity recordings in the ascending aorta and measure the diameter at the same level (4,5). On the other hand, Ihlen et al. intentionally measured flow area and flow velocities at two different levels, the aortic orifice and the proximal ascending aorta, respectively (6). Another major trend is to record flow velocities in the distal LVOT from the apex, and measure the diameter at the annulus (7,8).

Our initial experience with the method was that significant overestimation of cardiac output occured when flow area and flow velocity was measured in the ascending aorta, distal to the annulus (9). We therefore wanted to evaluate the applicability of the method at different levels in the ascending aorta and the LVOT.

METHODS.

Patients.

The first study group (group 1) consisted of 40 patients undergoing right heart catheterization. There were 25 men and 15 women. Age was 21 to 80 years (mean 57 years). Because Doppler measurements from the apex was not done when we began the study, a second group (group 2) was also studied. In this group 18 patients were included, 12 men and 6 women. Age was 22 to 69 years (mean 57 years). In both groups informed consent was obtained from all. No patient with aortic valve disease, subvalvular or supravalvular stenosis, or patients with shunts were included, otherwise there were no aexclusion criteria. Eighteen patients had ischemic heart disease, 3 congestive cardiomyopathia, 17 mitral valve disease. Eleven underwent hemodynamic evaluation of various pacemaker modalities, 5 had chronic lung disease with right ventricular failure, 2 patients had constrictive pericarditis, and 2 patients had no cardiac disease.

Ultrasonic recordings.

Group 1.

The diameter measurements were made with an ATL (Advanced Technology Laboratories) or an Irex III B (Irex corporation) two-dimensional echocardiographic instrument. The LVOT and the ascending aorta were visualized in the left parasternal long axis view. Diameters were measured just below the insertion of the aortic valve leaflets (annulus, level 1, representing the calibre of the distal LVOT), at the narrowest level just distal to the Sinus of Valsalva (level 2), and about 2 cm distal to the Sinus of Valsalva (level 3). For each level the image was

adjusted to optimally visualize that particular level. The image was frozen in systole, and the diameters were measured on printouts. Both trailing to leading edge and leading to leading edge measurements were made at each level. However, at any level the leading to leading edge measurement was not allowed to exceed the trailing to leading edge diameter by more than 2 mm.

Doppler measurements were made from the suprasternal notch with a 2 mHz, angled Doppler transducer. Two Doppler instruments were used (Pedof, Vingmed A/S and prototype from the same company designed for cardiac output measurements), both displaying maximal and mean velocities by analog frequency estimation. Automatic integration of any of the curves is possible with both instruments (figure 1).

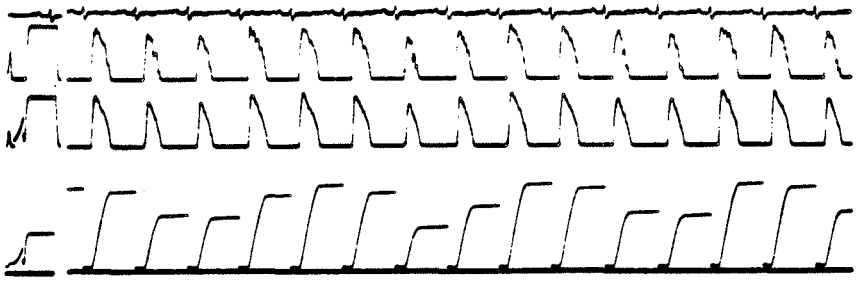

Figure 1.

Doppler recording of velocities in the aortic root, made from the suprasternal notch.

From top to bottom: ECG, mean velocity curve, maximal velocity curve, maximal velocity integral (mean velocity integral can be selected by a hardware switch). To the left the calibration signals are shown. The velocity calibration is 1 m/s, and the integral calibration is 10 cm. The respiratory variations in integral size is due to a constrictive pericarditis in this patient.

In pulsed mode the sample volume was moved down the ascending aorta until the opening click of the aortic valve was recorded. The sample volume was then moved up about 2 cm, thus being positioned close to level 2. The aortic cross section was scanned with the sample volume until the highest velocities at this level were recorded. Usually, but not always, these were the highest velocities that could be recorded in the ascending aorta.

Group 2.

The same instruments were used. In this group only the diameter at level 1 was recorded. The max velocities in the ascending aorta were recorded as described above. In addition, velocity measurements from the apex were also made. The sample volume was moved out the LVOT, and the recordings made when the closure click of the aortic valve was recorded. This recording position was assumed to correspond to level 1. In those patients where the diameter measurements were made with the Irex III B, the position was checked by simultaneous 2-D echo and Doppler recordings from the apex. The procedure was repeated from positions both lateral and medial to the apex to find the smallest possible angle to the direction of flow.

In both groups the Doppler recordings were made simultaneously with invasive cardiac output measurements. Averaging was made over 10 consecutive beats. When using Fick's method, the beats were selected just after collecting air in the Douglas bag. When thermodilution was used, beats recorded during the middle part of the thermodilution procedure were selected. Care was taken not to integrate velocities recorded before or after the ejection period.

Invasive measurements.

The thermodilution measurements were made with a Swan Gantz 7F catheter and a Elcath computer. Saline was cooled to 0 degree Celsius, and 10 ml. was injected rapidly into the right atrium. The thermistor was placed in the distal part of the main pulmonary artery or, more often, in the proximal part of the right or left branch, usually the right. Seven injections were made. The first, and the injections giving the lowest and the highest cardiac output were rejected. Averaging of the other four was made.

The Fick method was performed by collecting expirated air from the patient during 3 minutes and sampling blood from the pulmonary artery during the second minute.

In group 1, if the patient agreed to prolong the procedure, and the clinical condition of the patient did not indicate that the procedure should be as short as possible, both thermodilution and Fick's method were used in close succession (16 patients). Thermodilution was used as the only invasive method in 14 patients, and Fick in 10 patients.

In group 2 patients only one invasive method was used, Fick's method in 6, and thermodilution in 12 patients.

RESULTS.

Group 1.

The results are presented in table 1. The results obtained when the diameter at level 3 was combined with velocity recordings at level 2 are not shown since bisarre overestimations were made. Significant overestimation occured in some patients also when combining diameter and velocity measurements made at the narrowest part of the ascending aorta, level 2. Only the results calculated by the trailing to leading edge diameters and mean velocity integrals are shown since the larger leading to leading edge diamters and max velocity integrals resulted in even higher overestimations. When the velocity measurements at level 2 were combined with the diameter measurements at level 1, the aortic annulus, the correlation became significantly better. In a few patients important overestimations still occured when leading to leading edge diameters and max velocity integrals were used. When the mean velocity integrals were used, the correlations improved further. There was some tendency to underestimate the invasive results with the trailing to leading edge diameter, while the standard errors were slightly better than with the leading to leading edge diameters. The differences were, however, small.

When comparing the invasive methods, the standard error of the estimates to some extent depended on which method was used as the independent variable (table 1). The r-value of 0.92 was not significantly higher than that of 0.83 and 0.86 ($p>0.13$ and $p>0.21$).

In figure 2 the Fick results are plotted against the thermodilution results.

Table 1.

Echo/ Doppler	Inv. method	r value	SEE (l/min)	Regression equation	Mean % error	Range (%)	
Echo: Level 2 / Doppler: Level 2							
t-l/Vmean	Fick	0.50	1.62	y=0.76x+3.03	+44.5	+157.1	-15.2
t-l/Vmean	Thermo	0.65	1.44	y=0.61x+3.05	+25.6	+117.9	-17.6
Echo: Level 1, Doppler: level 2							
t-l/Vmax	Fick	0.80	0.64	y=0.68x+1.28	-2.8	+71.2	-28.8
l-l/Vmax	Fick	0.79	0.77	y=0.82x+1.50	+18.2	+103.7	-15.2
t-l/Vmax	Thermo	0.81	0.71	y=0.51x+1.78	-11.3	+29.9	-45.6
l-l/Vmax	Thermo	0.82	0.84	y=0.61x+2.11	+5.6	+54.5	-35.5
t-l/Vmean	Fick	0.86	0.53	y=0.73x+0.64	-12.6	+24.5	-34.2
l-l/Vmean	Fick	0.86	0.55	y=0.75x+1.11	+0.6	+48.1	-21.7
t-l/Vmean	Thermo	0.86	0.61	y=0.52x+1.38	-19.3	+11.9	-43.7
l-l/Vmean	Thermo	0.83	0.73	y=0.54x+1.83	-7.0	+40.9	-35.3
Invasive methods							
Thermo vs Fick		0.92	0.80	y=1.47x-1.57	+14.6	+37.5	-15.4
Fick vs Thermo		0.92	0.80	y=0.58x+1.68	-10.9	+18.2	-27.3

t-l: Trailing to leading edge diameter measurements. l-l: Leading to leading edge diameter measurements. Vmean: Mean velocity estimation. Vmax: Maximal velocity estimation.

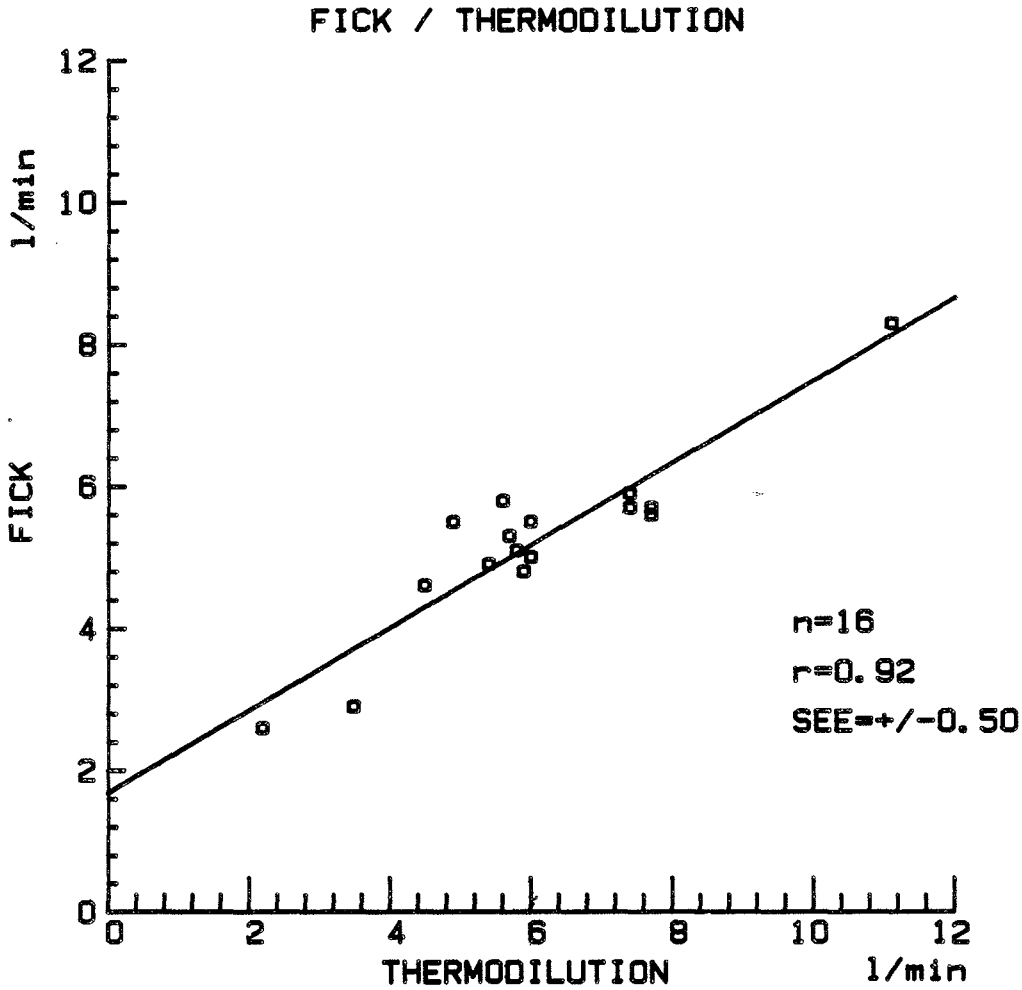

Figure 2.

Cardiac output measurements by the Fick method compared to thermodilution measurements in 16 group 1 patients.

Group 2.

Table 2 shows the correlations obtained. When the max velocity integrals at level 2 were combined with the diameter at level 1, the correlations to invasive measurements were somewhat different from those obtained by the same method in group 1. In fact, the correlations were quite similar to those obtained in group 1 when the mean velocity integrals were used. When both velocities and diameters were measured at the aortic annulus, the correlations remained in the same range.

In group 2 patients, measurements based on trailing to leading edge diameter measurements tended to give slightly smaller standard errors than measurements based on leading to leading edge diameters, while the latter gave regression lines closer to the line of identity, with a lower tendency to underestimate the invasive results.

Table 2.

Echo/ Doppler	r value	SEE (l/min)	Regression equation	Mean % error	Range (%)	
Echo: Level 1 / Doppler: Level 2						
t-l/Vmax	0.86	0.67	y=0.70x+0.38	-21.1	+17.5	-45.2
l-l/Vmax	0.83	0.83	y=0.77x+0.57	-10.0	+37.5	-38.7
Echo: Level 1 / Doppler: Level 1						
t-l/Vmax	0.85	0.74	y=0.74x+0.75	-10.2	+20.8	-31.6
l-l/Vmax	0.86	0.80	y=0.81x+1.0	+ 2.0	+37.5	-19.0

Abrevations as in table 1.

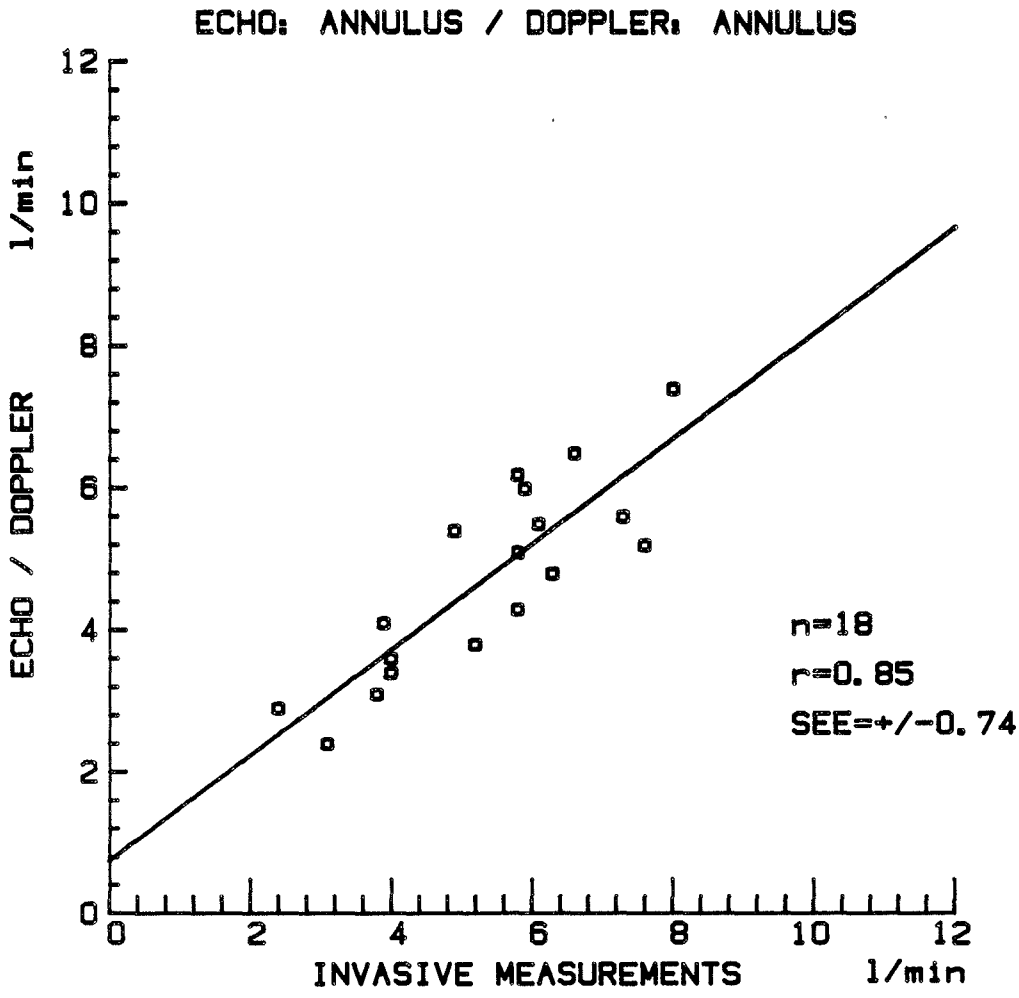

Figure 3.

Noninvasive cardiac output measurements based on velocity and diameter recordings at the aortic annulus, compared to invasive measurements (group 2). The maximal velocity curve is integrated, trailing to leading edge diameters are used.

When the highest velocites in the ascending aorta were not found at level 2, they were in 2 patients found 1-2 cm distally, being closer to level 3. The differences were fairly small, less than 20%. In two group 2 patients, where significant underestimation resulted when recording velocities from the suprasternal notch (-45.2 and -35.0%), clearly higher velocities were recorded from the right parasternal border, 1-2 cm downstream to the valve, reducing the underestimations to -18.3 and -8.2% (trailing to leading edge diameters used).

DISCUSSION.

Velocity and diameter measurement distal to the annulus.

Similar to the results of Ihlen and coworkers (6) and Rein et al. (10), significant overestimation of cardiac output occured in some patients when both velocities and diameters were measured in the ascending aorta, distal to the annulus. There may be several reasons for this. In the current study the highest velocities, or rather the velocity curve giving the highest integral were recorded. From fluid mechanics and experimental and clinical studies (11,12) it is known that the velocity profile in a curved vessel will develop a skew with the highest velocities at the inner curvature during the first part of the bend. The centerline velocity is expected to be more representative for the cross sectional mean velocity (11). However, in an earlier report where we were able to quantitate the effect of the skew in a small group of patients, only a fraction of the overestimation of cardiac output could be explained (3). Jenni et al. (12) found that an overestimation of 14-45% resulted when the highest velocities were integrated compared to the cross sectional mean, while Fisher et al. found that the effect of temporal skewing was negligible in an study on open chest dogs (13). To our knowledge, no experimental or clinical studies have demonstrated a degree of skewing that explains the overestimations made in the present series.

Even when the velocity profile is completely flat, the outer border of the spectral curve may overestimate the velocities. This is so because spectral analysis has a limit on frequency resolution. One frequency componenent is presented as a range of velocities, both higher and lower than the true velocity. With the instruments we have used, the overstimations may be up to 5-10%, theoretically (information obtained from the designers of the instrument). We did not use modal velocities, which would eliminate this overestimation. But there is little reason to believe that they would have produced lower estimates than those calculated by the mean velocities, which also produced quite significant overestimations (table 1).

The most likely explanation is the variation of the caliber of the LVOT and the ascending aorta (3). In some patients there is a sudden expansion of the cross sectional flow area distal to the aortic annulus. In such cases the velocity profile cannot be expected to be flat at all levels in the aorta, and during the whole ejection phase. When blood is ejected out the narrow annulus, it will take some time for the flow core to expand and fill the wider aorta. Until that happens, the velocities within the core must be higher than the spatial mean in the ascending aorta, being more representative for the spatial mean of the LVOT. This explains why the diameter of the aortic annulus gave better estimates of cardiac output, than when one of the diameters of the ascending aorta was used. Outside the expanding core of flow, deadwater zones are formed. According to fluid mechanics, in the deceleration part of the ejection period, these eddies expands downstream, heavily disturbing the velocity profile (14).

This explanation to our results fits the fact that the highest overestimations were made in patients whose aortas were most dilated compared to the annulus. Even if our results are in conflict with some earlier reports (1,2,4,5), they are in accordance with flow theory. One should note, however, that the apparently conflicting results may also be explained by the patient groups being different. Our study groups

included many old patients. According to preliminary analysis of a new series of diameter measurements, the dilatation of the ascending aorta relative to the annulus seems to increase with age. In a younger patient group, especially in children, we might have obtained results more in agreement with other publications.

Our results does indicate, however, that in an unselected group of patients it is not possible to estimate cardiac output reliably by measuring velocities and flow area in the proximal ascending aorta, distal to the annulus.

The velocities at level 3 were not routinely recorded, and we can therefore not exclude that this level may have produced better results than level 2. This is unlikely, however. Since the diameter at level 3 almost always was larger than the diameter at level 2, a sharp drop in blood velocity from level 2 to level 3 would be required to avoid the overestimations. Such sharp drops in velocity were very rarely recorded when the sample volume was moved down the ascending aorta to localize the aortic valve and level 2. In some patients where the velocities at level 3 were recorded, the overestimations were up to about 100%.

Measurements of velocities in the aortic root and diameters at the annulus.

In accordance with the studies of Ihlen et al. (6) and Rein et al. (10), the correlation to invasive measurements improved when we used the diameter at the aortic annulus together with the velocities in the aortic root. But neither this approach was in our experience completely devoid of problems.

When the max velocity integral at level 2 was combined with the diameter at the annulus, the results in group 2 tended to be lower than those in group 1. There was also a difference between the Fick and the thermodilution subgroups in group 1. The reason is probably that the groups were different with regard to some factors that influences the recorded velocities in the aortic root.

It is sometimes observed that there is an increase in velocities from the distal LVOT and up to the tips of the aortic cusps, indicating a smaller aortic valve area than annulus area. These higher velocities may prevail for some distance downstream, and with the added effect of a skewed velocity profile, cardiac output will in such patients be overestimated. This may be the explanation to the overestimations that occurred in some group 1 patients. On the other hand, the aortic root generally is angled in relation to the ultrasound beam when the transducer is placed in the suprasternal notch. An angle of 30° between the plane of the cusps and the long axis of the patient is common (15). If this angle is representative for the direction of flow in the aortic root, the corresponding velocity underestimation is 16%. The skewing of the velocity profile may compensate this underestimation, but in patients with horizontal hearts the angle may become large enough to cause a definite underestimation of cardiac output. This can be examplified by two patients in group 2 where underestimations of 45.2 and 35.0% were made (trailing to leading edge diameters, Vmax integrated). Clearly higher velocities were recorded from the right parasternal position, reducing the underestimations to -18.3 -8.2). The explanation is most likely a reduction of the angle of incidence. Different combinations of "vertical and "horizontal" aortic roots, and restrictive and nonrestrictive aortic valves could then produce both over- and underestimations of cardiac output. The wide range of errors we experienced, when both groups are considered, can then be readily explained. Althoug relatively few noninvasive estimates deviated more than 40 % from the invasive measurements (4 when using leading/leading edge diameters, 3 when using trailing/leading edge diameters), our results do indicate that also this method must be used with considerable care.

In the ascending aorta small movements of the transducer could result in quite different velocity curves being recorded, probably because of the formation of eddies (fig. 4). In some patients, considerable efforts was necessary to locate and keep the sought-after velocities at level 2.

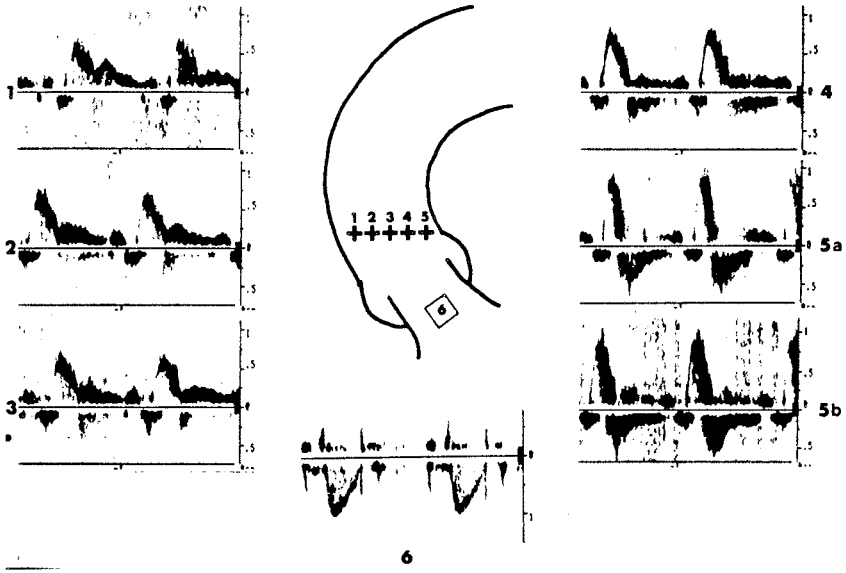

Figure 4.

Velocity recordings, displayed by spectral analysis, in the aortic root and at the annulus in a patient where the ascending aorta is dilated as compared to the annulus. In the aortic root, the shape of the velocity curves changes when the sample volume is moved from one side to the other, probably because of the formation of eddies. This was not the case at the annulus. Note also the smaller spectral broadening of the curve recorded at the annulus. Despite an almost two-fold increase in flow area from the annulus and up to the sinotubular junction, the integrals of the curves recorded at the annulus and at position 4 were the same.

Measurements of diameters and velocities at the annulus.

When the results obtained with diameter and velocity recordings at the annulus are compared to those where the velocities at level 2 were used, one should be aware that the first method were used in fewer patients (group 2 only), which may account for the lesser spread of results. However, our results are in agreement with those of other groups using similar methods (7,8), and this approach seem to give the least conflicting results. One reason is that the apical approach makes it

easier to adjust the position of the transducer to reduce the angle of incidence. One should be aware, however, that limited ultrasonic access may result in a fairly large angle even from this position. This problem is not easily solved, but if a situation like that is suspected, it may be wise to do recordings from other positions. If the velocities recorded at the tip of the aortic valve from the suprasternal notch or the right parasternal border are clearly higher than the velocities recorded at the same level from the apex, underestimation of LVOT velocities is likely.

An important experience was that stable velocity recordings were much easier to obtain in the distal LVOT from the apical position, than in the ascending aorta from the suprasternal notch. This is as expected from flow theory. The LVOT is a converging flow channel, and the blood is subjected to convective acceleration even in the late part of the systole, stabilizing and flattening the velocity profile. The chief problem we experienced when recording velocities in the distal LVOT, was that quite strong diastolic signals from the moving anterior mitral valve leaflet were picked up, possibly also signals from septal movements. Even the closure movement of the aortic valve could be quite disturbing. Because of the high intensity of these signals, they were difficult to remove by gain adjustments. A fairly high setting of the high pass filter was often necessary to prevent automatic integration of the signals. Since a high filter setting decreases the integral size, it would probably have been better to do a strip chart recording of the spectral velocity curves, and manually trace the curves. That might have reduced the underestimations made in some patients. It is difficult from our results to evaluate wether trailing to leading, or leading to leading edge diameter measurements should be preferred. The theoretical correctness of doing leading to leading edge measurements may be reflected by the lower tendency to underestimate cardiac output when the diameters were measured that way, while the difficulty in defining the leading edge of the anterior echo may be reflected by the larger standard error.

Averaging the results from several sampling sites seem to increase the correlation to invasive measurements according to Labovitz et al. (16). However, since cardiac output estimates in the ascending aorta may be so grossly erroneous, it is difficult to accept that such result should be used to correct possible errors at more optimal sampling sites.

Even if the lack of significantly higher correlation to some extent is explained by the low number of patients studied, it is interesting to note that the two invasive methods did not correlate much better than the best noninvasive method and either of the invasive methods, indicating that only moderately higher accuracy can be expected from invasive than from noninvasive measurements. Differences of up to 40% were seen between the invasive methods, and between the invasive and the best noninvasive method (apical approach) suggesting that errors of about +/- 20% may occur with any method.

In general, our experience was that noninvasive measurements of cardiac output in unselected patients demand skilled operators, especially when doing Doppler measurements from the suprasternal notch. Because of potential pitfalls, also the apical approch require experienced personel.

In conclusion, the present study indicate that the aortic annulus is the optimal level for diameter and velocity measurements when cardiac output is estimated noninvasively in the outflow system of the left heart. Compared to invasive measurements, the accuracy seems high enough to justify the incorporation of the method in the clinical routine.

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Noninvasive estimation of valve area in patients with aortic stenosis by Doppler ultrasound and two-dimensional echocardiography

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Noninvasive estimation of valve area in patients with aortic stenosis by Doppler ultrasound and two-dimensional echocardiography

TERJE SKJAERPE, M.D., LARS HEGRENAES, M.D., AND LIV HATLE, M.D.

ABSTRACT In 30 patients with aortic stenosis, 14 of whom also had significant aortic regurgitation, the velocities in the stenotic jet (V′) and below the valve (V) were recorded by Doppler ultrasound. With two-dimensional echocardiography, two subvalvular areas (A) were calculated from leading-to-leading edge ("large") and trailing-to-leading edge ("inner") diameter measurements. The aortic valve area was calculated by the equation of continuity (A′ = A × peak V/peak V′) and by calculating stroke volume below the valve [A × integral of V(t)] and dividing by the integral of V′(t) (= A′′). Based on cardiac output estimations from single-plane angiographic images, Gorlin's formula was used to calculate invasive valve areas. In patients with no or mild aortic regurgitation a second invasive estimate was based on cardiac output measured by the Fick method. The best correlation was found when A′ (with "large" diameter) was compared with invasive results based on cardiac output measured by the Fick method (r = .89, SEE ± 0.12, n = 16); the worst was found when A′′ (with "large" diameter) was compared with invasive results based on cardiac output measurements by single-plane angiography (r = .80, SEE ± 0.20, n = 30). The results indicate that valve area in patients with aortic stenosis can be reliably estimated noninvasively, even in those with significant aortic regurgitation.
Circulation 72, No. 4, 810–818, 1985.

BECAUSE the pressure drop across a stenotic valve depends both on the effective valve area and the flow across the valve, the information it offers about the severity of the obstruction is not complete. The quantitation of valvular stenosis therefore includes an estimation of the valve area.

Ideally a method for the estimation of a valve area should be independent of valve insufficiency, of diseases of other valves, and of ventricular performance. If the valve area varies during the antegrade flow period, the method should give an estimate of the effective area. The method should also be noninvasive to allow frequent follow-up of the patient. So far the various methods described to estimate the valve area in patients with aortic stenosis only partly fulfill these criteria, either because the methods are invasive or semi-invasive,[1, 2] or because they rely on the absence of significant aortic regurgitation[1-3] or mitral regurgitation.[4]

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In this work we evaluated a completely noninvasive method designed to be accurate despite any coexisting regurgitation.

Methods

Theoretical considerations. When there is an obstruction in a flow channel, the product of flow area and flow velocity will be constant at the obstructed and the nonobstructed part of the flow channel, according to the equation of continuity:

$$A \times V = A' \times V' \quad (1)$$

where V and A are the spatial mean velocity and the area proximal to the obstruction, and V′ and A′ are the spatial mean velocity and the area at the obstruction. If V, A, and V′ are known, the stenotic area can be calculated by rearranging equation 1 as follows:

$$A' = A \times V/V' \quad (2)$$

This equation is valid both in time-steady flow and at each moment during pulsatile flow, provided that the velocity profile does not change from A to A′. It is also assumed that A is constant. If A′ is elastic and varies because of the changing pressure drop across the obstruction during pulsatile flow, the size of the valve area calculated by equation 2 will depend on when V and V′ are recorded. If peak V and V′ are recorded, it can probably be assumed that the maximal A′ is calculated, since the pressure drop is highest at this moment, forcing the valve to open maximally.

There are two ways of calculating mean A′. One is to estimate the area at frequent intervals during one flow period and

ılculate a temporal mean area (mean A′). Equation 2 must then ɔ modified to:

$$\text{Mean } A' = A \times \text{int}(V(t)/V'(t))/t \quad (3)$$

here int(V(t)/V′(t)) is V/V′ integrated over one cycle and t is .e time of one flow period (see Appendix for how equation 3 is ɔveloped). This equation involves the laborious procedure of ıeasuring V and V′ at frequent corresponding intervals on two ɔlocity curves and integrating the quotients.

The other way is to calculate an effective area of A′ (A″) by ɔtimating volume flow through A and A′, which must be equal:

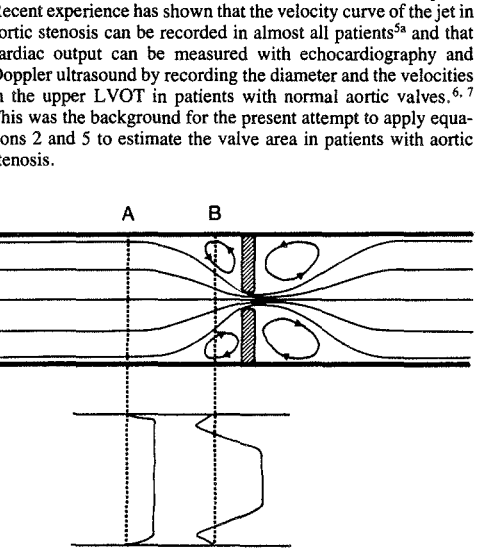
$$A \times \text{int}V(t) = A'' \times \text{int}V'(t) \quad (4)$$

here intV(t) and intV′(t) are the integrals of the velocity curves ɔcorded at A and A′. The effective valve area is obtained by ɔarranging equation 4 into:

$$A'' = A \times \text{int}V(t)/\text{int}V'(t) \quad (5)$$

he effective valve area is equivalent to a constant area that lets ıe same volume of flow pass through it when the pressure drop ıd the duration of flow are identical. The integrals of the ɔlocity curves are easily obtained with a computer and a digi-zing tablet or with a mechanical planimeter. The left side of quation 4, when applied to the left ventricular outflow tract _VOT), defines the stroke volume of the heart, which is an nportant advantage.

When the velocities proximal to the obstruction are recorded, must be realized that they are influenced by the obstruction. ɔccording to fluid mechanics, dead water zones are formed round a central, tapering core of flow with increasing velocities ɔigure 1). Because of the corresponding peaking of the velocity rofile, recordings representative of the cross-sectional mean elocity should be made proximal to this level. In our experi-nce, the increase in velocities in the LVOT, caused by the alvular stenosis, starts about 0.5 to 1.5 cm proximal to the alve. This is in agreement with the results of another report.[5] ɔecent experience has shown that the velocity curve of the jet in ortic stenosis can be recorded in almost all patients[5a] and that ardiac output can be measured with echocardiography and ɔoppler ultrasound by recording the diameter and the velocities ı the upper LVOT in patients with normal aortic valves.[6, 7] ɔhis was the background for the present attempt to apply equa-ons 2 and 5 to estimate the valve area in patients with aortic ɔenosis.



IGURE 1. When there is an obstruction of a flow channel, an increase ı velocities proximal to the obstruction (level B) will be observed. If, as ı the LVOT, the flow proximal to the obstruction is accelerated, the elocity profile at level A is flattened. Velocities representing the spatial ɔean velocity should therefore be recorded at this level.

Patients. Thirty-six consecutive patients were studied. Informed consent was obtained from all. Six were excluded, four because the cardiac output or the pressure drop was not obtained as a result of problems experienced during catheterization, and two because they had significant aortic and mitral regurgitation, precluding calculation of valve area from Gorlin's formula. None were excluded because of failure in obtaining the echocardiographic and Doppler measurements. Of the remaining 30 patients, 10 were women and 20 were men, ranging in age from 38 to 76 years (mean 63.1).

Twenty-one patients were examined with echocardiography/Doppler within 2 days of the catheterization, and three within 4 days. Six patients were examined just before replacement of the valve, and the interval from the time of catheterization ranged from 27 to 87 days (mean 60). None had clinical signs indicating increasing severity of the aortic stenosis during this period.

By angiographic criteria for regurgitation,[8] nine patients had mild (+) aortic regurgitation, nine had moderate (+ +), two had moderate to severe (+ + +), and three had severe (+ + + +). Eight patients had mild (+) mitral regurgitation. Three patients had atrial fibrillation.

Diameter measurements. For the subvalvular diameter measurements we used an Irex III B (Irex Corp.) 3.5 MHz phased-array or an ATL mark III (Advanced Technology Laboratories) 3 MHz mechanical sector scanner. The LVOT and the aortic root were visualized in the parasternal long-axis view, using the intercostal space from which the clearest image of these structures was obtained (figure 2).

At least five copies of the two-dimensional image were recorded. The transducer was repositioned between each recording. The diameter was measured preferably in systole, but sometimes measurements were made in late diastole because the upper part of the LVOT was clearly visualized only in this part of the cardiac cycle. Despite careful adjustment of gain, the presence of calcium deposits in the wall of the LVOT might cause some blooming of the echoes. Therefore, on each image two diameters were measured, one "large," obtained from leading edge measurements, and one "inner" diameter measured from the trailing edge of the anterior echo to the leading edge of the posterior echo. Because the leading edge of the anterior echo was often difficult to define due to the calcium deposits, the "large" diameter was not allowed to exceed the "inner" diameter by more than 2 mm. If the diameters measured on different recordings varied in size, the largest obtained in at least two

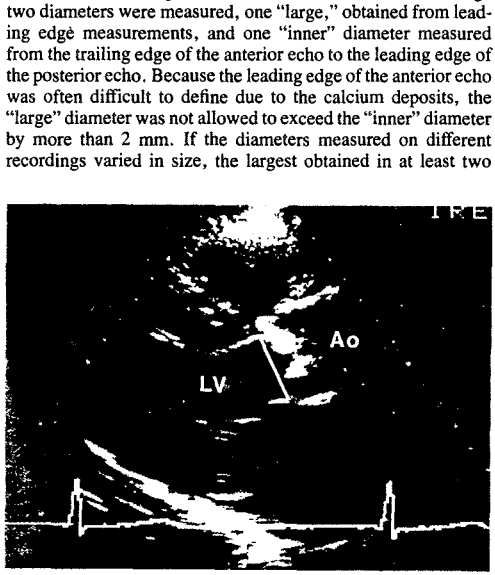


FIGURE 2. The diameter is measured just below the aortic valve to avoid any influence of the systolic thickening of the septum and movement of the anterior mitral valve leaflet. LV = left ventricle; Ao = aorta.

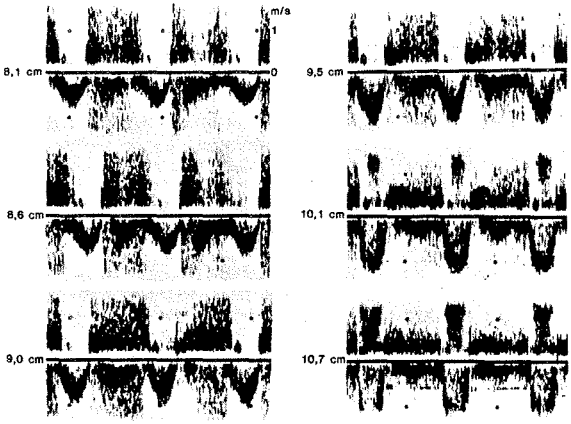


FIGURE 3. Velocities recorded with pulsed Doppler in the distal part of the LVOT. The aortic valve is located at about 10.7 cm from the transducer (the level showing maximal aliasing of the velocities). In this patient, steeply increasing velocities were recorded from 9.5 cm. The velocities recorded at 9.0 cm were therefore chosen as representing the spatial mean.

recordings was chosen for the subsequent calculations. The cross-sectional area was calculated by assuming a circular shape of the distal part of the LVOT.

Velocity recordings. The velocity recordings were obtained with the Irex III B, which includes a 2 MHz Doppler instrument (Pedof, Vingmed), allowing pulsed- or continuous-wave Doppler measurements to be made simultaneously with imaging. Velocity recordings with a separate, optimized Doppler transducer were also possible.

The subvalvular velocities were recorded from the apex with pulsed-wave Doppler. The image and the audio signal were used to align the ultrasound beam to the direction of flow. The positioning of the sample volume was made as follows: When the sample volume was moved out of the LVOT, gradually increasing velocities were recorded until a much steeper rise occurred 0.5 to 1.5 cm proximal to the valve (figure 3). The corresponding increase in pitch of the audio signal was easily heard. The sample volume was placed just proximal to this level.

The velocities in the stenotic jet were recorded with continuous-wave Doppler. A meticulous search for the highest velocities was made by recording from the apex, along the right sternal border, from the suprasternal notch, and sometimes also from the right and left supraclavicular regions. The separate Doppler transducer was used because the combined echocardiographic/Doppler transducer — even in Doppler-only mode — may fail to display the highest velocities because of suboptimal sensitivity. Typical recordings from the LVOT and the stenotic jet are shown in figure 4.

The peak and the mean pressure drops were calculated as described by Hegrenaes and Hatle.[5a] Integration of the velocity curves was made with a digitizing tablet and a computer (Cardio 80, Kontron AG) or with a mechanical planimeter (Cora senior, G. Coradi AG). The subvalvular integrals and peak velocities were averaged over 5 beats in sinus rhythm and over 10 beats in atrial fibrillation. In some patients it was difficult to obtain optimal velocity recordings from the stenotic jet in more than a few beats, and the integrals and peak velocities of the recordings from the jet were averaged over only 3 beats. Beats giving the largest integrals were selected, provided that the heart rate was stable. In three patients with atrial fibrillation it was possible to average the integral over 10 consecutive beats. The opening and closing of the aortic valve was identified to ensure that diastolic velocities were not included.

The valve area was calculated from both equations 2 and 5. Two estimates were obtained from each equation by the two diameters measured echocardiographically.

The subvalvular integral was multiplied with the cross-sectional area of the LVOT and the heart rate to give an estimate of total cardiac output (effective cardiac output + regurgitant volume).

In eight patients the velocities in the LVOT were recorded at multiple levels, and the differences in integral size and peak velocity from one level to the next were calculated.

Catheterization. The patients received a mild sedative 1 hr before catheterization; otherwise there was no change in medication between the noninvasive and invasive measurements.

The pressures were recorded with a fluid-filled No. 7F pigtail catheter connected to an Elema 746 pressure transducer. The pressure difference was calculated by superimposing pressure recordings obtained by pullback of the catheter from the left ventricle to the ascending aorta. Beats with comparable RR

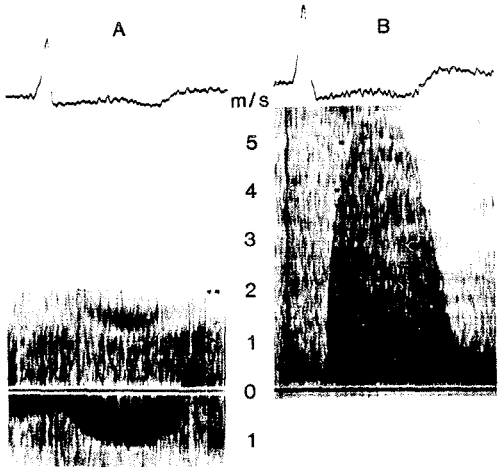


FIGURE 4. Representative subvalvular (*A*) and valvular (*B*) velocity recordings. The paper was run at a speed of 100 mm/sec to increase the accuracy when the curves were integrated.

TABLE 1
Catheterization data[A]

Patients	Pressure drop (mm Hg)		Cardiac output (l/min)		Aortic valve area (cm^2)	
	Peak	Mean	F	A	G/F	G/A
All (n = 30)	89±31	62±22	5.5±1.9	6.5±2.5		0.92±0.47
With no or mild AI (n = 16)	90±35	64±24	5.4±1.6	5.5±1.4	0.74±0.33	0.74±0.22
With > mild AI (n = 14)	88±29	60±20	5.6±2.3	7.7±3.0		1.14±0.58
Doppler < 4 days from cath. (n = 24)	91±32	64±23	5.7±2.0	6.7±2.7		0.95±0.52
Doppler > 4 days from cath. (n = 6)	84±32	54±16	4.8±1.4	5.9±1.3		0.82±0.13

F = Fick method; A = single-plane angiography; G = Gorlin's formula; AI = aortic regurgitation.

[A]In groups including patients with more than mild aortic regurgitation, only the valve areas for which the calculations are based on single-plane angiographic estimates of flow are shown. Values presented as mean ± 1 SD.

tervals were used for assessing the peak and mean pressure ops. In the presence of premature beats the following 2 sinus ats were disregarded. The peak pressure drop was obtained as average of the largest instantaneous pressure drop from 3 presentative beats. In the same beats the area between the ntricle and the aortic pressure curves in systole were planimered to give the mean pressure drop. In atrial fibrillation, beats ith a preceding RR interval representative of the mean heart te were chosen. Cardiac output was measured by the direct ick method.[9] The oxygen content in air and blood was measured with an OM-11 (Beckman Instruments Inc.) and an OSM (Radiometer, Copenhagen), respectively. Because effective ardiac output will underestimate flow across the valve in patients with aortic regurgitation, total cardiac output was also alculated from single-plane angiographic images by a modification[10] of the Dodge-Sandlers method. Aortic valve area was alculated with Gorlin's formula.[1]

Results

There were no statistically significant differences in eart rate ($p > .05$) and systolic and diastolic blood ressure ($p > .1$ and $p > .5$) between the noninvasive nd invasive examinations.

The invasive results are presented in table 1 and the oninvasive results in table 2. The "large" diameter as 2.2 ± 0.3 cm and the "inner" diameter was 2.0 ± .3 cm (mean ± SD). In patients with no or mild aortic egurgitation two invasive estimates of the valve area ere calculated, one with cardiac output measured by single-plane angiographic images and one with measurements obtained by the Fick method, assuming that the regurgitant volume in patients with mild aortic regurgitation is within the error of the Fick method. This is supported by the findings in figure 5, where cardiac output measurements by the two methods are compared. There is no tendency for the Fick method to underestimate flow across the valve in patients with mild aortic regurgitation when compared with single-plane angiographic measurements. The same assumption was made about the regurgitant volume in patients with mild mitral regurgitation when total cardiac output was quantitated from single-plane angiographic images.

Table 3 shows the correlations obtained when measurements of pressure drops, cardiac output, and valve areas were compared. The rather poor correlation between noninvasive and invasive measurements of flow might be explained by the time interval of 1 to 3 months between the two measurements in some patients. Therefore, noninvasive measurements of cardiac output were also compared with invasive measurements (Fick) in a subgroup of patients (patients with no or mild aortic regurgitation, examined with echocardiography/Doppler within 4 days of catheterization).

ABLE 2
oninvasive results[A]

Patients	Pressure drop (mm Hg)		Cardiac output (l/min)		Aortic valve area (cm^2)			
	Peak	Mean	D/El	D/Ei	A''/El	A''/Ei	A'/El	A'Ei
ll	92±32	61±23	5.9±1.8	5.0±1.6	0.82±0.35	0.70±0.29	0.92±0.42	0.78±0.36
ith no or mild AI	92±35	64±25	4.9±1.3	4.3±1.1	0.71±0.26	0.61±0.22	0.77±0.27	0.66±0.22
ith > mild AI	92±30	58±20	7.0±1.7	5.9±1.7	0.95±0.39	0.80±0.33	1.10±0.50	0.93±0.44
oppler < 4 days from cath.	93±33	62±24	5.9±1.9	5.0±1.7	0.83±0.38	0.71±0.32	0.94±0.47	0.79±0.40
oppler > 4 days from cath.	88±30	58±21	5.7±1.5	5.0±1.4	0.76±0.10	0.67±0.11	0.86±0.07	0.76±0.07

D/El = Doppler/echo measurements, "large" diameter used; D/Ei = Doppler/echo, "inner" diameter used; A'' = valve area calculated by equation "large" (El) and "inner" (Ei) diameter; A' = valve area calculated by equation 2, same diameters.

[A]Values presented as mean ± 1 SD.

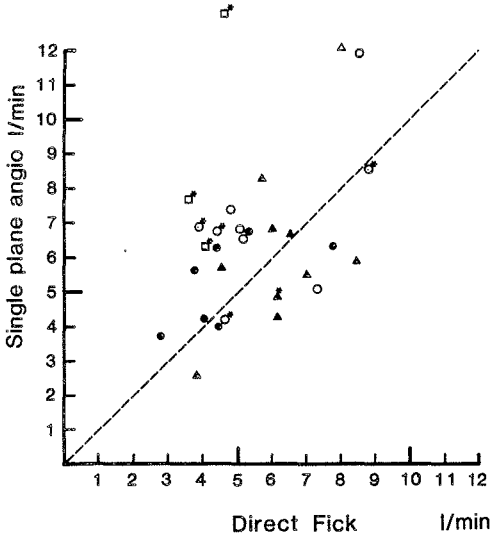


FIGURE 5. Effective cardiac output measurements obtained by the Fick method compared with total cardiac output measurements from single-plane angiographic images. Symbols represent patients with the following degrees of aortic regurgitation: ● = no aortic regurgitation; ▲ = mild; ○ = moderate; △ = moderate to severe; □ = severe. Data points marked with an asterisk represent patients who also had mild mitral regurgitation.

Figures 6 to 9 graphically present comparisons between noninvasive and invasive estimates of valve areas. The noninvasive areas were calculated with the "large" diameter. There is tendency for the larger valve areas to be underestimated, especially when calculated by equation 5. When the "inner" diameter was used, this tendency was more pronounced, affecting also smaller valve areas (table 3).

The valve areas calculated from equations 2 and 5 are compared in figure 10. The largest difference between the estimates in one patient was 37% (0.41 cm^2).

In five patients a second diameter measurement was made on another occasion. In two the diameter was identical to the first measurement, in two there was a difference of 1 mm, and in one there was a difference of 2 mm.

In the eight patients in whom the velocities were recorded at several levels in the LVOT, it was decided which was the last level to show only a gradual increase in velocities and which was the first level to definitely indicate the steeper rise. Peak velocities and velocity integrals were measured from recordings made at these two levels. Differences in peak velocity and integral size in paired recordings ranged from 5% to 14% (mean 9%).

TABLE 3
Comparison of pressure drops, cardiac output, and valve areas

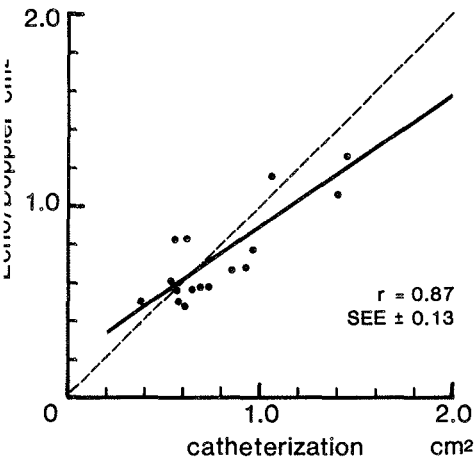
Comparisons	Correlations r value	p value	Regression equation	SEE	No. of patients
Pressure drops					
Peak	.93	<.001	y=0.94x+7.60	11.75	30
Mean	.92	<.001	y=0.95x+2.48	9.23	30
Cardiac output					
D/El-A	.70	<.001	y=0.52x+2.53	1.29	30
D/Ei-A	.71	<.001	y=0.46x+1.99	1.13	30
D/El-F	.46	<.1	y=0.37x+2.93	1.12	16
D/Ei-F	.45	<.1	y=0.32x+2.52	0.99	16
A-F	.49	<.1	y=0.34x+3.89	1.39	16
D/El-F[A]	.71	<.01	y=0.54x+1.76	0.88	12
D/Ei-F[A]	.69	<.02	y=0.47x+1.44	0.76	12
Valve area					
A''/El-G/A	.80	<.001	y=0.59x+0.27	0.20	30
A''/Ei-G/A	.82	<.001	y=0.51x+0.23	0.17	30
A'/El-G/A	.87	<.001	y=0.79x+0.20	0.21	30
A'/Ei-G/A	.87	<.001	y=0.68x+0.16	0.17	30
A''/El-G/F	.87	<.001	y=0.70x+0.18	0.13	16
A''/Ei-G/F	.86	<.001	y=0.59x+0.17	0.11	16
A'/El-G/F	.89	<.001	y=0.74x+0.21	0.12	16
A'/Ei-G/F	.89	<.001	y=0.62x+0.20	0.10	16
G/A-G/F	.73	<.01	y=0.49x+0.37	0.15	16

Abbreviations as in tables 1 and 2.

[A]Subgroup: patients with no or mild aortic regurgitation examined with echo/Doppler within 4 days of catheterization.

Discussion

The obvious difficulty in evaluating a new method for estimating valve areas is the problem of what to compare it with. Even in the absence of aortic regurgitation, Gorlin's formula may produce errors of up to 20% to 40% by including an empirical constant, the discharge coefficient.[11] Additional errors may be introduced by incorrect measurements of cardiac output and pressure drop. Besides, in the presence of aortic regurgitation, the valve area will be underestimated. It is possible to substitute effective cardiac output in the formula with total cardiac output calculated from single-plane angiographic images, except when there is significant mitral regurgitation. However, cardiac output measurements with this method may be less accurate in hypertrophic than in nonhypertrophic ventricles because of the influence of the larger papillary muscles. This may explain the poor correlation between invasive measurements of cardiac output and of valve area in these patients (table 3) and also the larger standard error of the estimate in figures 8 and 9. Considering these limitations, the correlations obtained in figures 6 to 9 indicate that the present method is



IGURE 6. Aortic valve area in patients with no or mild aortic regurgi-ıtion. Cardiac output (Gorlin's formula) was measured by the Fick ıethod. The noninvasive results are based on integration of the velocity urves (equation 5) and the "large" subvalvular diameter.

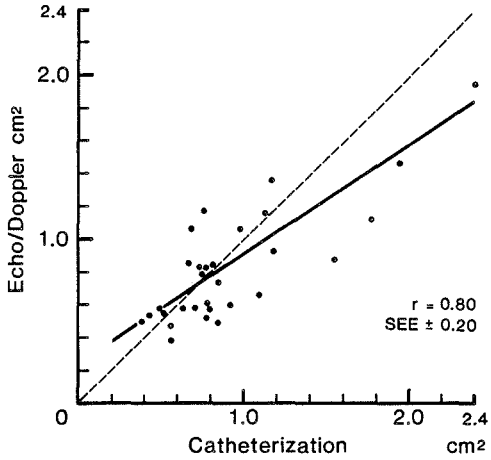
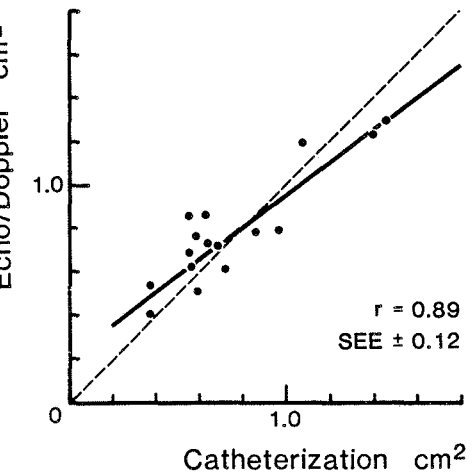


FIGURE 8. Aortic valve area in all patients. Cardiac output (Gorlin's formula) was measured by single-plane angiography. The noninvasive measurements are based on integration of the velocity curves (equation 5) and the "large" subvalvular diameter.

easonably accurate for estimating the aortic valve rea.

Figure 10 shows that valve areas calculated by equa-on 2 are equal to or larger than those calculated from quation 5. The assumption that equation 2 calculates a naximal area and equation 5 an effective area is thus upported. This difference seems to be less marked vhen the valve area is small, possibly indicating that nose valves are more calcified and less elastic. The ifference cannot be explained by the peak velocity in the stenotic jet being less well recorded than the rest of the velocity curve, since both the peak and mean pressure drops calculated from the velocity recordings correlated well with the corresponding invasive measurements (table 3).

Valve areas calculated from equation 2 correlated better with invasive results (figures 7 and 9) than did the valve areas calculated from equation 5 (figures 6 and 8). Especially the larger areas seemed to be underestimated by equation 5. The reason for this is difficult



IGURE 7. Same as in figure 6, except that the noninvasive results are ased on peak velocity measurements (equation 2).

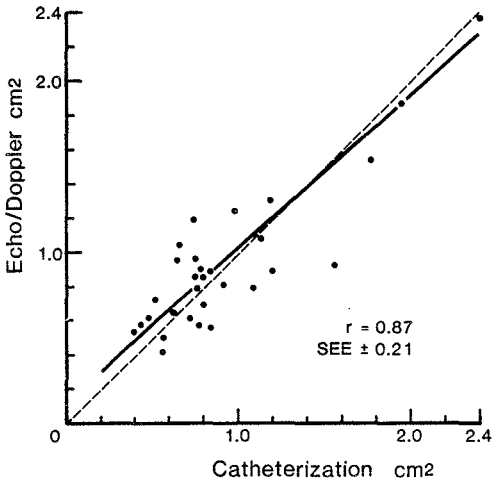


FIGURE 9. Same as in figure 8, except that the noninvasive results are based on peak velocity measurements (equation 2).

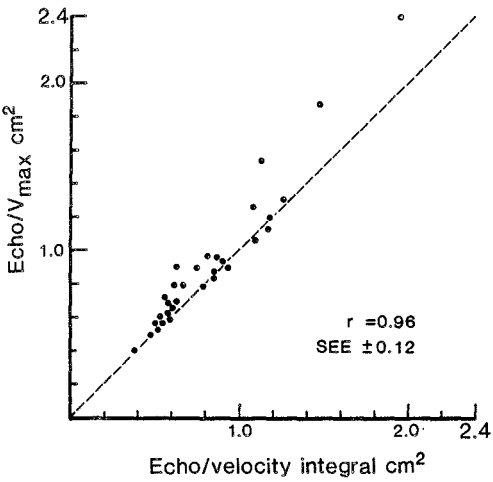


FIGURE 10. Aortic valve area calculated from equation 2 (vertical axis) and equation 5 (horizontal axis). The "large" diameter was used in both equations.

to ascertain because there were few patients with large valve areas in this series and because the measurements were not done simultaneously. However, according to Rodrigo,[11] the discharge coefficient in Gorlin's formula can only be assumed to be constant when the valve area is small compared with the area proximal to the valve. Larger valve areas will be overestimated. One therefore cannot exclude that the larger valve areas in figures 6 and 8 are overestimated by Gorlin's formula rather than underestimated by equation 5. Thus in figures 7 and 9 the overestimation of effective valve area made by equation 2 may seem to be balanced by an overestimation of similar magnitude by assuming the discharge coefficient in Gorlin's formula to be constant, accidentally causing the two methods to correlate well. The tendency for Gorlin's formula to overestimate large valve areas was also shown by Warth et al.[2]

Reliability of velocity and diameter measurements. The reliability of the estimates of the right sides of equations 2 and 5 is best established for V′ and intV′(t). In accordance with the results of Hegrenaes and Hatle,[5a] the peak and mean pressure drops calculated from the velocity recordings correlated well with the invasive results (table 3), indicating that the velocity curve of the stenotic jet was adequately recorded in all patients. The accuracy of valve area estimations will therefore largely depend on the reliability of subvalvular velocity and diameter recordings.

Ihlen et al.[12] and Gussenhoven et al.[13] found the reproducibility of subvalvular diameter measurements to be good in patients with noncalcified aortic valves. In the case of calcific aortic stenosis, the accuracy is probably less good. Cohen et al.[14] measured the subvalvular diameter to predict the prosthetic valve size in adult patients with aortic stenosis. Errors up to 2 mm were made. This is similar to the difference in diameters on repeated measurements that we found in five patients and indicates that an error of about 10% to 20% in the assessment of subvalvular area has to be expected in some patients.

In some patients the diameter was measured in late diastole because the upper LVOT was poorly visualized in systole. In the case of different diameters in systole and diastole, the systolic value should probably be used. However, there is reason to believe that this diameter does not change much during the cardiac cycle.[12]

Assessment of the reliability of the subvalvular velocity recordings is difficult. In some patients stable velocities were recorded over a distance of 1 to 2 cm in the upper LVOT, making the decision on which velocities to use easy. In general, however, the velocities increased gradually until the more rapid increase 0.5 to 1.5 cm proximal to the valve. Even if this rapid increase could occur quite suddenly, there was necessarily a range of velocities from which the correct one was difficult to choose. In figure 3 this range is represented by the velocities recorded at 9.0 and 9.5 cm. These recordings are from a patient in whom the change from gradually to steeply increasing velocities was less marked than usual. The resulting differences in maximal velocity and integral size, about 14%, therefore probably represent the upper limit of the error in calculated valve area, introduced by subvalvular velocity recordings. In seven other patients this range gave differences of 12% or less.

It may be argued that diameter and velocity measurements should be made at the same level in the LVOT (compare figures 2 and 3). Because the LVOT may show considerable axial movement during systole (we have noticed up to 1 cm), this is not possible. Pasipoularides et al.[5] found that the distance between the level just below the valve and the level of the stenosis ranged from 0.26 to 0.95 cm (mean 0.52). When this is added to the distance of the systolic movement of the LVOT, our procedure for diameter and velocity measurements appears to be acceptable.

The combined error produced by subvalvular velocity and diameter recordings can be evaluated by calculating cardiac output and comparing the results with invasive measurements (table 3). The poor correlation obtained is probably partly explained by the fact that

e measurements were done simultaneously, partly by e inaccuracy of subvalvular velocity and diameter easurements as discussed above, but also by the inac- ıracies of the invasive methods (table 3). Neverthe- ss, the standard error of the estimate as obtained in e subgroup of patients is not very different from that ported in other studies in which the noninvasive and vasive measurements of cardiac output were made multaneously.[12, 15–18] In the same group of patients the rgest underestimation of cardiac output occurring hen the "inner" diameter was used was 49%, com- ıred with 38% when the "large" diameter was used. his may indicate that the "large" diameter is to be eferred for calculations of volume flow and valve ea, but again, due to the fact that measurements were ot made simultaneously, it is difficult to make any ıal evaluation of the problem.

The reasons why noninvasive measurements of essure drops correlated better with the invasive re- ılts than did the measurements of cardiac output are obably that both the invasive and noninvasive meth- ds are more accurate in assessing pressure drops than olume flow and that variations in flow will produce mparatively smaller variations in pressure drop be- ause the ejection time also varies. Variations in heart te will also influence cardiac output and pressure op differently.

If the errors in diameter and velocity measurements e added, they may be of the same magnitude as the fference between the valve areas estimated from uation 2 and 5. The simplicity of equation 2 prob- ly justifies its use in the clinical routine. If the esti- ated area is borderline regarding the decision of how treat the patient, it might be helpful to calculate the ea from equation 5 as well.

Our results show that this method can also be used hen there is significant aortic regurgitation, which as present in 14 patients. Even though it was not esent in this series, significant mitral regurgitation ould not invalidate the method. Thus the limitations other methods[1–4] are eliminated. No empirical con- ant is used, reducing errors caused by individual vari- ions. The approximation made by Kosturakis et al.[3] assuming the temporal mean velocity in the stenotic to be 0.88 × peak velocity is also avoided. Accord- g to our observations, the quotient Vmean/Vpeak an range from 0.69 to 0.87. By assuming a fixed uotient, the method will tend to average the results. n the other hand, the error made may not be impor- nt compared with other errors inherent in any method sed to estimate valve areas.

The left and right sides of equation 4 describe the stroke volume at two levels that are in series and closely located. Hemodynamic changes should therefore affect both sides of the equation similarly, making the method independent of left ventricular performance.

Fujii et al.[19] have simultaneously and independently of our study used equation 2 to calculate the valve area in aortic stenosis and have derived similar results.

There may be some practical problems in using equations 2 and 5. Especially in patients with emphysema, in whom the subvalvular diameter can be very difficult to measure, the results should be accepted with some reservation. Caution should also be exercised when calcifications are seen to protrude into the LVOT. This was seen in two patients in the present series. We attempted to solve the problem by placing the sample volume just proximal to the calcification. Thereby the increase in velocity, secondary to the decrease in cross-sectional area caused by the calcifications, was avoided. The calculation of the subvalvular area was made as described earlier, disregarding the calcifications. This apparently was successful, since the noninvasive estimates of valve area were quite close to the invasive results (invasive results, based on Fick method, 0.59 and 0.86 cm^2; noninvasive results with the "large" diameter, 0.50 and 0.66 cm^2 [equation 5], 0.56 and 0.79 cm^2 [equation 2]).

If subvalvular obstruction caused by septal hypertrophy is present, the subvalvular velocity profile may be unpredictable and calculated valve area should be looked upon only as a rough estimate. Significant subvalvular obstruction was not seen in any patient in this study. In fact, problems were more frequently encountered when collecting the invasive than the noninvasive data, emphasizing the usefulness of the noninvasive method.

Appendix

If the stenotic area (A′) of a flow channel varies because of the changing pressure drop across the obstruction during pulsatile flow, the equation of continuity can be developed to calculate the temporal mean of A′.

The equation of continuity is valid at any moment during pulsatile flow:

$$A \times k(prof) \times V = A' \times k(prof)' \times V'$$

A and V are the area and maximal velocity proximal to the obstruction, and V′ is the maximal velocity at the obstruction; k(prof) is a constant describing the velocity profile [k(prof) = 1 if the profile is flat, k(prof) = ½ if the profile is parabolic]. If k(prof) = k(prof)′, the equation can be reduced and rearranged to:

$$A'/A = V/V'$$

Both sides are then integrated over one flow period:

$$\text{int}(A'/A) = \text{int}(V/V')$$

If A is constant:

$$\text{int}(A') = A \times \text{int}(V/V')$$

Temporal mean of $A' = \text{int}(A')/t$, where t is the duration of one flow period:

$$\text{mean } A' = A \times \text{int}(V/V')/t$$

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